

UNIT 10

Metabolic and Endocrine Function

Case Study

APPLYING PATIENT-CENTERED CARE FOR THE PATIENT WITH DIABETES



You are a certified diabetes care and education specialist in a community hospital. Today you are making a home visit to a 53-year-old Black man to follow up on the results of a 13% HgbA_{1c} that was drawn a few weeks ago. He is 5'5", weighs 180 lb, his finger stick glucose today is 310 mg/dL, and he lives with his adult daughter. While discussing the associations between an HgbA_{1c}

and poor blood glucose control, you review the pharmacologic and nonpharmacologic treatments. During the visit the patient and his daughter state they want to try cinnamon tea and other alternative and complementary therapies to help control his blood glucose naturally. You intend to collaborate with the family's preferences and educate them on the various ways he can maintain glucose control through diet, exercise, and medications.

QSEN Competency Focus: Patient-Centered Care

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.***

Patient-Centered Care Definition: Recognize the patient or designee as the source of control and full partner in providing compassionate and coordinated care based on respect for patient's preferences, values, and needs.

SELECT PRE-LICENSURE KSAs	APPLICATION AND REFLECTION
Knowledge	
Skills	
<p>Integrate understanding of multiple dimensions of patient centered care:</p> <ul style="list-style-type: none"> • patient/family/community preferences, values • coordination and integration of care • information, communication, and education • physical comfort and emotional support • involvement of family and friends • transition and continuity 	<p>Based on this patient's preferences, how can you provide education to this patient and his family regarding the management of his blood glucose? Identify how you can incorporate the patient's preferences for alternative and complementary therapies with diet and exercise management.</p>
<p>Describe how diverse cultural, ethnic and social backgrounds function as sources of patient, family, and community values</p>	
<p>Communicate patient values, preferences and expressed needs to other members of health care team</p>	<p>During the visit, you discuss with the patient and family ways they can adhere to a diet, exercise, and medication regimen. How can you communicate support of their choice to incorporate alternative and complementary therapies in managing the patient's blood glucose so that healthy glycemic control is achieved?</p>
Attitudes	
<p>Value seeing health care situations "through patients' eyes"</p>	<p>Reflect on the complex interrelationships between the patient's desire to manage his blood glucose with alternative therapies. Think about your own desire for this patient to adhere to a diet, exercise, and medications regimen.</p>
<p>Respect and encourage individual expression of</p>	

patient values, preferences,
and expressed needs

How might you demonstrate that you
value the patient's preferences to utilize
alternative and complementary
therapies?

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; Patient centered care*. Retrieved on
8/15/2020 at: qsen.org/competencies/pre-licensure-ksas/#patient-centered_care

42 Assessment and

Management of Patients with Obesity

LEARNING OUTCOMES

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

1. Describe the causes, risks, and pathophysiology associated with obesity.
2. Discriminate between normal and abnormal assessment findings identified in the patient with obesity.
3. Identify strategies aimed at preventing and treating obesity, including lifestyle modification, pharmacologic therapy, and nonsurgical interventions.
4. Explain nursing management considerations for the patient with obesity using nonsurgical interventions.
5. Compare and contrast surgical modalities indicated to treat patients with obesity in terms of preoperative, postoperative, and long-term management and complications.
6. Use the nursing process as a framework for care of the patient who undergoes bariatric surgery.

NURSING CONCEPTS

GLOSSARY

adiposopathy: dysfunction of adipose tissue that causes chronic inflammation and disease

bariatric: relating to obesity; term derives from two Greek words meaning “weight” and “treatment”

body mass index (BMI): a weight-to-height ratio, calculated by dividing weight in kilograms by height in meters squared; the most common measure used to classify and diagnose obesity

dumping syndrome: physiologic response to rapid emptying of gastric contents into the jejunum, manifested by nausea, weakness, sweating, palpitations, syncope, and possibly diarrhea (*synonym:* vagotomy syndrome)

dysphagia: difficulty swallowing

genome: the total complement of individual genes in an organism

microbiome: the collective genome of all microbes in a microbiota

microbiota: the complement of microbes in a given environment

obesity: a disease characterized by an abnormal or excessive accumulation of body fat that impairs health

obesogenic: a factor that promotes weight gain and obesity

orexigenic: a factor that stimulates appetite

satiety: feeling of having eaten sufficient quantities of food

Obesity in the United States and globally has reached pandemic proportions. Given the high prevalence of obesity, nurses will encounter adults with obesity in every inpatient and outpatient clinical setting. This chapter describes the etiology, risks, assessment, clinical manifestations, management, and related nursing care of the patient with obesity. The management of patients with obesity using both nonsurgical and surgical treatments is discussed.



Obesity

Obesity is defined by the World Health Organization (WHO) as an “abnormal or excessive fat accumulation that may impair health” (WHO, 2018, p. 1). As a response to endorsements by multiple health care organizations and societies, including the American College of Cardiology, the Endocrine Society, and the

American College of Surgeons, to name a few, the American Medical Association (AMA) House of Delegates in 2013 officially resolved that obesity should be diagnosed and treated as a disease (AMA, 2013). This resolution was based on the scientific observation that obesity followed criteria commonly used for defining a disease; namely, it can be said that obesity impairs normal bodily function, possesses characteristic signs and symptoms, and causes morbidity (AMA, 2013).

Epidemiology of Obesity

Worldwide, over 650 million adults have obesity, and another 1.9 billion are overweight (WHO, 2018). Since 1975, the prevalence of obesity has more than tripled for men, and more than doubled for women. In particular, 3% of the world's men and 6% of the world's women had obesity in 1975, whereas 11% of the world's men and 15% of the world's women had obesity in 2016 (WHO, 2018). The burden of obesity is significant in both developed and developing nations. The WHO (2018) notes that many developing nations are now facing a "double-burden" effect from disorders linked to nutrition and metabolism; that is, these nations must simultaneously deal with the public health threats of both undernutrition and obesity. In developing nations, obesity has become particularly prevalent in urban settings (WHO, 2018).

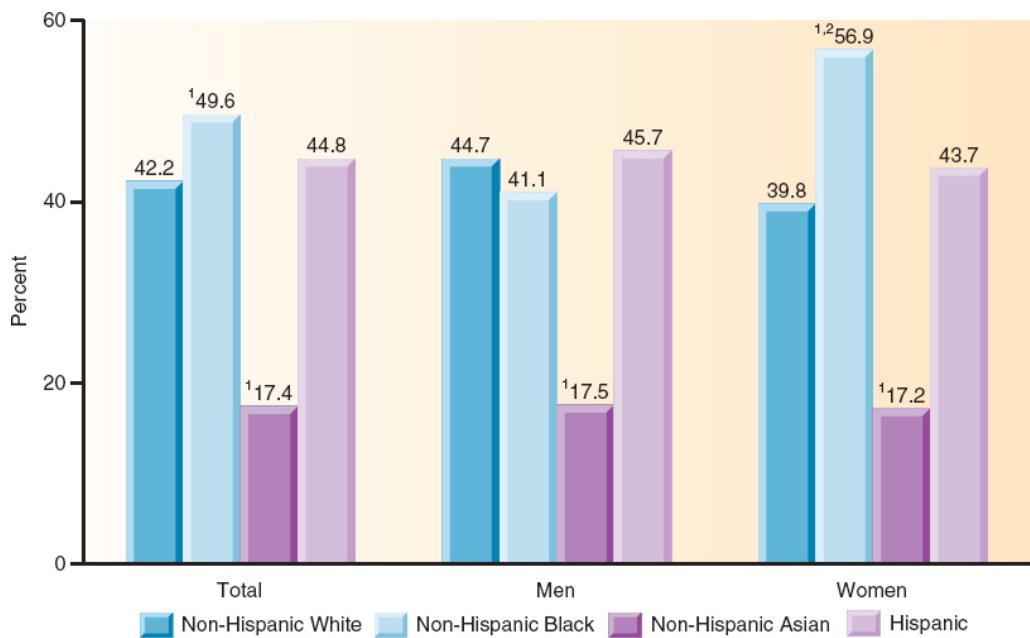
Since 1980, the number of adults with obesity in the United States has continued to rise (Henry, 2018; Trust for America's Health [TFAH] & Robert Wood Johnson Foundation [RWJF], 2018). The prevalence of obesity in the United States is now the 12th highest among nations in the world, with Nauru, an island nation in Micronesia, being first, with an obesity prevalence of 61% (Hales, Carroll, Fryar, et al., 2020; Hales, Fryar, Carroll, et al., 2018). Among American adults, an estimated 42.4% have obesity (Hales et al., 2020); an estimated 70.9% have obesity or are overweight (Hales et al., 2018). The overall prevalence of overweight and obesity is slightly higher among American women than American men, and among African Americans and Hispanics than among Whites or Asian Americans (see Fig. 42-1). In general, those who are less educated and earn less income are more likely to have obesity, reflecting socioeconomic disparities in the disease burden of obesity (Hales et al., 2018; TFAH & RWJF, 2018). Being overweight or having obesity is the primary reason why young American adults are excluded from military service (Warren, Beck, & Rayburn, 2018) (see later discussion in Veterans Considerations section).

The economic burden of obesity to American society extends beyond limiting young adults with obesity from pursuing military service commitments. It is estimated that annual health care costs tied to obesity are approximately \$190 billion (Warren et al., 2018), and that average annual health care expenditures for Americans with obesity are \$3429 higher per person than for those Americans without obesity (Biener, Cawley, & Meyerhoefer, 2017).

Obesity Risks

The causes of obesity are complex and multifactorial, and include behavioral, environmental, physiologic, and genetic factors. While there are certain demographic groups who seem to be at risk for obesity (see [Fig. 42-1](#)) and while there are notable familial patterns of obesity, identification of risk factors that specify odds of being diagnosed with obesity is not as clearly elucidated as those for other diseases (Centers for Disease Control and Prevention [CDC], 2020), such as coronary artery disease (see [Chapter 23](#)) and cerebrovascular disease (see [Chapter 62](#)).

What is abundantly clear, however, is that obesity incurs a greater overall risk of mortality. Obesity alone does not decrease a person's lifespan (Kuk, Rotondi, Sui, et al., 2018). However, obesity is associated with a 2- to 6-year decrease in overall life expectancy when coupled with metabolic disease or another chronic illness (Khan, Ning, & Wilkins, 2018). Furthermore, obesity is associated with morbidity and mortality from numerous other diseases (see [Fig. 42-2](#) and [Chart 42-1](#)). For instance, as body mass index (BMI) increases, so does the overall risk of cancer and risk for death from cancer; obesity is responsible for up to 90,000 deaths from cancer annually. Having obesity increases the likelihood of having type 2 diabetes by 10-fold and the likelihood of having either asthma or hypertension by nearly fourfold (American Society for Metabolic and Bariatric Surgery [ASMBS], 2019b). Adults with obesity are twice as likely to eventually be diagnosed with Alzheimer's disease than those adults who maintain a normal weight (Biener et al., 2017).



¹Significantly different from all other race and Hispanic-origin groups.

²Significantly different from men for same race and Hispanic-origin group.

NOTES: Estimates were age adjusted by the direct method to the 2000 U.S. Census population using the age groups 20–39, 40–59, and 60 and over. Access data table for this figure at: <https://www.cdc.gov/nchs/data/databriefs/db360-tables-508.pdf#2>.

SOURCE: NCHS, National Health and Nutrition Examination Survey, 2017–2018.

Figure 42-1 • Prevalence of obesity among adults aged 20 and older, by gender and race/ethnicity in the United States (2017–2018).

Reprinted from Hales, C. M., Carroll, M. D., Fryar, C. D., et al. (2020). Prevalence of obesity among adults and youth: United States, 2017–2018. Figure 2, page 2, National Center for Health Statistics. NCHS Data Brief, no. 360. Hyattsville, MD. Retrieved on 11/16/2020 at: www.cdc.gov/nchs/data/databriefs/db360-h.pdf

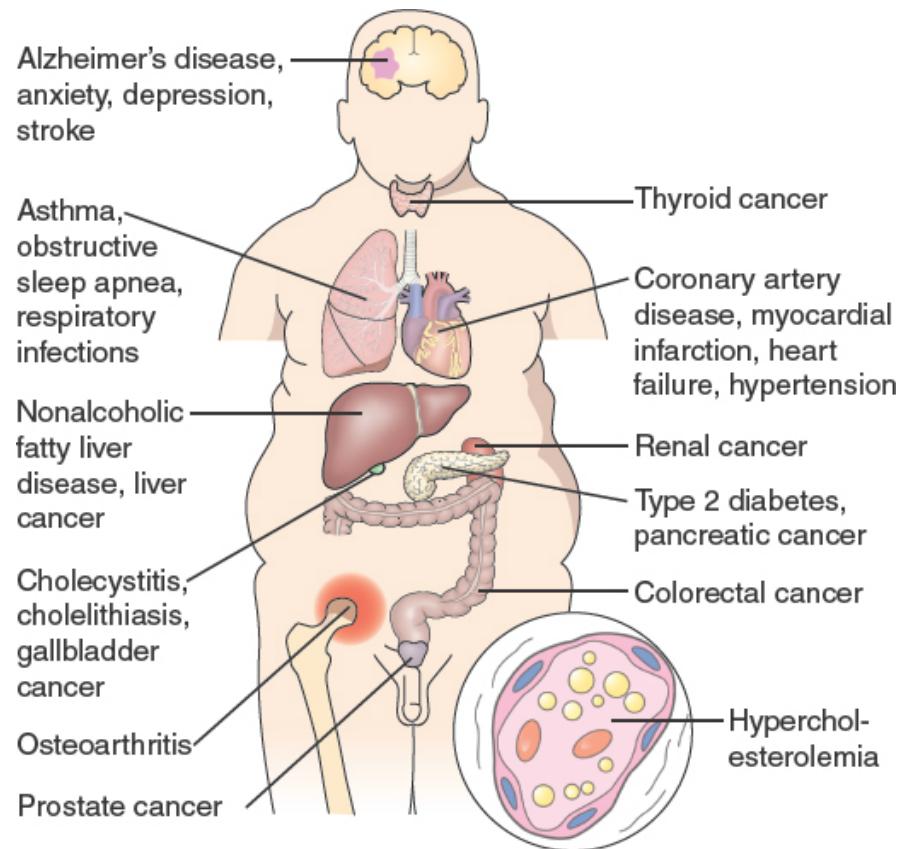


Figure 42-2 • Morbid diseases and disorders associated with obesity.

Chart 42-1

Diseases and Disorders Associated with Obesity

Alzheimer's disease
Anxiety and depression
Asthma
Cancers (breast, cervical, colorectal, endometrial, esophageal, gallbladder, liver, ovarian, non-Hodgkin lymphoma, pancreatic, prostate, kidney, thyroid)
Chronic low back pain
Coronary artery disease (angina, acute coronary syndrome, myocardial infarction)
Diabetes (type 2)
Gallbladder disease (cholecystitis, cholelithiasis)
Gastroesophageal reflux disease (GERD)
Gout
Heart failure
Hypercholesterolemia
Hypertension
Nonalcoholic fatty liver disease
Obstructive sleep apnea
Osteoarthritis
Respiratory infections
Stroke

Adapted from American Society for Metabolic and Bariatric Surgery (ASMBS). (2019b). The impact of obesity on your body and health. Retrieved on 5/1/2020 at: www.asmbs.org/patients/impact-of-obesity; Centers for Disease Control and Prevention (CDC). (2020). Adult obesity causes & consequences. Retrieved on 5/1/2020 at: www.cdc.gov/obesity/adult/causes.html

Pathophysiology

Obesity is a chronic, relapsing disease characterized by an excessive accumulation of body fat and weight gain (ASMBS, 2019a). These increases in body fat cause **adiposopathy**, a dysfunction of adipose tissue, which promotes the development of metabolic, biomechanical, and psychosocial diseases and disorders (Fruh, 2017; Wu & Berry, 2018). Dysfunctional adipose tissue cells release biochemical mediators that cause chronic inflammatory changes, which can lead to a multitude of diseases, including heart disease, hypertension, and type 2 diabetes (ASMBS, 2018a).

At the most fundamental level, obesity results from a metabolic imbalance, characterized by an excess of caloric consumption relative to caloric expenditures. That is, too many foods are consumed and too little physical activity is conducted over the long term, resulting in weight gain (Norris, 2019). According to the “thrifty gene” hypothesis, the human **genome** (i.e., the total complement of genes in humans) was sequenced during times when finding and storing food sources expended more energy than during contemporary times.

Hunting for scarce food sources during prehistoric times consumed a lot of energy, and food sources were not abundant. Storing fat to provide energy sources during times of food scarcity was a physiologic adaptive response to these environmental challenges (van Meijel, Blaak, & Goossens, 2018).

The “thrifty gene” hypothesis has come under scrutiny in recent years, however, as human genome sequencing research findings suggest that a far more complex genetic explanation may be responsible for the recent global obesity pandemic. Research has identified 79 syndromes and 31 genes that cause obesity through monogenic (single gene) mutations and polygenic (multiple gene) mutations as well as gene–environment interactions (Rohde, Keller, la Cour Poulsen, et al., 2019). Although to date only 19 syndromes have been genetically characterized, it has been recognized that having at least one different genetic mutation can strongly predispose people who have ready access to food sources to having obesity (Rohde et al., 2019). For instance, the presence of a variant mutation of the *FTO* gene is associated with more daily meals, snacks, and fat and sweet intake (Rohde et al., 2019). However, these types of single genetic mutations are relatively rare occurrences, and therefore cannot account for the high prevalence of obesity. Most people who are predisposed to obesity are thought to have a collection of several genetic mutations from more than 700 possible mutated genes that each can contribute to several pounds of additional body fat (Yengo, Sidorenko, Kemper, et al., 2018).

The types and quantities of foods consumed affect complex digestive and metabolic pathways. Certain processed and high caloric foods that contain fructose corn syrup, simple sugars, or trans fats, are hypothesized to be **obesogenic** (i.e., promote weight gain) because they are associated with food cravings consonant with other type of addictive cravings (Campana, Brasiel, de Aguiar, et al., 2019). Additionally, the portions of both entrees and desserts served in fast food restaurants have increased over the past 30 years, subtly affecting consumers’ feelings of **satiety** (i.e., feeling of having eaten sufficient amounts of food) (McCrory, Harbaugh, Appedu, et al., 2019). Furthermore, there is a greater variety of entrees and desserts that may be selected on the menus of most fast food restaurants, providing the illusion of healthier meal options although there are actually less healthy food alternatives available for selection than 30 years ago (McCrory et al., 2019).

Multiple hormones that control food cravings and feelings of fullness could be affected by individual genes. In response to periods of fasting, the hormone ghrelin is secreted by the stomach and the hormone neuropeptide Y (NPY) is secreted by the small intestines. These hormones are **orexigenic**, meaning that they stimulate appetite through central nervous system (CNS) pathways that lead to the hypothalamus, signaling higher neural pathways that lead to eating behaviors. Once eating occurs, multiple hormones are released throughout the gastrointestinal (GI) tract that promote satiety, including somatostatin, cholecystokinin (CCK), and insulin, to name a few. CCK also slows gastric motility and emptying, stimulates gallbladder contraction and release of bile into

the duodenum, and stimulates the release of pancreatic digestive enzymes, all of which serve to enhance the digestive process. Somatostatin also slows gastric emptying but has other effects that are in opposition to CCK, such as decreasing the secretion of bile, depending on foods consumed and metabolic needs (Gimeno, Briere, & Seeley, 2020).

Increases in fat stores, or adipose tissue, result in increases in the hormone leptin, which is secreted by fat cells. Leptin also has the effect of signaling satiety in the hypothalamus. Patients with obesity who lose weight are thought to also have drops in leptin levels that persist for the long term, creating persistent feelings of hunger, which may partially explain why many patients with obesity who lose weight tend to regain it (Campana et al., 2019).

By adulthood, the GI tract **microbiota**, which is the complement of microbes within the gut, has been found to contain up to 100 trillion microbes, or 10 times the cells present in the human body (McElroy, Chung, & Regan, 2017). The collective genome of the microbiota, or the gut **microbiome**, has more than 100 times more genes than in the human genome. It has long been known that gut microbes perform numerous digestive, metabolic, and immunologic functions. The composition and diversity of these microbes may very well be tied in with obesity (McElroy et al., 2017). For instance, patients with obesity tend to have less diverse microbiota than patients who are of normal weight. In turn, patients with obesity who have less diverse microbiota typically also have dyslipidemia, impaired glucose metabolism, and low-grade generalized inflammatory disorders. “Western-style diets,” which are high in processed foods, fat, and sugars and low in fiber, are thought to not only negatively affect the diversity of the gut microbiota, but also to negatively affect the complement of *Bacteroidetes* species microbes, which are associated with a leaner type of microbiome (McElroy et al., 2017). Whether or not it is possible to regulate the composition of the gut microbiota and prevent or treat obesity is the focus of ongoing research.

Assessment of Obesity

Assessment of the patient with obesity includes a health history and physical examination that evaluates the effects of obesity on the health of the patient.

Health History

Nurses need to approach patients with obesity with the same respectful, courteous, and empathetic behavior that they extend to patients without obesity. Confronting their own attitudes and beliefs about patients with obesity may help to mitigate biases. For instance, using patient-first language for all patients with diagnosed diseases, including the disease of obesity, can be an effective way to dispel bias. By referencing *the patient with obesity*, the nurse is effectively noting that the patient, not the disease, is the central point of concern and that the disease is amenable to treatment. On the other hand, referencing *the obese*

patient tends to define the person by having obesity, which can lead to subliminal impressions that the patient is somehow responsible for having obesity. Research studies report that many health care providers, including nurses, hold negative attitudes toward patients with obesity and believe that they are indulgent, lazy, and lack willpower (Robstad, Westergren, Siebler, et al., 2019; Smigelski-Theiss, Gampong, & Kurasaki, 2017).

Patients with obesity should be assessed to see if there have been any recent increases or decreases in body weight. If the patient has recently lost or gained weight, determining if that is intentional weight loss or gain may give clues to whether or not changes in weight might be secondary to another disease process. Other useful health history information to gather includes how long the patient has had obesity (e.g., since childhood, since a pregnancy) and whether or not there is a family history of obesity. Any patterns of weight loss over time, and prior successful or unsuccessful weight loss strategies should be analyzed. A history of exercise patterns and dietary patterns (see [Chapter 4](#) for discussion of nutritional assessment) is also assessed. Some patients with obesity report sleep pattern disturbances (e.g., difficulty falling asleep, difficulty staying asleep); therefore, typical sleeping habits are evaluated. Some patients who quit smoking report significant weight gain after smoking cessation is achieved; consequently, the patient's smoking status is also determined (Perreault, 2020).

Some patients with obesity may develop secondary diseases or disorders (see [Chart 42-1](#)) and may be prescribed medications aimed at treating those secondary diseases or disorders that can exacerbate weight gain (see [Chart 42-2](#)). Other patients may not have had a previous history of obesity, but may gain weight after being prescribed specific medications associated with weight gain. A history of weight gain that parallels the time frame of commencing treatment with specific prescriptive medications may suggest that the medication has a key role in promoting weight gain. In some instances, prescription medication dosages may be adjusted or another medication may be prescribed. For instance, patients with type 2 diabetes and obesity may reap the dual benefits of achieving better glycemic control and weight loss by taking the prescription drug metformin (Apovian, Aronne, Bessesen, et al., 2015).

Physical Assessment

Patient's height and weight are measured to determine the **body mass index (BMI)**. The BMI is the definitive measure used to determine whether or not a patient has obesity; this is based on a ratio of body weight in kilograms and height in meters (see [Chapter 4, Table 4-1](#)). Patients identified as overweight or pre-obese have a BMI of 25 to 29.9 kg/m² and those with obesity have a BMI that exceeds 30 kg/m². Those with a BMI exceeding 40 kg/m² are considered to have severe or extreme obesity (Nguyen, Brethauer, Morton, et al., 2020; WHO, 2017) (see [Fig. 42-3](#) and [Table 42-1](#)).

Chart 42-2  **PHARMACOLOGY**

Select Medications That Affect Body Weight

Many medications prescribed to treat a variety of chronic diseases and disorders have the untoward side effect of weight gain, whereas others are associated with weight loss. The following list contains some examples of each of these.

Medications Associated with Weight Gain

Anticonvulsant Medications:

- Carbamazepine
- Gabapentin
- Pregabalin
- Valproate
- Vigabatrin

Antidepressant Medications:

- Selective Serotonin Reuptake Inhibitors (SSRIs) (Note—these tend to be associated with early weight loss followed by gain within 6 months in some patients):
 - Citalopram
 - Escitalopram
 - Fluvoxamine
 - Paroxetine
 - Sertraline
- Tricyclic Antidepressants:
 - Amitriptyline
 - Clomipramine
 - Doxepin
 - Imipramine
 - Mirtazapine
 - Nortriptyline
 - Protriptyline
 - Trimipramine

Antihistamines:

- Azelastine
- Cetirizine
- Cyproheptadine
- Diphenhydramine
- Fexofenadine

Antihypertensive Medications:

- Alpha-Blocker:
 - Terazosin
- Beta-Blockers:

- Atenolol
- Metoprolol
- Propranolol
- Dihydropyridine Calcium Channel Blockers:
 - Amlodipine
 - Felodipine
 - Nifedipine

Antipsychotic Medications:

- Asenapine
- Chlorpromazine
- Clozapine
- Haloperidol
- Iloperidone
- Olanzapine
- Paliperidone
- Quetiapine
- Risperidone

Diabetes Medications:

- Insulins:
 - Insulin aspart
 - Insulin glulisine
 - Insulin lispro
- Meglitinides:
 - Nateglinide
 - Repaglinide
- Sulfonylureas:
 - Chlorpropamide
 - Glimepiride
 - Glipizide
 - Glyburide
 - Tolbutamide
- Thiazolidinedione:
 - Pioglitazone

Hormones:

- Corticosteroids:
 - Prednisone
 - Budesonide
 - Methylprednisolone
- Hormonal Contraceptive:
 - Medroxyprogesterone

Mood Stabilizers:

- Lithium

Medications Associated with Weight Loss

Anticonvulsant Medications:

- Lamotrigine
- Topiramate
- Zonisamide

Antidepressant Medication:

- Bupropion
- Desvenlafaxine
- Venlafaxine

Diabetes Medication:

- Dulaglutide
- Exenatide
- Liraglutide
- Lixisenatide
- Metformin
- Semaglutide

Adapted from Comerford, K. C., & Durkin, M. T. (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer; VA/DoD Clinical Practice Guideline. (2020). Medications and their affects on weight. Retrieved on 3/7/2021 at:

www.healthquality.va.gov/guidelines/CD/obesity/MedsEffectsWeightProviderToolFINAL50817Dec2020.pdf; Welcome, A. (2017). Medications that may increase weight. Retrieved on 5/1/2020 at:

www.obesitymedicine.org/medications-that-cause-weight-gain

Patients with obesity may also have their waist circumferences assessed. Women with waist circumferences more than 35 inches and men with waist circumferences more than 40 inches have greater risks for obesity-related morbidity (see [Chart 42-1](#)) than those with smaller waistlines (Meigs, 2019). The hip may also be measured and the waist-to-hip ratio assessed. Women with waist-to-hip ratios greater than 0.80 and men with waist-to-hip ratios greater than 0.90 are presumed to have proportionally more visceral (i.e., abdominal) fat stores. This morphologic appearance is called android obesity, and is sometimes referenced as an “apple-shaped” appearance. Patients with android obesity have greater risk for developing hypertension, coronary artery disease, stroke, and type 2 diabetes, than patients who have gynoid obesity, also called the “pear-shaped” body (Weber & Kelley, 2018) (see [Fig. 42-4](#)).

		Body Mass Index Table																																			
		Normal						Overweight						Obese						Extreme Obesity																	
BMI		19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54
Height (inches)														Body Weight (pounds)																							
58		91	96	100	105	110	115	119	124	129	134	138	143	148	153	158	162	167	172	177	181	186	191	196	201	205	210	215	220	224	229	234	239	244	248	253	258
59		94	99	104	109	114	119	124	128	133	138	143	148	153	158	163	168	173	178	183	188	193	198	203	208	212	217	222	227	232	237	242	247	252	257	262	267
60		97	102	107	112	118	123	128	133	138	143	148	153	158	163	168	174	179	184	189	194	199	204	209	215	220	225	230	235	240	245	250	255	261	266	271	276
61		100	106	111	116	122	127	132	137	143	148	153	158	164	169	174	180	185	190	195	201	206	211	217	222	227	232	238	243	248	254	259	264	269	275	280	285
62		104	109	115	120	126	131	136	142	147	153	158	164	169	175	180	186	191	196	202	207	213	218	224	229	235	240	246	251	256	262	267	273	278	284	289	295
63		107	113	118	124	130	135	141	146	152	158	163	169	175	180	186	191	197	203	208	214	220	225	231	237	242	248	254	259	265	270	278	282	287	293	299	304
64		110	116	122	128	134	140	145	151	157	163	169	174	180	186	192	197	204	209	215	221	227	232	238	244	250	256	262	267	273	279	285	291	296	302	308	314
65		114	120	126	132	138	144	150	156	162	168	174	180	186	192	198	204	210	216	222	228	234	240	246	252	258	264	270	276	282	288	294	300	306	312	318	324
66		118	124	130	136	142	148	155	161	167	173	179	186	192	198	204	210	216	223	229	235	241	247	253	260	266	272	278	284	291	297	303	309	315	322	328	334
67		121	127	134	140	146	153	159	166	172	178	185	191	198	204	211	217	223	230	236	242	249	255	261	268	274	280	287	293	299	306	312	319	325	331	338	344
68		125	131	138	144	151	158	164	171	177	184	190	197	203	210	216	223	230	236	243	249	256	262	269	276	282	289	295	302	308	315	322	328	335	341	348	354
69		128	135	142	149	155	162	169	176	182	189	196	203	209	216	223	230	236	243	250	257	263	270	277	284	291	297	304	311	318	324	331	338	345	351	358	365
70		132	139	146	153	160	167	174	181	188	195	202	209	216	222	229	236	243	250	257	264	271	278	285	292	299	306	313	320	327	334	341	348	355	362	369	376
71		136	143	150	157	165	172	179	186	193	200	208	215	222	229	236	243	250	257	265	272	279	286	293	301	308	315	322	329	338	343	351	358	365	372	379	386
72		140	147	154	162	169	177	184	191	199	206	213	221	228	235	242	250	258	265	272	279	287	294	302	309	316	324	331	338	346	353	361	368	375	383	390	397
73		144	151	159	166	174	182	189	197	204	212	219	227	235	242	250	257	265	272	280	288	295	302	310	316	325	333	340	348	355	363	371	378	386	393	401	408
74		148	155	163	171	179	186	194	202	210	218	225	233	241	249	256	264	272	280	287	295	303	311	319	326	334	342	350	358	365	373	381	389	396	404	412	420
75		152	160	168	176	184	192	200	208	216	224	232	240	248	256	264	272	279	287	295	303	311	319	327	335	343	351	359	367	375	383	391	399	407	415	423	431
76		156	164	172	180	189	197	205	213	221	230	238	246	254	263	271	279	287	295	304	312	320	328	336	344	353	361	369	377	385	394	402	410	418	426	435	443

Adapted from Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report.

Figure 42-3 • Body mass index table. Adapted from National Heart, Lung, and Blood Institute (NHLBI) of the National Institutes of Health (2020). Aim for a healthy weight: Body mass index table. Retrieved on 8/12/2020 at:
www.nhlbi.nih.gov/health/educational/lose_wt/BMI/bmi_tbl.pdf

Diagnostic Evaluation

Patients with obesity may have other diagnostic laboratory studies done to screen for cardiovascular diseases, such as cholesterol and triglycerides (see Chapter 23), for type 2 diabetes, such as fasting blood glucose and glycosylated hemoglobin (hemoglobin A1c) (see Chapter 46), or for nonalcoholic fatty liver disease, such as aspartate aminotransferase (AST) and alanine aminotransferase (ALT) (Orringer, Harrison, Nichani, et al., 2020) (see Chapter 43). Obesity is the most important risk factor for obstructive sleep apnea (OSA), particularly among older men (Kline, 2020); therefore, the older male patient with obesity and sleep disturbances may undergo diagnostic sleep studies (see Chapter 18).

TABLE 42-1 Body Mass Index (BMI) Classification for Overweight and Obesity

Classification	BMI Range (kg/m ²)
Overweight/pre-obese	25–29.9
Class I obesity	30–34.9
Class II obesity	35–39.9
Class III (also called “extreme” or “severe”) obesity	≥40

Adapted from Centers for Disease Control and Prevention (CDC). (2017). Defining adult overweight and obesity. Retrieved on 8/3/2019 at: www.cdc.gov/obesity/adult/defining.html

In some instances, obesity may be secondary to other diseases or disorders, such as hypothyroidism or Cushing’s syndrome (see [Chapter 45](#)). In those particular cases, the diagnostic workup follows that prescribed for the primary disease or disorder, and, when the therapeutic regimen is implemented, the patient may lose weight and the obesity may even resolve (Orringer et al., 2020).

Medical Management

Treatment of obesity generally includes lifestyle modification, pharmacologic management, and nonsurgical or surgical interventions.

Lifestyle Modification

The first approach used to treat obesity consists of lifestyle modification aimed at weight loss and then weight maintenance. The U.S. Preventive Services Task Force (USPSTF) recommends that all adults with BMIs in excess of 30 kg/m² be advised to engage in multicomponent behavioral interventions that include (Curry, Krist, Owens, et al., 2018; LeBlanc, Patnode, Webber, et al., 2018):

- Setting weight loss goals,
- Improving lifestyle behaviors (e.g., diet habits, physical activity),
- Addressing barriers to change,
- Considering use of adjunctive pharmacotherapy agents, and
- Self-monitoring and strategizing ongoing lifestyle changes aimed at a healthy weight.

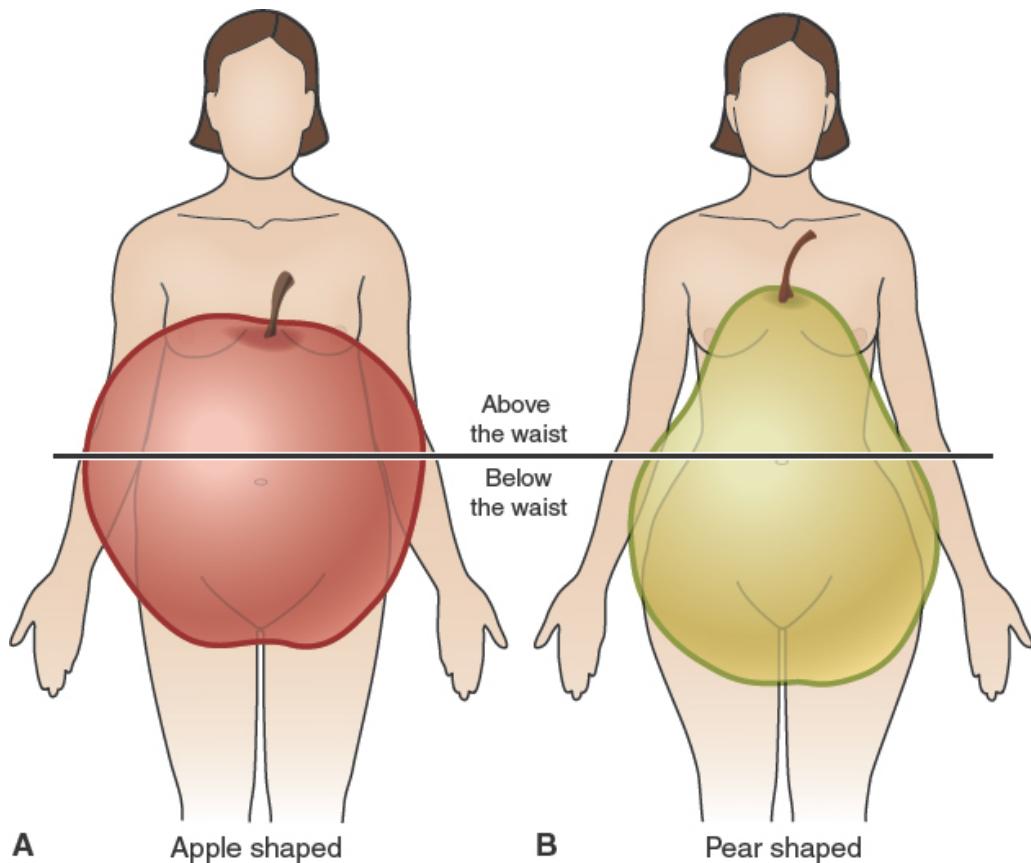


Figure 42-4 • A. Android obesity, with greater visceral/abdominal fat stores. **B.** Gynoid obesity. Reprinted with permission from Weber, J. R., & Kelley, J. H. (2018). *Health assessment in nursing* (6th ed.). Philadelphia, PA: Wolters Kluwer.

The most effective behavioral interventions are those considered high intensity; consisting of 12 to 24 sessions annually, which may include individual counseling sessions between the primary provider and patient, group nutrition education sessions, and physical activity sessions, to name a few (LeBlanc et al., 2018; Wadden, Tsai, & Tronieri, 2019). These interventions are called intensive behavioral therapy. The USPSTF notes that modest weight loss of 5% total body weight can be associated with significant clinical improvements and benefits to patients with obesity (LeBlanc et al., 2018).

A patient with obesity should be counseled to plan a caloric deficit of between 500 and 1000 calories daily from baseline, in order to achieve a 5% to 10% reduction in weight within about 6 months. This can be achieved through increasing physical activity and decreasing caloric dietary intake (Orringer et al., 2020).

Increasing physical activity through promotion of an exercise regimen is a key recommendation that can burn calories and result in weight loss. Physical activity recommendations for all adults (those with and without obesity) include at least 150 minutes of moderate-intensity aerobic exercise weekly or 75 minutes

of vigorous-intensity aerobic exercise weekly. In addition, muscle-strengthening exercises that engage all major muscle groups should be done at least twice weekly (Orringer et al., 2020). Patients with obesity who were previously sedentary and deconditioned may not be able to achieve this at the start; however, encouragement to move more and sit less along with 10 to 20 minutes of daily physical activity can result in weight loss and improved exercise tolerance (Campbell & Rutherford, 2018; DiPietro & Stachenfeld, 2017; Orringer et al., 2020).

Patients with obesity should be counseled that reducing dietary caloric intake is a necessary component of weight loss therapy. This must also include a change in dietary habits in order for weight loss to be sustained over the long term. It is important to identify current dietary patterns and typical daily caloric intake in order to recommend an appropriate diet plan. Assessing food records or 24-hour recalls or conducting dietary interviews are all effective methods to gather baseline dietary information from patients (see [Chapter 4](#): Dietary Data). There are a plethora of commercial diets that patients may select (see [Chart 42-3](#)). Regardless of the diet plan chosen, successful weight loss occurs when patients consistently make healthier dietary choices (Thorn & Lean, 2017). To date, there have not been sufficiently rigorous longitudinal studies to determine which diet plans are superior to others in terms of achieving long-term weight loss, however (Yeh, Glick-Bauer, & Katz, 2017).

Patients need not purchase commercial diet plans in order to achieve weight loss and embrace healthy diet habits. Most nutritionists advocate that healthy diets include few processed foods, sugars, and trans fats, and are heavy in plant-based foods (Yeh et al., 2017). The Dietary Approaches to Stop Hypertension (DASH) diet is an example of a superior healthy noncommercial diet. Although initially promoted to manage hypertension, the DASH diet also provides a solid foundation for achieving and maintaining weight loss due to its focus on low intake of fat and carbohydrates (Goldstein, Mayer, Graybill, et al., 2020; Thorn & Lean, 2017) (see [Chapter 27, Table 27-3](#): The DASH Diet). Another healthy diet plan that may form a basis for weight loss, though not specifically designed for this purpose, is the Mediterranean Diet (see [Chapter 23](#)). Patient education tips for managing ongoing healthy eating strategies are noted in [Chart 42-4](#).

Chart 42-3

Select Popular Commercial Diets

Atkins Diet, www.atkins.com
Jenny Craig, www.jennycraig.com
Ketogenic “keto” diet, www.dietdoctor.com/low-carb/keto
Medifast, www.mendifast1.com/index.jsp
Nutrisystem, www.nutrisystem.com/jsp_hmr/home/index.jsp
Optifast, www.optifast.com
Ornish Diet, www.ornish.com/proven-program/nutrition
South Beach Diet, www.southbeachdiet.com/home/index.jsp
WW: Weight Watchers, www.weightwatchers.com/us
Zone Diet, www.zonediet.com

Chart 42-4



PATIENT EDUCATION

Healthy Eating Strategies

The nurse instructs the patient about the healthy eating strategies outlined below.

Limit or eliminate the following:

- Processed foods with limited nutritional value (e.g., packaged cakes, cookies, chips)
- High caloric beverages (sugar-sweetened, juices, cream-enhanced)
- Fast foods
- Vending machine foods
- Foods high in sugars (e.g., candies) and saturated fats (e.g., fried foods, hot dogs)

Track the following:

- Daily food intake (food journals, diaries, smartphone, and tablet applications)
- Nutritional value and caloric content on food labels

Encourage the following:

- Reduce portions; use smaller plates and measure foods
- Schedule and plan meals and snacks in advance for each day; prepack lunches and snacks when you are out of the home (e.g., for work)
- Eat at home more often than you would eat outside the home; when eating out or ordering take-out food, avoid fried foods and choose lean meats and vegetables and salads with condiments on the side
- Eat with your family members so that together you create a healthy eating environment
- Avoid screen time when eating
- Limit exposure to food and beverage marketing
- Eat breakfast
- Limit snacks
- Eat a variety of nutritious foods; monitoring quality of foods consumed is as important as the quantity of foods consumed
- Drink plenty of water
- Stay within your daily caloric intake plan; do not become discouraged if one day you do not adhere to your plan

Adapted from Orringer, K. A., Harrison, A. V., Nichani, S. S., et al. (2020). University of Michigan Health System Clinical Alignment and Performance Excellence Guideline: Obesity prevention and management. Retrieved on 5/1/2020 at: www.med.umich.edu/1info/FHP/practiceguides/obesity/obesity.pdf

In addition to promoting healthy exercise and diet habits, ensuring healthy sleep habits is an additional lifestyle strategy that is associated with weight loss

and maintenance of a healthy weight. It is posited that sleep deprivation can cause changes in cortisol levels that promote weight gain (Orringer et al., 2020). Advising patients with sleep disturbances to plan to be in bed with lights out at least 7 hours prior to wake-up time, to create a dark, relaxing bedroom environment, to avoid activities that can cause arousal around bedtime (e.g., text messaging), and to avoid beverages with caffeine after lunchtime can all be helpful strategies aimed at ensuring a restful night's sleep that is consonant with weight reduction (Orringer et al., 2020).

Pharmacologic Therapy

Patients who are not successful at meeting weight loss goals from lifestyle modification alone may be prescribed antiobesity medications. Some patients may exhibit initial success with lifestyle modifications, but be stymied with attempts to maintain a lower BMI over the long term. These patients may also benefit from antiobesity medications. Patients who are prescribed antiobesity medications should be counseled that these prescriptions are meant to supplement, not supplant diet modification and exercise (Coulter, Rebello, & Greenway, 2018; Saunders, Umashanker, Igel, et al., 2018). Indications for antiobesity medications include a BMI greater than 30 kg/m² or a BMI greater than 27 kg/m² with concomitant morbidity that is related to being overweight (e.g., type 2 diabetes, hypertension) (Saunders et al., 2018).

Antiobesity medications work by either inhibiting GI absorption of fats, or by altering central brain receptors to enhance satiety or reduce cravings (National Institute of Diabetes and Digestive and Kidney Diseases [NIDDK], 2016b). Each of these medications has distinct side effects and contraindications; therefore, selection of these agents is individualized (see [Table 42-2](#)). The patient is monitored while taking the selected medication; if the patient does not lose at least 5% body weight after 12 weeks, the prescription may need to be changed, or the patient may be referred for other weight-reduction therapy (e.g., bariatric surgery) (NIDDK, 2016b).



Quality and Safety Nursing Alert

Antiobesity medications are believed to be teratogenic. Women with obesity who are of childbearing years should be screened carefully and advised to avoid pregnancy if they seek a prescription for an antiobesity medication.

The class of antiobesity medications known as the sympathomimetic amines are only supposed to be prescribed for short-term use (i.e., no longer than 12 weeks). They have many side effects (see [Table 42-2](#)), and patients do tend to regain weight once they are no longer taking these medications (NIDDK, 2016b).

Nonsurgical Interventions

Adult patients with obesity that does not respond to lifestyle interventions or antiobesity medications and who have either Class III/severe/extreme obesity (i.e., BMI in excess of 40 kg/m²) or Class II obesity (BMI 35 to 39.9 kg/m²) with obesity-related diseases or disorders (e.g., OSA, type 2 diabetes) may be candidates for bariatric surgical interventions (see later discussion). As an alternative, some patients may elect to pursue minimally invasive interventions, which may include vagal blocking therapy, intragastric balloon therapy, or bariatric embolization.

Vagal blocking therapy, also known as gastric stimulation, involves placement of a pacemakerlike device (vBloc™) into the subcutaneous tissue in the lateral thoracic cavity with two leads that are laparoscopically implanted at the point where the vagus nerve truncates, at the gastroesophageal junction. A pre-programmed, pulsating signal is delivered for 12 hours daily. This signal causes intermittent “blocking” of the vagus nerve. Vagal blocking results in diminished gastric contraction and emptying, limited ghrelin secretion, and diminished pancreatic enzyme secretion; these cause increased satiety, decreased cravings, and diminished absorption of calories, all of which lead to weight loss (Papasavas, El Chaar, Kothari, et al., 2016). Results from a randomized controlled trial found greater initial and 2-year sustained weight loss with participants with obesity who received vagal blocking versus control sham device implantation (Vairavamurthy, Cheskin, Kraitchman, et al., 2017). There are few adverse effects noted with use of this device, which include GI symptoms (e.g., heartburn, belching). Patients must be educated to recharge the device twice weekly for about an hour with an external coil device.

TABLE 42-2

Medications Prescribed to Treat Obesity

Medication	Adverse Effects	Nursing Considerations ^a
Gastrointestinal Lipase Inhibitor		
Mechanism of Action: Diminishes intestinal absorption and metabolism of fats, particularly triglycerides		
Orlistat <i>Note: Also available in lower dosages over-the-counter</i>	Diarrhea Flatus Oily stools Fecal incontinence	<p>Patients may have associated problems with malabsorption of nutrients; advise them to take a concomitant daily multivitamin.</p> <p>Caution in patients with known history of renal insufficiency, liver disease, or gallbladder disease as concomitant use is associated with renal calculi, liver failure, and cholelithiasis.</p> <p>Do not administer with cyclosporine.</p>
Selective Serotonin Receptor Agonist		
Mechanism of Action: Stimulates serotonin 5-HT2C receptors, causing excretion of the alpha-melanocortin-stimulating hormone (alpha-MSH) and elicits appetite suppression		
Lorcaserin	Fatigue Dizziness Nausea Headaches Cough Dry mouth Constipation	<p>Encourage patient to stay well hydrated. Can be associated with deficits in attention or memory; administer with caution in patients who drive or work with hazardous equipment when first prescribed until effects are realized.</p> <p>Can cause hypoglycemia in patients with diabetes.</p> <p>Contraindicated for patients taking antidepressants or migraine medications due to synergistic effects.</p> <p>Discontinue in patients who express suicidal ideation.</p> <p>Rarely, serotonin syndrome may develop—be alert for high fevers, brisk reflexes, agitation, and diarrhea; notify primary provider immediately and hold medication if these occur.</p>
GLP-1 Receptor Agonists		
Mechanism of Action: Mimics the effects of incretins, resulting in delayed gastric emptying, thus curbing appetite		

Liraglutide <i>Note: Also available in lower dosages to treat type 2 diabetes</i>	Nausea Diarrhea or constipation Headache Tachycardia	Must be given subcutaneously on a daily basis in the abdomen, thigh, or upper arm. Dosage increases weekly until week 5. Discontinue in patients who express suicide ideation. May be associated with pancreatitis; also associated with thyroid tumors in animal models.
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Sympathomimetic Amines

Mechanism of Action: Stimulate central noradrenergic receptors, causing appetite suppression

Phentermine Benzphetamine Diethylpropion Phendimetrazine	Palpitations and tachycardia Tremors Hypertension Dizziness Insomnia Diarrhea or constipation Mouth dryness Restlessness Alterations in taste	These are only FDA approved for short-term use (i.e., no more than 12 weeks). Contraindications include heart disease, uncontrolled hypertension, hyperthyroidism, and glaucoma. Caution patients to not drink alcohol while taking one of these medications.
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Dual Agents

Mechanism of Action: Each of these medications combines two medications with known antiobesity effects; when taken together, the effects are synergistic

Phentermine/topiramate-ER Mechanism of Action: combines the effectiveness of phentermine (see above) and topiramate, an anticonvulsant, which stimulates central GABA receptors and inhibits central glutamate receptors, suppressing appetite	Paresthesias Dizziness Insomnia Mouth dryness Alterations in taste Insomnia Constipation Tachycardia Constipation or diarrhea Nausea Vomiting Insomnia Dizziness Mouth dryness Hypertension Tachycardia	Same contraindications apply as for phentermine (see above). May need to monitor electrolytes, as hypokalemia and metabolic acidosis are possible. Monitor creatinine and watch for manifestations of renal calculi. Contraindications include uncontrolled hypertension, epilepsy, history of eating disorder such as anorexia nervosa or bulimia, and history of alcohol abuse or history of substance use disorder. Discontinue in patients who express suicidal ideation.
Naltrexone/bupropion Mechanism of action: inhibits central opioid receptors and inhibits reuptake of dopamine and norepinephrine selectively, resulting in diminished appetite and cravings		

^aAll antiobesity medications may be teratogenic; pregnancy is a contraindication to all of these agents.

FDA, U.S. Food and Drug Administration.

Adapted from Coulter, A. A., Rebello, C. J., & Greenway, F. L. (2018). Centrally acting agents for obesity: Past, Present, and Future. *Drugs*, 78(11), 1113–1132; Munoz-Mantilla, D. (2018). Top weight loss medications. Retrieved on 12/15/2019 at:

www.obesitymedicine.org/weight-loss-medications; National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK). (2016b). Prescription medications to treat overweight and obesity. Retrieved on 2/18/2020 at: www.niddk.nih.gov/health-information/weight-management/prescription-medications-treat-overweight-obesity

Intragastric balloon therapy involves endoscopic placement of a gas-filled balloon (ORBERA™) or a saline-filled dual balloon (ReShape™) into the stomach. The mechanism by which these devices result in weight loss is poorly understood, but thought to be related to increased feelings of satiety and decreased gastric emptying (Kurian, Kroh, Chand, et al., 2018). Post insertion, the intragastric balloon(s) remain in place for 3 to 6 months, and are then deflated and removed. Studies suggest greater weight loss with these than with sham therapy or with lifestyle interventions alone (Ali, Moustarah, Kim, et al., 2015; Kurian et al., 2018). Early adverse effects include complaints of nausea and vomiting, which are generally transient and do not require balloon removal. Pancreatitis and gastric or esophageal perforation may also occur although these are rare adverse events (Moore & Rosenthal, 2018). Balloon rupture can occur over the long term, however, which may lead to intestinal obstruction. In order to monitor for this serious complication, it is recommended that the balloon be impregnated with methylene blue pre-insertion so that patients with silent ruptures can report the presence of green urine to their primary providers and receive timely interventions to remove the deflated balloons before they cause obstruction. Patients who seem unlikely to return for follow-up appointments should not be candidates for intragastric balloons. Balloons should be removed within 6 months of placement; longer placement periods are associated with increased likelihood of rupture and intestinal obstruction (Ali et al., 2015).

Endovascular bariatric embolization is in the process of undergoing clinical trials to validate its safety and effectiveness. During this procedure, the gastric fundus is embolized with microspheres (beads) via the left gastric artery. The gastric fundus secretes approximately 90% of the orexigenic hormone ghrelin that is secreted in the body; therefore, it is posited that embolizing the fundus should effectively result in diminished secretion of ghrelin (Hafezi-Nejad, Bailey, Gunn, et al., 2019; Kurian et al., 2018). Preliminary results from clinical trials report an estimated weight loss ranging from 8% to 18% that is sustained for at least 1 year, without reported adverse events (Vairavamurthy et al., 2017).



Gerontologic Considerations

According to findings from the 2017–2018 United States National Health and Nutrition Examination Survey (NHANES), the prevalence of obesity for adults

60 years of age and older is 42.8%, slightly higher than the prevalence of obesity among all adults (Hales et al., 2020). Climbing rates of obesity among older adults mirrors the rate increases among all adults over the past few decades.

As adults age, lean skeletal mass decreases and adipose tissue increases. Adipose tissue does not burn calories as efficiently as lean skeletal mass; furthermore, basal metabolism drops by 2% for each additional decade of adult life. Therefore, older adults are more likely to gain weight unless they either increase activity levels or decrease their caloric intake (Eliopoulos, 2018).

Research suggests that older adults with obesity are at risk for complications that may negatively affect their quality of life. In particular, older adults with obesity may be at greater risk of falls and mobility impairments (Batsis & Zagaria, 2018; Messier, Resnik, Beavers, et al., 2018). Another study found that older adults with higher BMIs had more evidence of cognitive dysfunction compared to their normal weight counterparts (Rambod, Ghodsbin, & Moradi, 2020). More older adults with obesity are admitted to nursing homes than normal weight older adults (Zhou, Kozikowski, Pekmezaris, et al., 2017).

There is some evidence that older adults with an overweight body mass (i.e., between 25 and 29.9 kg/m²) have reasonably good health outcomes; therefore, older adults who are overweight are not necessarily counseled to lose weight (Orringer et al., 2020). However, older adults with obesity (i.e., with BMIs in excess of 30 kg/m²) should be encouraged to engage in lifestyle modifications, including diet and exercise, similar to younger adults (Orringer et al., 2020). Along with decreasing dietary caloric intake, older adult patients should be counseled that the quality of calories consumed should be the focus of the daily diet (see [Chapter 8, Fig. 8-2](#)). Fats should be limited to less than 30% of total calories, while proteins should comprise 10% to 20% of the daily diet. Intake of soluble fibers (e.g., oats, pectin) is particularly important since these can lower cholesterol levels and prevent cardiovascular diseases and cancers. Fewer than one third of older adults consume the recommended five servings of fruits and vegetables daily (Eliopoulos, 2018). Identifying barriers to consumption of these important sources of nutrients may help the older adult patient with obesity lose weight and adopt healthier eating habits. For instance, the older adult who has difficulty chewing raw fruits may enjoy fruit smoothies.

Whether or not older adult patients with obesity should be candidates for bariatric surgery (see later discussion) has been a point of ongoing debate. Research findings confirm that many older adults with obesity benefit from bariatric surgery, having outcomes and complications at rates comparable to their younger adult counterparts (Chouillard, Alsabah, Chahine, et al., 2018; Giordano & Victorzon, 2015). At present, there are no guidelines that identify the profile of which older adult patients with obesity can most benefit from bariatric surgery.



Veterans Considerations

Recruits into any branch of the U.S. military must meet prescribed height and weight requirements. Although these requirements vary slightly between branches of service, their general aim is to restrict recruitment to only applicants of normal weight. Despite having to meet height and weight standards during the recruitment process and then demonstrate the ability to meet standards of physical fitness on at least an annual basis, the rates of service members who have obesity or are overweight has reportedly tripled in the past 20 years (McCarthy, Elshaw, Szekeley, et al., 2017). The U.S. Department of Defense (DoD, 2019) reported that the overall prevalence of obesity (i.e., BMI greater than or equal to 30 kg/m²) among men and women of all ages who serve in the Army, Navy, Marine Corps, and Air Force was 17.4% in 2019, reflecting a steady annual increase since 2014. Obesity in service members can hamper their ability to functionally meet their job requirements; as such, obesity in large sectors of the military population may adversely affect overall military readiness (McCarthy et al., 2017; Shiozawa, Madsen, Banaag, et al., 2019). Furthermore, service members with obesity utilize more health service resources than those who are of normal weight and risk nonvoluntary separation from military service because they cannot meet physical fitness requirements (Shiozawa et al., 2019).

The prevalence of obesity and overweight among the nearly 21 million military veterans in the United States is estimated to be higher than that in the general civilian population, suggesting that weight gain is widespread after separation or retirement from military service (Tarlov, Zenk, Matthews, et al., 2017). Tarlov and colleagues (2017) found that 89% of all veterans (approximately 19 million veterans) lived in areas of the country where access to supermarkets, grocery stores, fitness facilities, and parks was limited, which could partially explain weight gain in veterans. Other researchers have noted that cessation of having to meet physical fitness standards, changes in daily routines, and stresses incurred during military service can also partially explain why veterans may be at greater risk of obesity (Bookwalter, Porter, Jacobson, et al., 2019). Bookwalter and colleagues (2019) prospectively studied over 28,000 veterans who separated or retired from military service and found that approximately 36% of normal weight veterans became overweight and 26% of overweight veterans had obesity within 6 years post separation. Factors that mitigated weight gains in these veterans included engaging in at least 150 minutes of moderate activity weekly, not engaging in more than 7 hours of sedentary lifestyle behaviors daily, eating fast foods less than once per week, sleeping between 7 and 9 hours nightly, not currently smoking, and drinking at least one but no more than 14 alcoholic beverages weekly for men and no more than seven alcoholic beverages weekly for women (Bookwalter et al., 2019). These findings are consonant with the tenets of the United States Veterans Administration (VA) MOVE program, whose aim is to improve the health status of veterans with obesity by encouraging physical activity, good dietary choices, and weight reduction strategies (McCarthy et al., 2017; Tarlov et al., 2017).



COVID-19 Considerations

Several risks for both severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection and pathogenesis to coronavirus disease 2019 (COVID-19) have been posed (see [Chapter 66](#)). Early epidemiologic data from China did not identify obesity as a risk for becoming infected with SARS-CoV-2. However, with the spread of the pandemic to both Europe and North America, the two continents with the highest prevalence of obesity in the world, that risk became apparent (Moriconi, Masi, Rebelos, et al., 2020). In particular, obesity seems to be a significant risk factor for younger adults requiring hospitalization to manage COVID-19 (Kass, Duggal, & Cingolani, 2020). Obesity is also associated with prolonged hospitalization, admission to critical-care units, and overall poorer outcomes in patients with COVID-19 (Kalligeros, Shehadeh, Mylona, et al., 2020; Moriconi et al., 2020; Tamara & Tahapary, 2020).

Nursing Management

Patients with obesity report feeling socially marginalized, judged, and unsupported by society at large. Many times, patients with obesity report that they suffer stigmatization by their health care providers, including nurses (Eisenberg, Noria, Grover, et al., 2019). The bias that nurses may have against patients with obesity may not directly affect their nursing care; however, indirectly, the consequences of this stigmatization can lead to poor health outcomes. Patients with obesity who feel stigmatized by their health care providers report increased depression, low self-esteem, and avoidance of health care appointments and health maintenance activities (e.g., diet and exercise) (Smigelski-Theiss et al., 2017).

The patient with obesity who requires medical-surgical nursing care, whether it is because the patient is admitted to the hospital, needs home health care, or is admitted to a transitional care setting, merits special considerations. Obesity can adversely affect the mechanics of ventilation and circulation, pharmacokinetics and pharmacodynamics, skin integrity, and body mechanics and mobility. These adverse effects are more common with higher BMIs; thus, the patient with Class III obesity (BMI in excess of 40 kg/m^2) is particularly vulnerable.

Obesity can result in anatomic remodeling, including compression of the oropharynx and increased neck circumference and chest diameter. These changes can predispose the patient with obesity to OSA (see [Chapter 18](#)), respiratory failure, and obesity hypoventilation syndrome. Obesity hypoventilation syndrome is characterized by daytime hypoventilation with hypercapnea (i.e., PaCO_2 greater than 45 mm Hg) and hypoxemia (i.e., PaO_2 less than 80 mm Hg), and sleep-disordered breathing. Potential adverse effects of obesity hypoventilation syndrome can be mitigated by maintaining the patient in the low Fowler position, which maximizes diaphragmatic chest expansion. Continuous

pulse oximetry monitoring may be advisable, as well as supplemental oxygen therapy (see [Chapter 19](#)) and frequent respiratory assessments (at least every shift). For the patient with a known diagnosis of OSA, ensuring that the patient utilizes prescribed therapy (e.g., oral appliance, continuous positive airway pressure [CPAP]) if newly hospitalized or in a different transitional care environment is important in order to ensure breathing effectiveness and avoid respiratory failure (Haesler, 2018; Holsworth & Gallagher, 2017; Petcu, 2017).

The patient with obesity may have central and peripheral circulatory compromise. Heart failure is more commonplace among patients with obesity (see [Chapter 25](#)). Hypertension is also more prevalent; the nurse must use appropriately sized blood pressure cuffs to obtain valid blood pressure readings (see [Chapter 27](#)). Peripheral blood flow can be compromised for the patient with obesity, resulting in stasis of blood flow, one of three components of the Virchow triad, which is the broad category of risk for venous thromboembolism (VTE) (see [Chapter 26](#), [Chart 26-8](#)). Peripheral circulatory compromise not only can increase the risk for VTE formation (e.g., pulmonary embolism [PE] and deep vein thrombosis [DVT]), but it can also make finding venous access difficult when the patient with obesity requires intravenous (IV) therapy. Finding appropriate venous access can be exacerbated by the presence of increased adipose tissue in the extremities. Ultrasound guidance may be required in order to successfully gain IV access and place an IV cannula in the patient with obesity (Oliver, Oliver, Ohanyan, et al., 2019).

The patient with obesity may have differences in both pharmacokinetics (i.e., the movement of drug metabolites within the body) and pharmacodynamics (i.e., how drugs are metabolized and the effects of drugs) that can affect drug dosages, drug effectiveness, and patient safety. The effectiveness of many drugs is affected by the ratio of lean skeletal muscle mass to adipose tissue. The active metabolites of many drugs are protein bound in the plasma. In patients with greater adipose tissue, more of these active metabolites can be unbound in plasma, or free, and exert greater effects. Some drugs readily bind to adipose tissue, which may either inactivate them or prolong their effects. In addition, increased adiposity can have indirect effects on metabolic pathways within the liver, resulting in changes in drug metabolic pathways, which can result in either increased or decreased drug metabolism, depending on the drug and the affected metabolic pathway. In other words, some drugs have enhanced effects while others have diminished effects with patients with obesity compared to patients of normal weight. For instance, research studies have found that patients with obesity who are critically ill with sepsis require proportionally lower dosages of IV drip norepinephrine, which is typically given based on weight-based calculations, than patients with normal weight (Droege & Ernst, 2017; Radosevich, Patanwala, & Erstad, 2016). On the other hand, patients with obesity who require opioid agents to treat pain frequently require higher dosages of opioid agents to achieve pain relief, but are more likely to have serious adverse effects of sedation and respiratory depression (Meisenberg, Ness, Rao, et

al., 2017). The nurse should be cognizant that weight-based calculations of drug dosages for patients with obesity may need to be altered, depending on the patient and the drug, and should consult with clinical pharmacologists and the patient's primary provider as needed to ensure optimal drug effectiveness and patient safety.

Patients with obesity are particularly vulnerable to developing pressure injuries (see [Chart 42-5](#)). Increased adipose tissue can diminish the supply of blood, oxygen, and nutrients to peripheral tissue. The presence of more folds in the skin is associated with more skin moisture and increased skin friction, which are pressure injury risks. Moreover, skin folds may be present in uncommon areas, including under the breasts, under the lower abdomen, within gluteal folds, and at the nape of the neck (Haesler, 2018; Williamson, 2020). In addition, patients with obesity frequently have more limitations in mobility than patients of normal weight. Immobility is another risk for pressure injury development (Williamson, 2020). Consultation with a wound-ostomy-continence (WOC) nurse may be advisable to ensure that pressure injury risks for the patient with obesity are minimized.

Chart 42-5



ETHICAL DILEMMA

When Is Prescribed Care Beneficent and When Is It Paternalistic?

Case Scenario

T.N. is a 40-year-old woman admitted to the hospital 2 days ago for treatment of nonhealing and infected stage III pressure injuries. T.N. is 64 inches tall and weighs 275 pounds; thus, her BMI is 47.2 kg/m^2 , consistent with Class III/severe obesity. T.N. has had obesity since childhood and reportedly has not been willing to listen to her family practice primary provider's repeated cautions to increased physical activity and decreased caloric dietary intake. The admitting primary provider has prescribed wound débridement, intravenous antibiotics, and a diet restricted to 1000 calories daily during her hospitalization. You are a staff nurse on the unit where T.N. has been admitted and are assigned to care for her. During oncoming shift report, you hear that staff members suspect T.N. is somehow having additional food brought into her room. During your initial assessment of T.N. she says to you "This hospital food is terrible. It tastes bad and I am not getting nearly enough to eat." The charge nurse suggests that T.N.'s visitors could be screened to discourage them from bringing T.N. outside food and to assist T.N. to adhere to the therapeutic regime to help facilitate healing.

Discussion

Patients with severe obesity are at risk for pressure injuries. Therefore, it seems reasonable to assume that T.N.'s overall health status will improve with weight reduction. Furthermore, it is common practice that patients are placed on dietary restrictions while hospitalized. For instance, patients with hypertension are typically prescribed low sodium diets. There are some bioethicists and clinicians who argue that patients should consent to such restrictions, and that placing patients on dietary restriction smacks of paternalism (i.e., "father knows best"). Others would argue that dietary restrictions are part of the holistic health care plan that benefits patients who are hospitalized.

Analysis

- Describe the ethical principles that are in conflict in this case (see [Chapter 1, Chart 1-7](#)). Does T.N. have the autonomous right to not adhere to her prescribed diet? Is it reasonable to enforce a caloric restriction on her while she is being hospitalized? Should her visitors be subjected to requests that they not bring her food, or does that breach the confidentiality of her treatment plan?
- Does placing T.N. on a calorie restricted diet during her hospitalization benefit her? If she does lose weight while she is hospitalized, might that accelerate the healing of her pressure injuries? Or might she become malnourished? Do a risk analysis. Are there more potential risks or more potential benefits entailed with this prescribed diet?
- T.N. has reportedly been resistant to changing her habits. It is not known whether or not her motivation to lose weight has ever been assessed;

yet, patients with severe obesity must be motivated to make changes in order to successfully manage their disease. Assume that her motivation is assessed and that she is motivated and ready to lose weight. What are the next steps? Admonishing her that she should adhere to the prescribed diet? Or are there other resources that might be mobilized to assist her to successfully manage her disease?

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Resources

See [Chapter 1, Chart 1-10](#) for Steps of an Ethical Analysis and Ethics Resources.

The nurse must ensure that appropriate specialty equipment is utilized as needed so that the patient with obesity who is immobilized is turned and mobilized as indicated to prevent pressure injuries. For many years, the traditional nursing protocol was that the patient who is immobilized and bedfast should be turned every 2 hours to prevent pressure injuries; that is now updated so that more frequent patient movements are encouraged, particularly in the patient with obesity (Haesler, 2018). Nurses should be familiar with and comfortable using specialized durable medical bariatric equipment (e.g., lifts, transport equipment, commodes) so that the patient with obesity receives necessary care. It is also important to enforce and implement safe patient handling protocols so that the nurse does not incur musculoskeletal injury.

The Patient Undergoing Bariatric Surgery

The term **bariatric** is derived from two Greek words meaning “weight” and “treatment.” Thus, bariatric surgery is surgery indicated to treat obesity. Surgery is typically performed after other nonsurgical attempts at weight control have failed. Insurance coverage for bariatric surgery varies widely, but most insurance companies will consider surgery as a treatment if the patient has Class III obesity or Class II obesity with a related medical condition (e.g., type 2 diabetes, OSA) (see [Table 42-1](#)) (Obesity Action Coalition [OAC], 2019a).

According to estimates by the ASMBS (2018b), the number of bariatric surgeries performed in the United States grew by nearly 30% between 2011 and 2017. Bariatric surgical procedures work by restricting a patient’s ability to eat

(restrictive procedure), interfering with ingested nutrient absorption (malabsorptive procedures), or both. The different types of bariatric surgical procedures require unique lifestyle modifications. In order to optimize their success, patients should be well informed about the specific lifestyle changes, eating habits, and bowel habits that may result from each type of procedure.

Bariatric surgery typically results in a weight loss of 10% to 35% of total body weight within 2 years postoperatively, with the majority of weight loss occurring within the first year (OAC, 2019b; Shanti & Patel, 2019). Comorbid conditions such as type 2 diabetes, hypertension, and OSA may resolve; and dyslipidemia improves (Nguyen et al., 2020; Shanti & Patel, 2019). Bariatric surgery has been extended to carefully selected adolescents with severe obesity and comorbidity because of the positive results it has achieved in adults (Ruiz-Cota, Bacardí-Gascón, & Jiménez-Cruz, 2019). However, the long-term benefits for patients who undergo bariatric surgery during adolescence are debatable, as weight regain and risk reduction of comorbidities tend to resume within 5 years postoperatively (Ruiz-Cota et al., 2019).

Patient selection is critical, and the preliminary process may necessitate months of counseling, education, and evaluation by a multidisciplinary team, including social workers, dietitians, a nurse counselor, a psychologist or psychiatrist, and a bariatric surgeon. The selection criteria for patients has changed considerably since the advent of bariatric surgery, with patients with BMIs as low as 30 kg/m² now considered candidates for surgical intervention if they have comorbid conditions that may demonstrably improve post weight loss (e.g., type 2 diabetes) (Nguyen et al., 2020; NIDDK, 2016a) (see [Chart 42-6](#)).

Because bariatric surgery involves a drastic change in the functioning of the digestive system, patients need counseling before and after the surgery. Guidelines have been developed to assist in the care of patients having bariatric surgery (Mechanick, Apovian, Brethauer, et al., 2019).

Chart 42-6

Selection Criteria for Bariatric Surgery

Patients with the Following BMIs and Associated Factors

- BMI $\geq 40 \text{ kg/m}^2$ without excessive surgical risk
- BMI $\geq 35 \text{ kg/m}^2$ and one or more severe obesity-associated comorbid conditions (e.g., hyperlipidemia, obstructive sleep apnea, obesity hypoventilation syndrome, nonalcoholic fatty liver disease, hypertension, asthma, debilitating arthritis, or considerably impaired quality of life)
- BMI $\geq 30 \text{ kg/m}^2$ with type 2 diabetes with poor glycemic control despite optimal medical treatments and lifestyle changes

Inclusion Criteria

- Ability to perform activities of daily living and self-care
- Presence of a support network of family and friends
- Failure of previous nonsurgical attempts at weight loss, including nonprofessional programs
- Expectation that patient will adhere to postoperative care, follow-up visits, and recommended medical management, including the use of dietary supplements

Exclusion Criteria

Reversible endocrine or other disorders that can cause obesity

Current substance use disorder (SUD: e.g., drug or alcohol abuse)

Uncontrolled, severe psychiatric illness

Lack of comprehension of risks, benefits, expected outcomes, alternatives, and lifestyle changes required with bariatric surgery

Adapted from Mechanick, J. I., Apovian, C., Brethauer, S., et al. (2019). Clinical practice guidelines for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient—2019 update: Cosponsored by the American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic & Bariatric Surgery. *Endocrine Practice*, 25(12), 1–75.

Surgical Procedures

Sleeve gastrectomy, Roux-en-Y gastric bypass (RYGB), biliopancreatic diversion with duodenal switch, and gastric banding are the current bariatric procedures that might be performed. These procedures may be performed by laparoscopy or by an open surgical technique. Currently, the sleeve gastrectomy is the most commonly performed procedure, followed by the RYGB; postoperative outcomes are equally favorable between sleeve gastrectomy and RYGB, and generally better than outcomes with gastric banding (ASMBS, 2018b; Kizy, Jahansouz, Downey, et al., 2017; Mechanick et al., 2019). Gastric banding tends to be done less commonly, as there have been reports of problems

with band failures and a need for more frequent patient postoperative monitoring (Tsai, Zehetner, Beel, et al., 2019). Biliopancreatic diversion with duodenal switch tends to result in the most postoperative weight loss, and is therefore more commonly indicated for patients with very high BMIs (Nguyen et al., 2020).

The RYGB is a combined restrictive and malabsorptive procedure. The gastric banding and sleeve gastrectomy are restrictive procedures, and the biliopancreatic diversion with duodenal switch combines gastric restriction with intestinal malabsorption. Figure 42-5A–D provides additional details about these procedures.

NURSING PROCESS

The Patient Undergoing Bariatric Surgery

Assessment

Preoperatively, the nurse assesses for contraindications to major abdominal surgery. Previous attempts at losing weight are also assessed, including strategies such as nutritional counseling, dieting, or exercise programs. The nurse ensures the patient has received education and counseling regarding the possible risks and benefits of bariatric surgery including the complications, postsurgical outcomes, dietary changes, and the need for lifelong follow-up. The nurse also confirms that the patient has been screened for behavioral disorders that may interfere with postsurgical outcomes. Dietary counseling is initiated preoperatively to prepare for postoperative dietary changes (Mechanick et al., 2019; OAC, 2019b).

The nurse ensures that preoperative screening tests are obtained and scrutinizes the results. Typical laboratory tests include a complete blood cell count (CBC), electrolytes, blood urea nitrogen (BUN), and creatinine. See Appendix A on **thePoint** for normal values for these laboratory tests. Patients with obesity may have OSA, gastroesophageal reflux disease (GERD), heart disease, nonalcoholic fatty liver disease, diabetes (or prediabetes), and vitamin and mineral deficiencies; thus, other screening tests that may be obtained include a sleep study, upper endoscopy, electrocardiogram (ECG), lipid panel, AST, ALT, glucose, and hemoglobin A1c, as well as iron, vitamin B₁₂, thiamine, folate, vitamin D, and calcium levels.

Postoperatively, the nurse assesses the patient to ensure that goals for recovery are met and that the patient exhibits absence of complications secondary to the surgical intervention. See **Chapter 16** for general assessment of the postoperative patient.

Diagnosis

NURSING DIAGNOSES

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

- Lack of knowledge about the nature of the surgical procedure, dietary limitations, and activities
- Anxiety associated with impending surgery
- Acute pain associated with surgical procedure
- Risk for hypovolaemia associated with nausea, gastric irritation, and pain
- Risk for infection associated with anastomotic leak
- Impaired nutritional status associated with dietary restrictions

- Disturbed body image associated with body changes from bariatric surgery

COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS

Potential complications may include the following:

- Change in bowel habits, including diarrhea and/or constipation
- Hemorrhage
- Venous thromboembolism (VTE)
- Bile reflux
- Dumping syndrome
- Dysphagia
- Bowel or gastric outlet obstruction

Planning and Goals

Preoperative goals include that the patient will become knowledgeable about the preoperative and postoperative dietary routine/restrictions and will have decreased anxiety about the surgery. Postoperative goals include relief of pain, maintenance of homeostatic fluid balance, prevention of infection, adherence to detailed diet instructions to include progression of food intake as well as fluid intake (to prevent dehydration), knowledge about vitamin supplements and the need for lifelong follow-up, achievement of a positive body image, and maintenance of normal bowel habits (ASMBS, 2013; Mechanick et al., 2019).

Nursing Interventions



Fostering Patient Knowledge

Although care of patients with obesity who are undergoing surgery is best achieved with a multidisciplinary care team, nurses have the opportunity to lead patient education initiatives. A systematic review noted that weight management centers within and outside of the United States have not extensively tested education practices for their effectiveness (Groller, 2017). Furthermore, patient education was not comparable across centers with much variance between educational content and delivery practices specific to teaching style and educator role (Groller, 2017).

Education for patients undergoing bariatric surgery should include information on the surgical procedure, nutrition requirements, activity, and psychosocial behaviors (Groller, 2017). Providing this education to patients in small group and individualized teaching sessions will enable patients to ask questions and have the nurse evaluate patient understanding to promote adherence to the treatment plan.

The nurse counsels the patient anticipating bariatric surgery to ingest nothing but clear liquids for a specified period of time preoperatively

(typically about 24 to 48 hours before). Nutritional support for patients scheduled for bariatric surgery is tailored to meet each patient's individual need to ensure optimal consumption of micronutrients. Bariatric diets usually follow a slow progression from clear liquids only continuing for up to 48 hours postoperatively, to full liquids with sugar-free or low sugar options, to pureed diet, to soft solids and, eventually by approximately the 8-week postoperative time frame, to solid foods (Mechanick et al., 2019; Petcu, 2017). This slow progression is necessary to maximize weight loss, and to prevent complications such as nausea, vomiting, bile reflux, and diarrhea.

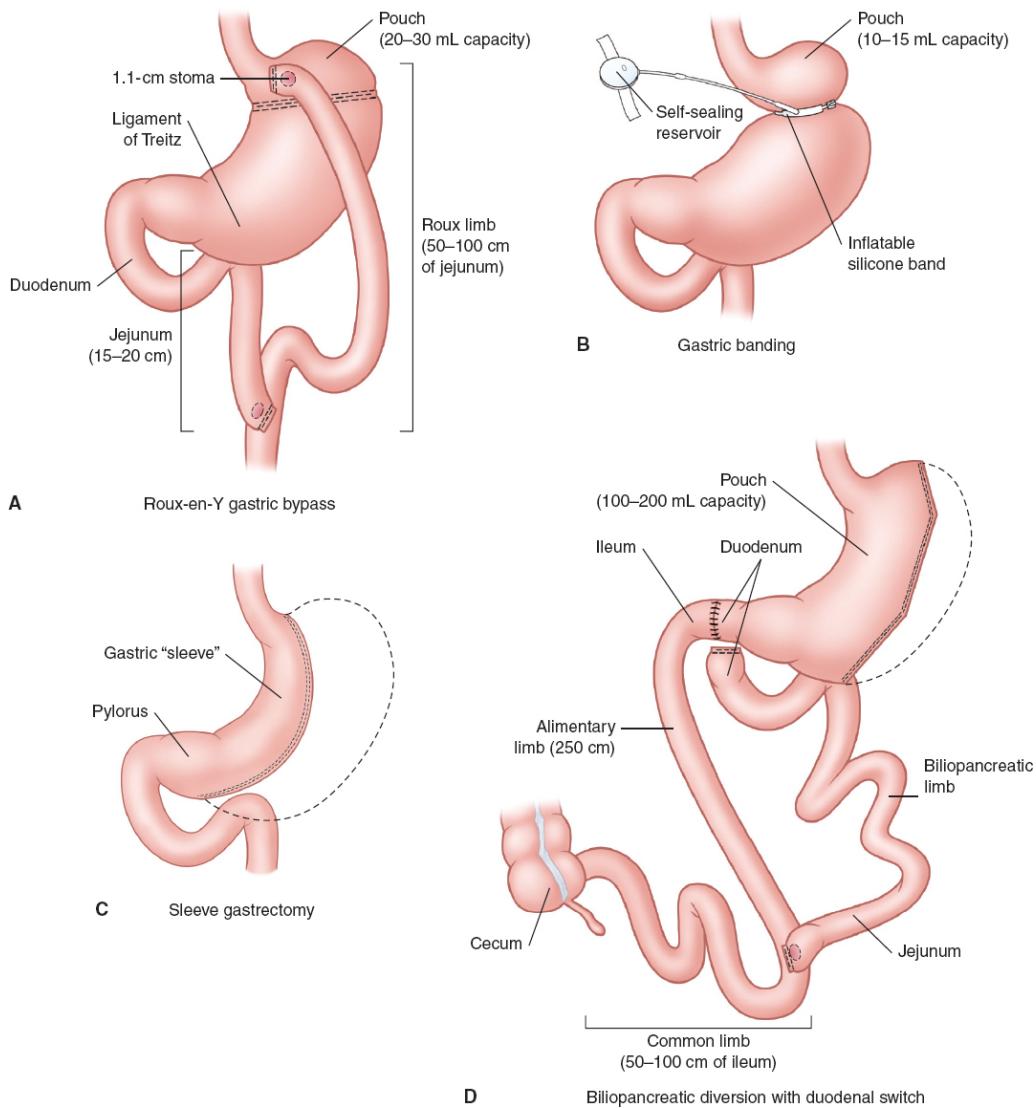


Figure 42-5 • Bariatric surgical procedures. **A.** Roux-en-Y gastric bypass. A horizontal row of staples across the fundus of the stomach creates a pouch with a capacity of 20 to 30 mL. The jejunum is divided distal to the ligament of Treitz, and the distal end is anastomosed to the new pouch. The proximal segment is anastomosed to the jejunum. **B.** Gastric banding. A prosthetic device is used to restrict oral intake by creating a small pouch of 10 to 15 mL that empties through the narrow outlet into the remainder of the stomach. **C.** Sleeve gastrectomy. The stomach is incised vertically and up to 85% of the stomach is surgically removed, leaving a “sleeve”-shaped tube that retains intact nervous innervation and does not obstruct or decrease the size of the gastric outlet. **D.** Biliopancreatic diversion with duodenal switch (also called *sleeve gastrectomy with duodenal switch*). Half of the stomach is removed, leaving a small area that holds about 60 mL. The entire jejunum is excluded from the rest of the gastrointestinal tract. The duodenum is disconnected and sealed off. The ileum is

divided above the ileocecal junction, and the distal end of the jejunum is anastomosed to the first portion of the duodenum. The distal end of the biliopancreatic limb is anastomosed to the ileum.

The patient's diet likely will be limited upon discharge from the hospital; because of this, patients scheduled for bariatric surgery are given guidelines prior to surgery on which foods and liquids they may consume postoperatively so that they may stock up on these items at home before they are admitted to the hospital. These typically include sugar-free drinks, gelatins and puddings, flavored electrolyte drinks, fat-free milk, protein drinks, sugar-free applesauce, and low-fat soups (Mechanick et al., 2019; Petcu, 2017).

REDUCING ANXIETY

The nurse provides the patient preparing for bariatric surgery anticipatory guidance as to what to expect during the surgery and postoperatively. In addition, the nurse may encourage the patient to join a bariatric surgery support group preoperatively, with the intent that the patient will continue to participate in this group postoperatively. Most bariatric surgery centers sponsor patient support groups that meet in person or online. These support groups provide a forum where patients contemplating bariatric surgery may talk with patients who have had the surgery and may provide them with guidance and tips that can help to lessen their anxiety (Mechanick et al., 2019).

RELIEVING PAIN

After surgery, analgesic agents may be given as prescribed to relieve pain and discomfort. In the past, patient-controlled analgesic pumps were used postoperatively; however, this is no longer recommended practice (Nguyen et al., 2020). The opioid crisis has stimulated research to question opioid prescription and use practices in this population (Heinberg, Pudalov, Alameddin, et al., 2019). New recommendations favor nonopioid agents and restriction to no more than 15 doses of oral opioids during any postoperative recovery (Friedman, Ghiassi, Hubbard, et al., 2019; Nguyen et al., 2020). Patients are usually prescribed oral immediate-release opioids (e.g., oxycodone) and other nonopioid agents, such as acetaminophen. The nurse should educate the patient about these medications and monitor their effectiveness. It is especially important to provide adequate pain relief so that the patient can perform pulmonary care activities (deep breathing and coughing) and leg exercises, turn from side to side, and ambulate. The nurse assesses the effectiveness of analgesic intervention and consults with other members of the health care team if pain is not adequately controlled (see [Chapter 9](#)). Positioning the patient in a low or high Fowler position promotes comfort and emptying of the stomach after any type of gastric surgery, including bariatric procedures.

ENSURING FLUID VOLUME BALANCE

Patients who have had bariatric surgery usually receive IV fluids for the first several hours postoperatively. Once they are awake and alert on the surgical unit, they are encouraged to begin intake of sugar-free oral fluids. Introducing small volumes of these liquids is believed to stimulate GI peristalsis and perfusion and thwart gastric reflux. Sugar-free fluids are preferred because they are not implicated in causing dumping syndrome (see later discussion). With a typical regimen, patients are encouraged to slowly sip 30 mL of these fluids every 15 minutes (Fencl, Walsh, & Vocke, 2015). Patients should stop ingesting fluids, however, if they feel nauseated or full. Antiemetic agents may be prescribed to relieve nausea and prevent vomiting, which could cause strain on the surgical site and cause either a hemorrhage or anastomotic leak (Mechanick et al., 2019).

PREVENTING INFECTION/ANASTOMOTIC LEAK

Disruption at the site of anastomosis (i.e., surgically resected site) may cause leakage of gastric contents into the peritoneal cavity, causing infection and possible sepsis. Patients at risk for this particular complication tend to be older, male, and with greater body mass. In addition, anastomotic leak is more commonly associated with open rather than laparoscopic procedures. Patients with anastomotic leaks typically exhibit nonspecific signs and symptoms that include fever, abdominal pain, tachycardia, and leukocytosis. This may progress to sepsis and possibly septic shock if not recognized and treated early (see [Chapter 11](#)). The nurse must be astute in recognizing these manifestations and alerting the patient's primary provider should they occur (Petcu, 2017).

A patient suspected of having an anastomotic leak may have an upper GI series that includes follow-up computed tomography (CT) scan with contrast dye, which may find leaking contrast dye, thus confirming the diagnosis. Treatment varies depending on the timing (early or late postoperatively) and severity of the leak. CT-guided drainage of the area may be appropriate for a less severe leak in the later postoperative phase of recovery, but an early or severe leak requires immediate open surgical intervention to repair the leak (Petcu, 2017).

ENSURING ADEQUATE NUTRITIONAL STATUS

After bowel sounds have returned and oral intake is resumed, six small feedings consisting of a total of 600 to 800 calories per day are provided, and consumption of fluids between meals is encouraged to prevent dehydration. The nurse instructs the patient to eat slowly and stop when feeling full. Eating too much or too fast or eating high-calorie liquids and soft foods can result in vomiting or painful esophageal distention. Gastric retention may be evidenced by abdominal distention, nausea, and vomiting. A nutritionist is typically consulted to assist with diet restrictions and diet progression (Mechanick et al., 2019; Nguyen et al., 2020) (see [Chart 42-7](#)).

Common dietary deficiencies in patients who have had bariatric surgery include malabsorption of organic iron, which may require supplementation with oral or parenteral iron, and a low serum level of vitamin B₁₂; the patient may be prescribed monthly vitamin B₁₂ intramuscular injections to prevent pernicious anemia (Holsworth & Gallagher, 2017; Mechanick et al., 2019) (see [Chapter 29](#) for further discussion of pernicious anemia).

SUPPORTING BODY IMAGE CHANGES

After bariatric surgery most patients report greatly improved perceptions of their body image, as well as improved quality of life. In particular, patients identified satisfaction with bariatric surgery when they achieved personal weight goals, adhered to postoperative care rules, and saw physical health improvements (Groller, Teel, Stegenga, et al., 2018). However, some patients report lingering dissatisfaction with their body images. In particular, some patients may report dissatisfaction related to loose skin folds and may eventually seek elective body-contouring surgical options (e.g., breast reductions, breast lifts, abdominoplasty) (Groller et al., 2018). The nurse provides support to the patient who reports dissatisfaction with body image post weight loss by acknowledging the patient's feelings as real, sharing that these perceptions are not unusual, and providing links to live or online supports groups or counselors, as necessary (see the Nursing Research Profile in [Chart 42-8](#)).

Chart 42-7



PATIENT EDUCATION

Dietary Guidelines for the Patient Who Has Had Bariatric Surgery

The nurse instructs the patient to:

- Eat smaller but more frequent meals that contain protein and fiber; each meal size should not exceed 1 cup.
- Eat only foods high in nutrients (e.g., peanut butter, cheese, chicken, fish, beans).
- Consume fat as tolerated.
- Ensure a low carbohydrate intake; in particular, avoid concentrated sources of carbohydrates (e.g., candy).
- Eat two protein snacks daily; animal protein may be poorly tolerated after Roux-en-Y gastric bypass, however.
- Eat slowly and chew thoroughly or may feel food “sticking” in throat.
- Assume a low Fowler position during mealtime and then remain in that position for 20 to 30 minutes after mealtime—this delays stomach emptying and decreases the likelihood of dumping syndrome.
- Know that antispasmodic agents, as prescribed, also may aid in delaying the emptying of the stomach.
- Avoid drinking fluid with meals; instead, consume fluids up to 30 minutes before a meal and 60 minutes after mealtime.
- Do drink plenty of water; refrain from drinking liquid calories (e.g., alcoholic beverages, fruit drinks, nondiet sodas).
- Take prescribed dietary supplements of vitamins and medium-chain triglycerides.
- Follow up with primary provider for monthly injections of vitamin B₁₂ and iron as prescribed.
- Walk for at least 30 minutes daily.

Adapted from Groller, K. D., Teel, C., Stegenga, K. H., et al. (2018). Patient perspectives about bariatric surgery unveil experiences, education, satisfaction, and recommendations for improvement. *Surgery for Obesity and Related Diseases*, 14(6), 785–796; Mechanick, J. I., Apovian, C., Brethauer S., et al. (2019). Clinical practice guidelines for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient—2019 update: Cosponsored by the American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic & Bariatric Surgery. *Endocrine Practice*, 25(12), 1–75.

MONITORING AND MANAGING POTENTIAL COMPLICATIONS

After surgery, the nurse assesses the patient for complications from the bariatric surgery, such as changes in bowel habits, hemorrhage, venous thromboembolism (VTE), bile reflux, dumping syndrome, dysphagia, and bowel or gastric outlet obstruction.

Change in Bowel Habits. Patients may complain of either diarrhea or constipation postoperatively. Diarrhea is more common an occurrence after

bariatric surgery, particularly after malabsorptive procedures (Mechanick et al., 2019). Both may be prevented if the patient consumes a nutritious diet that is high in fiber. Steatorrhea also may occur as a result of rapid gastric emptying, which prevents adequate mixing with pancreatic and biliary secretions (Mechanick et al., 2019). In mild cases, reducing the intake of fat and administering an antimotility medication (e.g., loperamide) may control symptoms. Persistent diarrhea or steatorrhea may warrant further diagnostic testing, such as an upper endoscopy or colonoscopy with biopsies to rule out the presence of additional pathology, such as celiac disease or *Clostridium difficile* infection (Mechanick et al., 2019) (see [Chapter 41](#)).

Hemorrhage. Postoperative hemorrhage may be a complication following bariatric surgery. Intra-abdominal hemorrhage may be evident by frank, bright red oral or rectal bleeding, tarry melena, bloody output from the wound or drains, if present, as well as typical clinical manifestations of severe bleeding and hemorrhagic shock (e.g., tachycardia, hypotension, syncope) (see [Chapter 11](#)). Bleeding within the first 72 hours postoperatively is most likely caused by disruption in a staple or suture. Bleeding 72 hours to 30 days postoperatively is most likely from formation of a gastric or duodenal ulcer (Nguyen et al., 2020; Petcu, 2017) (see [Chapter 40](#)).

Venous Thromboembolism. Patients who have bariatric surgery are at moderate to high risk of VTE, including both PE and DVT. Patients who are older, have higher BMIs, and have a prior history of a VTE or coagulation defect are at higher risk (Holsworth & Gallagher, 2017; Nguyen et al., 2020). ASMB guidelines for VTE prevention specify that in the immediate postoperative period, patients who have had bariatric surgery should be prescribed mechanical compression (e.g., intermittent pneumatic compression devices) and prophylactic anticoagulation with subcutaneous low-molecular-weight heparin (LMWH) agents (e.g., dalteparin, enoxaparin). The duration of time that mechanical compression and anticoagulation should continue postoperatively is not described, however, and is left to the discretion of the patient and primary provider. In addition to implementing this prescribed therapy, nurses caring for patients post bariatric surgery should encourage them to begin early ambulation to further deter the advent of VTE (Holsworth & Gallagher, 2017; Nguyen et al., 2020) (see [Chapter 26](#)).

Bile Reflux. Bile reflux may occur with procedures that manipulate or remove the pylorus, which acts as a barrier to the reflux of duodenal contents. Reflux of bile can cause gastritis or esophagitis (inflammation of the stomach or esophagus, respectively). Burning epigastric pain and vomiting of bilious material manifest this condition. Eating or vomiting does not relieve the symptoms. Bile reflux may be managed with proton pump inhibitors (e.g., omeprazole) (Nguyen et al., 2020).

Dumping Syndrome. **Dumping syndrome** is an unpleasant set of vasomotor and GI symptoms that commonly occur in patients who have had bariatric surgery. For many years, it had been theorized that the hypertonic

gastric food boluses that quickly transit into the intestines drew extracellular fluid from the circulating blood volume into the small intestines to dilute the high concentration of electrolytes and sugars, resulting in symptoms. Now, it is thought that this rapid transit of the food bolus from the stomach into the small intestines instead causes a rapid and exuberant release of metabolic peptides that are responsible for the symptoms of dumping syndrome (Mattar & Rogers, 2020).

Chart 42-8



NURSING RESEARCH PROFILE

Patient Experiences with Education and Satisfaction in Weight Loss Surgery

Groller, K. D., Teel, C., Stegenga, K. H., et al. (2018). Patient perspectives about bariatric surgery unveil experiences, education, satisfaction, and recommendations for improvement. *Surgery for Obesity and Related Diseases*, 14(6), 785–796.

Purpose

Accreditation standards require weight management centers to provide programs before and after weight loss surgery (WLS) to educate and support patients throughout lifestyle transitions. The educational programs offered at weight management centers vary by curriculum, timing, and delivery approach and may not be evidence based or patient centered. Despite these educational efforts, up to 35% of patients who undergo WLS will experience some type of weight recidivism (gain) within the first 2 postoperative years. Patient risk of regaining 5% or more from maximum weight lost continues to increase each year postoperatively. First-line treatment for patients who regain previously lost weight after WLS is to enroll and participate in additional educational programs that reinforce previously learned concepts about lifestyle demands post surgery. This study sought to obtain patient perspectives about their WLS journey with emphasis on their experiences with education, satisfaction, and recommendations to enhance the experience for future patients.

Design

This qualitative descriptive study used a purposive sampling method to recruit adult patients from an accredited weight management center. Fifty percent of all WLS cases completed within the previous 6 months were randomly selected and were mailed an invitation letter to participate in the research study. English-speaking adult patients who responded to the invitation letter were interviewed. All 11 participants, 36% male, participated in an audio-recorded interview with one researcher using a semistructured interview guide. Interview recordings were transcribed verbatim and evaluated using Colaizzi's method for inductive content analysis. Interview responses were categorized and grouped into codes, subthemes, and main themes once data saturation was achieved. Member-checking occurred to confirm final themes and subthemes that emerged from the interview data.

Findings

The study sample was fairly homogenous and oversampled males to obtain perspectives from both genders. The concept of A New Me-Version 2.0 included three main themes that emerged from participant interviews. Programming and Tools (Theme 1) provided insight on how individuals undergoing WLS obtained support from the weight management center program. Updates and Upgrades (Theme 2) explained lifestyle challenges and routines before and after WLS and quality-of-life concerns. The last theme, Lessons Learned and Future Considerations (Theme 3), described

satisfaction level through the lived experience and provided suggestions for improving the experience for future patients.

Nursing Implications

Results from this study provide insight into the lived WLS patient experience. Through emerged themes, WLS success was associated with meeting weight goals, adhering to new lifestyle routines, and seeing improved health. Participants emphasized education efforts should focus on explaining program objectives, incorporating technology to support monitoring of holistic transformations, and fostering a network of community members. This study also identified the need for future research to develop WLS education best practices and study the impact WLS education has on clinical outcomes.

Symptoms of dumping syndrome typically occur within a few minutes to 2 hours after eating and include tachycardia, dizziness, sweating, nausea, vomiting, bloating, abdominal cramping, and diarrhea (Mattar & Rogers, 2020). These symptoms typically resolve once the intestine has been evacuated (i.e., with defecation). Later, blood glucose rises rapidly, followed by increased insulin secretion. This results in a reactive hypoglycemia, which also is unpleasant for the patient. Vasomotor symptoms that occur 10 to 90 minutes after eating include pallor, perspiration, palpitations, headache, and feelings of warmth, dizziness, and even drowsiness. Anorexia may also result from dumping syndrome, because the patient may be reluctant to eat (Mattar & Rogers, 2020).

Dysphagia. **Dysphagia**, or difficulty swallowing, may occur in patients who have had any type of restrictive bariatric procedure. If it occurs, it tends to be most severe 4 to 6 weeks postoperatively and may persist for up to 6 months after surgery. Dysphagia may be prevented by educating patients to eat slowly, to chew food thoroughly, and to avoid eating tough foods such as steak or dry chicken or doughy bread. Patients with severe dysphagia who have had gastric banding may benefit from having their bands adjusted. Patients who have had other restrictive procedures may experience relief of symptoms after having stomal strictures relieved endoscopically (Mechanick et al., 2019).

Bowel and Gastric Outlet Obstruction. Bowel or gastric outlet obstruction may occur as a complication of bariatric surgery. The typical manifestations and treatments of gastric outlet obstruction are described in [Chapter 40](#); however, there is a key difference in the treatment of a patient who has undergone bariatric surgery with a gastric outlet obstruction. It is contraindicated to insert a nasogastric (NG) tube in patients that have had bariatric surgery, even if they have a gastric outlet obstruction. Alternative treatment options may include endoscopic procedures aimed at relieving the obstruction, such as balloon dilation, or surgical revisions (King & Herron, 2020).



Quality and Safety Nursing Alert

Insertion of NG tubes is contraindicated in the patient post bariatric surgery. This procedure may disrupt the surgical suture line and cause anastomotic leak or hemorrhage.

PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE

Patients are usually discharged from the hospital within 4 days postoperatively (this may be within 24 to 72 hours for patients who have had laparoscopic procedures) with detailed dietary instructions (see [Chart 42-7](#)) as well as instructions about how to either begin or resume an appropriate exercise regimen. Instructions on making follow-up appointments with the bariatric surgeon for routine postoperative visits or for complications are shared with the patient (Mechanick et al., 2019).



Educating Patients About Self-Care. The nurse provides education with the patient about nutrition, nutritional supplements, pain management, the importance of physical activity, and the symptoms of dumping syndrome and measures to prevent or minimize these symptoms. Patients who undergo laparoscopic or open RYGB procedures may have one or more Jackson–Pratt drains, which may remain in place after discharge. The nurse educates the patient or caregiver about how to empty, measure, and record the amount of drainage. Patients should be instructed to avoid taking nonsteroidal anti-inflammatory drugs (NSAIDs) (e.g., ibuprofen) post discharge, as they have been implicated in development of stomach ulcers (Mechanick et al., 2019; Peterson & Kempenich, 2020). The nurse must emphasize the continued need for follow-up (even after weight loss goals are met) and continued support group participation.

Continuing and Transitional Care. After bariatric surgery, all patients require lifelong monitoring of weight, comorbidities, metabolic and nutritional status, and dietary and activity behaviors because they are at risk for developing malnutrition or weight gain. Women of childbearing age who have bariatric surgery are advised to use contraceptives for at least 18 months after surgery to avoid pregnancy until their weight stabilizes. After weight loss, the patient may elect additional surgical interventions for body contouring. These may include breast reductions, lipoplasty to remove fat deposits, or a panniculectomy or abdominoplasty to remove excess abdominal skin folds (Mechanick et al., 2019; OAC, 2019b).

Evaluation

Expected patient outcomes may include the following:

1. Improved knowledge
 - a. Reports appropriate expectations for surgical procedure
 - b. Verbalizes understanding of diet and fluid restrictions post surgery

2. Diminished anxiety
 - a. Exhibits calm demeanor
 - b. Identifies support resources
3. Relief of pain
 - a. Reports relief of pain
 - b. Engages in early mobilization activities as prescribed
4. Maintenance of fluid balance
 - a. Able to tolerate progressive fluid intake without complaints of nausea or gastric reflux
 - b. Voids at least 400 mL in 24 hours and 0.5 mL/kg/h for any 6-hour time frame postoperatively
5. Maintenance of asepsis
 - a. No evidence of infection (e.g., no fever, no leukocytosis, no complaints of abdominal pain)
6. Achievement of nutritional balance
 - a. Able to consume small, frequent meals as prescribed
 - b. Adheres to prescribed intake of vitamins and supplements
 - c. Achieves and maintains weight reduction goals
7. Promotion of positive body image
 - a. Verbalizes continued satisfaction with weight reduction plan and its effect on body image
8. Has no complications (e.g., no diarrhea, constipation, bleeding, VTE, bile reflux, dumping syndrome, dysphagia, or bowel or gastric outlet obstruction)

CRITICAL THINKING EXERCISES

1  ebp You work as a staff nurse in a women's health clinic. A 50-year-old female patient presents for her annual physical examination. You measure her height as 65 inches and weight as 196 pounds. As you record these into her electronic health record, you note that last year she weighed 180 pounds. What is her BMI? As you reconcile her medications and allergies, she discloses how unhappy she is with regard to her increase in body weight despite her daily physical activity regimen of brisk walking or strength training. She reports symptoms of menopause. She asks, "Can you recommend a diet that will help me lose the 16 pounds of weight I gained over this year? I just want to get back to the normal me." What other information will you need to elicit from this patient in order to provide her with guidance about a healthier lifestyle? What diet may you recommend for her? Describe the strength of the evidence you use to offer her the healthiest weight loss outcomes.

2  pq A 47-year-old male patient is 6 weeks postoperative from a sleeve gastrectomy procedure. He presents to the emergency department with vomiting and dehydration. His temperature is 99.9°F (37.7°C), BP is 130/76 mm Hg, heart rate is 118 bpm, and respiratory rate is 22 breaths/min with an SpO₂ of 97%. During triage, his wife shares that he has been unable to eat for the past 3 days as he vomits soon afterward. He has not been able "keep liquids down" for the past 12 hours. Explain what nursing assessments you will conduct. What nursing interventions would you implement and why?

3  ipc You are a nurse who works at a weight management center. A 25-year-old female patient is referred by her primary care provider to visit the weight management center for treatment as she has had repeated attempts of unsuccessful weight loss for the past 18 months. She is 5 feet tall with a current weight of 192 pounds. She reports being newly married with a history of polycystic ovary syndrome (PCOS). She wishes to become pregnant. Although her last menstrual period was 3 months ago, she is not currently pregnant. What members of the interdisciplinary team could be consulted to help this woman achieve her goals of both weight loss and pregnancy?

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*Asterisk indicates nursing research.

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Resources

- American Society for Metabolic and Bariatric Surgery (ASMBS), www.asmbs.org
 Centers for Disease Control and Prevention (CDC), www.cdc.gov/obesity
 DASH Diet for Healthy Eating, www.dashdiet.org
 National Heart, Lung, and Blood Institute of the National Institutes of Health, Aim for a Healthy Weight, www.nhlbi.nih.gov/health/educational/lose_wt/index.htm
 National Institute of Diabetes and Digestive and Kidney Diseases, Weight Management, www.niddk.nih.gov/health-information/weight-management
 Obesity Action Coalition, www.obesityaction.org
 Obesity Medicine Association, www.obesitymedicine.org
 The Obesity Society (TOS), www.obesity.org
 UConn Rudd Center for Food Policy and Obesity, www.uconnruddcenter.org

43 Assessment and Management of Patients with Hepatic Disorders

LEARNING OUTCOMES

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

- 1.** Identify the metabolic functions of the liver, the pathophysiologic alterations and clinical manifestations that occur with hepatic disorders, and the significance of liver function test findings.
- 2.** Explain and demonstrate the proper techniques to perform a health history and physical assessment and discriminate between normal and abnormal findings identified in the patient with alterations of the liver.
- 3.** Relate jaundice, portal hypertension, ascites, varices, nutritional deficiencies, and hepatic encephalopathy and coma to pathophysiologic alterations of the liver.
- 4.** Describe the medical, surgical, and nursing management of patients with esophageal varices.
- 5.** Compare the various types of hepatitis and their causes, prevention, clinical manifestations, management, prognosis, and home health care needs.
- 6.** Use the nursing process as a framework for care of the patient with cirrhosis of the liver.
- 7.** Specify nonsurgical management, surgical management, and nursing care of patients with cancer of the liver and of patients undergoing liver transplantation.

NURSING CONCEPTS

Assessment
Cellular Regulation
Clotting
Comfort
Family
Infection
Metabolism
Nutrition
Patient Education
Safety

GLOSSARY

acute hepatic failure: sudden, severe onset of acute liver failure that occurs within 8 weeks after the first symptoms of jaundice (*formerly: fulminant hepatic failure*)

ascites: an albumin-rich fluid accumulation in the peritoneal cavity

asterixis: involuntary flapping movements of the hands

cirrhosis: a chronic liver disease characterized by fibrotic changes, the formation of dense connective tissue within the liver, subsequent degenerative changes, and loss of functioning cells

constructional apraxia: inability to draw figures in two or three dimensions

endoscopic variceal ligation (EVL): procedure that uses a modified endoscope loaded with an elastic rubber band passed through an overtube directly onto the varix (or varices) to be banded to ligate the area and stop bleeding (*synonym: variceal banding*)

fetor hepaticus: sweet, slightly fecal odor to the breath, presumed to be of intestinal origin

hepatic encephalopathy: central nervous system dysfunction frequently associated with elevated ammonia levels that produce changes in mental status, altered level of consciousness, and coma

jaundice: condition where the body tissues, including the sclerae and the skin, become tinged yellow or greenish-yellow, due to high bilirubin levels

orthotopic liver transplantation (OLT): grafting of a donor liver into the normal anatomic location, with removal of the diseased native liver

portal hypertension: elevated pressure in the portal circulation resulting from obstruction of venous flow into and through the liver

sclerotherapy: the injection of substances into or around esophagogastric varices to cause constriction, thickening, and hardening of the vessel and stop bleeding

Liver function is complex, and hepatic dysfunction affects all body systems. For this reason, the nurse must understand how the liver functions and must have expert clinical assessment and management skills to care for patients undergoing diagnostic and treatment procedures. The nurse also must have an understanding of technologic advances in the management of hepatic disorders. Liver disorders are common and may result from a virus, obesity, insulin resistance, or exposure to toxic substances, such as alcohol, or tumors (Norris, 2019).

ASSESSMENT OF THE LIVER

Anatomic and Physiologic Overview

The liver, the largest gland of the body and a major organ, can be considered a chemical factory that manufactures, stores, alters, and excretes a large number of substances involved in metabolism (Hammer & McPhee, 2019; Sanyal, Boyer, Terrault, et al., 2018). The location of the liver is essential because it receives nutrient-rich blood directly from the gastrointestinal (GI) tract and then either stores or transforms these nutrients into chemicals that are used elsewhere in the body for metabolic needs. The liver is especially important in the regulation of glucose and protein metabolism. The liver manufactures and secretes bile, which has a major role in the digestion and absorption of fats in the GI tract. The liver removes waste products from the bloodstream and secretes them into the bile. The bile produced by the liver is stored temporarily in the gallbladder until it is needed for digestion, at which time the gallbladder empties and bile enters the intestine (see [Fig. 43-1](#)).

Anatomy of the Liver

The liver is a large, highly vascular organ located behind the ribs in the upper right portion of the abdominal cavity. It weighs between 1200 and 1500 g in the average adult and is divided into four lobes. A thin layer of connective tissue surrounds each lobe, extending into the lobe itself and dividing the liver mass into small, functional units called *lobules* (Barrett, Barman, Brooks, et al., 2019; Hammer & McPhee, 2019).

The circulation of the blood into and out of the liver is of major importance to liver function. The blood that perfuses the liver comes from two sources. Approximately 80% of the blood supply comes from the portal vein, which drains the GI tract and is rich in nutrients but lacks oxygen. The remainder of the blood supply enters by way of the hepatic artery and is rich in oxygen. Terminal branches of these two blood vessels join to form common capillary beds, which constitute the sinusoids of the liver (see [Fig. 43-2](#)). Thus, a mixture of venous and arterial blood bathes the hepatocytes (liver cells). The sinusoids empty into venules that occupy the center of each liver lobule and are called the *central veins*. The central veins join to form the hepatic vein, which constitutes the venous drainage from the liver and empties into the inferior vena cava, close to the diaphragm (Barrett et al., 2019; Hammer & McPhee, 2019; Sanyal et al., 2018).

In addition to hepatocytes, phagocytic cells belonging to the reticuloendothelial system are present in the liver. Other organs that contain reticuloendothelial cells are the spleen, bone marrow, lymph nodes, and lungs. In the liver, these cells are called *Kupffer cells* (Barrett et al., 2019; Hammer & McPhee, 2019). As the most common phagocyte in the human body, their main function is to engulf particulate matter (e.g., bacteria) that enters the liver through the portal blood.

The smallest bile ducts, called *canaliculi*, are located between the lobules of the liver. The canaliculi receive secretions from the hepatocytes and carry them to larger bile ducts, which eventually form the hepatic duct. The hepatic duct from the liver and the cystic duct from the gallbladder join to form the common bile duct, which empties into the small intestine. The sphincter of Oddi, located at the junction where the common bile duct enters the duodenum, controls the flow of bile into the intestine.

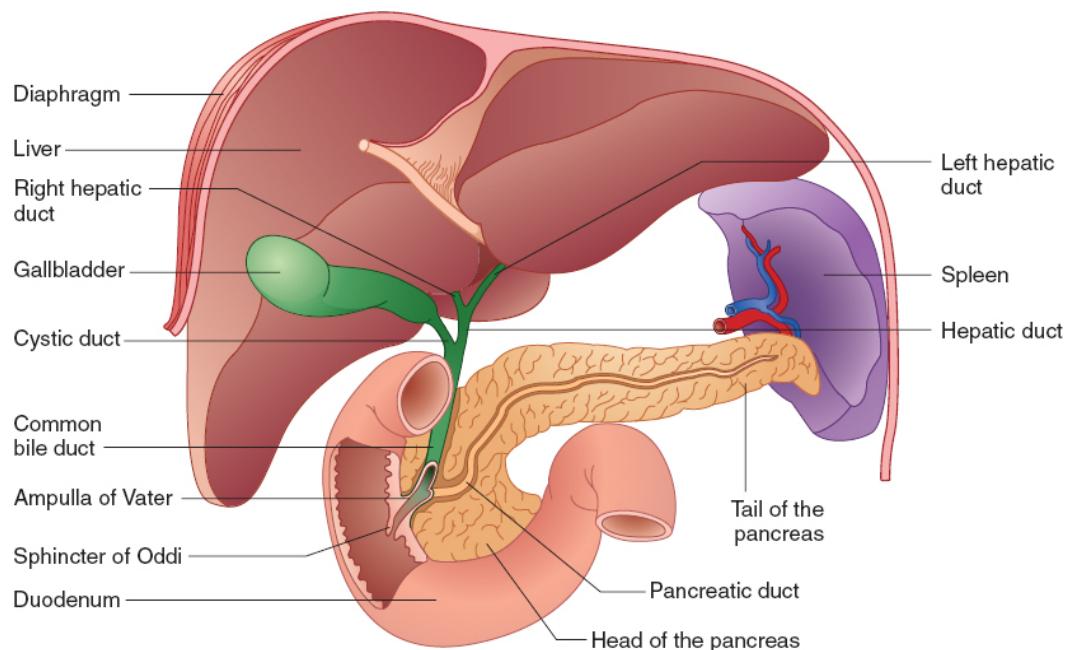


Figure 43-1 • The liver and biliary system, including the gallbladder and bile ducts. Reprinted with permission from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health states* (10th ed., Fig. 38.1). Philadelphia, PA: Wolters Kluwer.

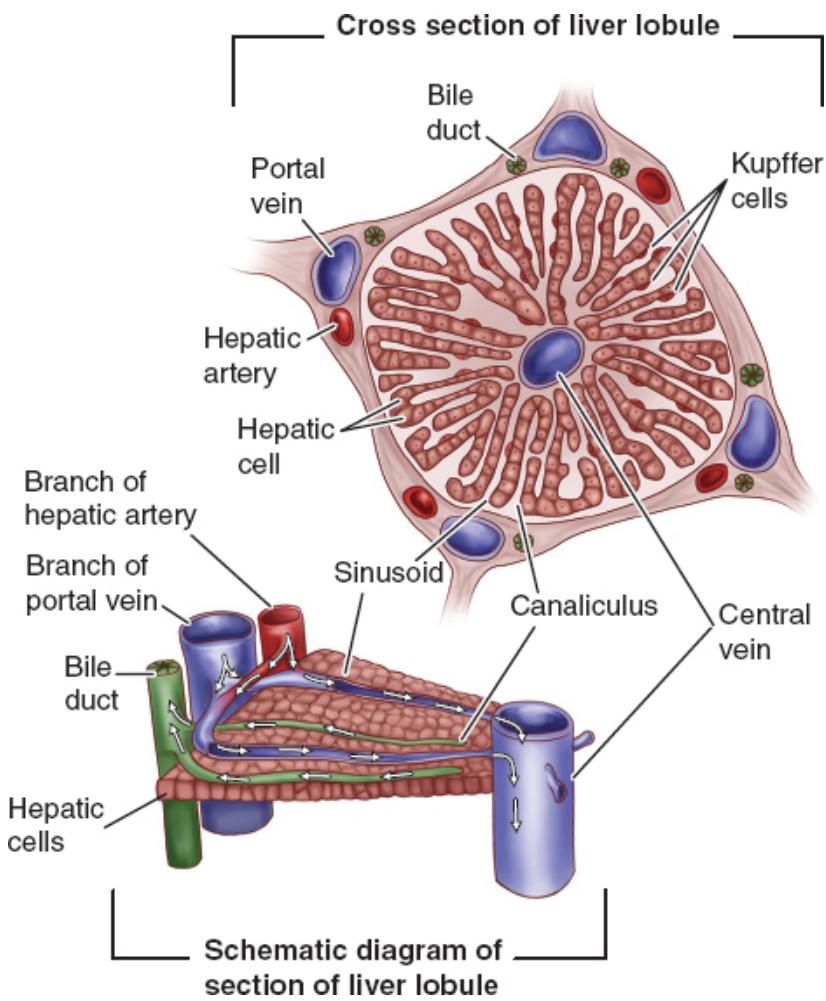


Figure 43-2 • A section of liver lobule showing the location of hepatic veins, hepatic cells, liver sinusoids, and branches of the portal vein and hepatic artery.

Functions of the Liver

Glucose Metabolism

The liver plays a major role in the metabolism of glucose and the regulation of blood glucose concentration. After a meal, glucose is taken up from the portal venous blood by the liver and converted into glycogen, which is stored in the hepatocytes. Subsequently, the glycogen is converted back to glucose through a process called glycogenolysis and is released as needed into the bloodstream to maintain normal levels of blood glucose. However, this process provides a limited amount of glucose. Additional glucose can be synthesized by the liver through a process called *gluconeogenesis*. For this process, the liver uses amino acids from protein breakdown or lactate produced by exercising

muscles. This process occurs in response to hypoglycemia (Barrett et al., 2019; Hammer & McPhee, 2019).

Ammonia Conversion

The use of amino acids from protein for gluconeogenesis results in the formation of ammonia as a by-product. The liver converts this metabolically generated ammonia into urea. Ammonia produced by bacteria in the intestines is also removed from portal blood for urea synthesis. In this way, the liver converts ammonia, a potential toxin, into urea, a compound that is excreted in the urine (Barrett et al., 2019; Hammer & McPhee, 2019).

Protein Metabolism

The liver also plays an important role in protein metabolism. It synthesizes almost all of the plasma proteins (except gamma-globulin), including albumin, alpha-globulins and beta-globulins, blood clotting factors, specific transport proteins, and most of the plasma lipoproteins. Vitamin K is required by the liver for synthesis of prothrombin and some of the other clotting factors. Amino acids are used by the liver for protein synthesis (Barrett et al., 2019; Hammer & McPhee, 2019).

Fat Metabolism

The liver is also active in fat metabolism. Fatty acids can be broken down for the production of energy and ketone bodies (acetoacetic acid, beta-hydroxybutyric acid, and acetone). Ketone bodies are small compounds that can enter the bloodstream and provide a source of energy for muscles and other tissues. Breakdown of fatty acids into ketone bodies occurs primarily when the availability of glucose for metabolism is limited, as in starvation or in uncontrolled diabetes. Fatty acids and their metabolic products are also used for the synthesis of cholesterol, lecithin, lipoproteins, and other complex lipids (Hammer & McPhee, 2019; Sanyal et al., 2018).

Vitamin and Iron Storage

Vitamins A, B, and D and several of the B-complex vitamins are stored in large amounts in the liver. Certain substances, such as iron and copper, are also stored in the liver.

Bile Formation

Bile is continuously formed by the hepatocytes and collected in the canaliculi and bile ducts. It is composed mainly of water and electrolytes such as sodium, potassium, calcium, chloride, and bicarbonate, and it also contains significant amounts of lecithin, fatty acids, cholesterol, bilirubin, and bile salts. Bile is collected and stored in the gallbladder and is emptied into the intestine as

needed for digestion. The functions of bile are excretory, as in the excretion of bilirubin; bile also serves as an aid to digestion through the emulsification of fats by bile salts.

Bile salts are synthesized by the hepatocytes from cholesterol. After conjugation or binding with amino acids (taurine and glycine), bile salts are excreted into the bile. The bile salts, together with cholesterol and lecithin, are required for emulsification of fats in the intestine, which is necessary for efficient digestion and absorption. Bile salts are then reabsorbed, primarily in the distal ileum, into portal blood for return to the liver and are again excreted into the bile. This pathway from hepatocytes to bile to intestine and back to the hepatocytes is called the *enterohepatic circulation*. Because of the enterohepatic circulation, only a small fraction of the bile salts that enter the intestine are excreted in the feces. This decreases the need for active synthesis of bile salts by the liver cells (Hammer & McPhee, 2019).

Bilirubin Excretion

Bilirubin is a pigment derived from the breakdown of hemoglobin by cells of the reticuloendothelial system, including the Kupffer cells of the liver. Hepatocytes remove bilirubin from the blood and chemically modify it through conjugation to glucuronic acid, which makes the bilirubin more soluble in aqueous solutions. The conjugated bilirubin is secreted by the hepatocytes into the adjacent bile canaliculi and is eventually carried in the bile into the duodenum.

In the small intestine, bilirubin is converted into urobilinogen, which is partially excreted in the feces and partially absorbed through the intestinal mucosa into the portal blood. Much of this reabsorbed urobilinogen is removed by the hepatocytes and secreted into the bile once again (enterohepatic circulation). Some of the urobilinogen enters the systemic circulation and is excreted by the kidneys in the urine. Elimination of bilirubin in the bile represents the major route of its excretion.

Drug Metabolism

The liver metabolizes many medications, such as barbiturates, opioids, sedatives, anesthetics, and amphetamines (Goldman & Schafer, 2019; Hammer & McPhee, 2019; Sanyal et al., 2018). Metabolism generally results in drug inactivation, although activation may also occur. One of the important pathways for medication metabolism involves conjugation (binding) of the medication with a variety of compounds, such as glucuronic acid or acetic acid, to form more soluble substances. These substances may be excreted in the feces or urine, similar to bilirubin excretion. Bioavailability is the fraction of the given medication that actually reaches the systemic circulation. The bioavailability of an oral medication (absorbed from the GI tract) can be

decreased if the medication is metabolized to a great extent by the liver before it reaches the systemic circulation; this is known as first-pass effect. Some medications have such a large first-pass effect that their use is essentially limited to the parenteral route, or oral doses must be substantially larger than parenteral doses to achieve the same effect.

Gerontologic Considerations

Chart 43-1 summarizes age-related changes in the liver. In the older adult, the most common change in the liver is a decrease in size and weight, accompanied by a decrease in total hepatic blood flow. However, in general, these decreases are proportional to the decreases in body size and weight seen in normal aging. Results of liver function tests do not normally change with age; abnormal results in older patients indicate abnormal liver function and are not a result of the aging process itself.

Chart 43-1



Age-Related Changes of the Hepatobiliary System

- Atypical clinical presentation of biliary disease
- Decreases in the following:
 - Clearance of hepatitis B surface antigen
 - Drug metabolism and clearance capabilities
 - Intestinal and portal vein blood flow
 - Gallbladder contraction after a meal
 - Rate of replacement and or repair of liver cells after injury
 - Size and weight of the liver, particularly in women
- Increased prevalence of gallstones due to the increase in cholesterol secretion in bile
- More rapid progression of hepatitis C infection and lower response rate to therapy
- More severe complications of biliary tract disease

Adapted from Townsend, C. M., Beauchamp, R. D., Evers, B. M., et al. (2016). *Sabiston's textbook of surgery: The biological basis of modern surgical practice*. Philadelphia, PA: Elsevier.

Metabolism of medications by the liver decreases in the older adult, but such changes are usually accompanied by changes in intestinal absorption, renal excretion, and altered body distribution of some medications secondary to changes in fat deposition. These alterations necessitate careful medication

administration and monitoring; if appropriate, reduced dosages may be needed to prevent medication toxicity.

Assessment

Health History

If liver function test results are abnormal, the patient is evaluated for liver disease. In such cases, the health history focuses on previous exposure of the patient to hepatotoxic substances or infectious agents. The patient's occupational, recreational, and travel history may assist in identifying exposure to hepatotoxins (e.g., industrial chemicals, other toxins). The patient's history of alcohol and drug use, including but not limited to the use of intravenous (IV) or injection drugs, provides additional information about exposure to toxins and infectious agents. Many medications (including acetaminophen, ketoconazole, and valproic acid) are responsible for hepatic dysfunction and disease (Friedman & Martin, 2018). A thorough medication history should address all current and past prescription medications, over-the-counter (OTC) medications, herbal remedies, illicit drugs, and dietary supplements.

Lifestyle behaviors that increase the risk of exposure to infectious agents are identified. IV or injection drug use, sexual practices, and foreign travel are all potential risk factors for liver disease. The amount and type of alcohol consumed are identified using screening tools (questionnaires) that have been developed for this purpose (see [Chapter 4](#)). The amount of alcohol required to produce chronic liver disease varies widely, but men who consume 60 to 80 g/day of alcohol (approximately four glasses of beer, wine, or mixed drinks) and women whose alcohol intake is 40 to 60 g/day are considered at high risk for cirrhosis. **Cirrhosis** is a chronic liver disorder characterized by fibrotic changes, the formation of dense connective tissue within the liver, subsequent degenerative changes, and loss of functional liver tissue (Barrett et al., 2019; Sanyal et al., 2018).

The history also includes an evaluation of the patient's past medical history to identify risk factors for the development of liver disease. Current and past medical conditions, including those of a psychological or psychiatric nature, are identified. The family history includes questions about familial liver disorders that may have their origin in alcohol abuse or gallstone disease, as well as other familial or genetic disorders (see [Chart 43-2](#)).

The history also addresses symptoms that suggest liver disease. Symptoms that may have their origin in liver disease but are not specific to hepatic dysfunction include jaundice, malaise, weakness, fatigue, pruritus, abdominal pain, fever, anorexia, weight gain, edema, increasing abdominal girth, hematemesis, melena, hematchezia (passage of bloody stools), easy bruising,

changes in mental acuity, personality changes, sleep disturbances, and decreased libido in men and secondary amenorrhea in women.

Chart 43-2



GENETICS IN NURSING PRACTICE

Hepatic Disorders

A number of hepatic disorders have an underlying genetic cause. However, other genetic disorders associated with metabolic, gastrointestinal, or bleeding disorders will also impact the function of the liver. Some examples of hepatic disorders caused by genetic abnormalities include:

Autosomal Dominant:

- Alagille syndrome
- Hereditary coproporphyria
- Polycystic liver disease

Autosomal Recessive:

- Crigler–Najjar syndrome
- Dublin–Johnson syndrome
- Hemochromatosis
- Progressive familial intrahepatic cholestasis
- Thalassemia
- Wilson disease

Inheritance Pattern is not distinct; however, there is a genetic predisposition for the disorder:

- Biliary atresia
- Gilbert syndrome

Other genetic disorders that impact the hepatic system:

- Alpha-1 antitrypsin deficiency
- Cystic fibrosis
- Glycogen storage disease
- Lysosomal storage disease
- Polycystic kidney disease
- Zellweger syndrome

Nursing Assessments

Refer to [Chapter 4, Chart 4-2: Genetics in Nursing Practice: Genetic Aspects of Health Assessment](#)

Family History Assessment Related to Hepatic Disorders

- Collect family history for three generations of maternal and paternal relatives in the patient's family
- Assess family history for relatives with early-onset hepatic disease

Patient Assessment Related to Genetic Hepatic Disorders

- Assess for physical signs or history of the following:

- Abdominal bloating, and constipation
- Changes to skin color or yellow hue to sclera
- Enlarged liver, spleen, or abdomen
- Episodes of nausea and vomiting
- Hemorrhoids, esophageal varices, or gallstones
- Intolerance to fatty foods or alcohol
- Pale-colored stools
- Presence and frequency of indigestion or reflux
- Unexplained weight loss
- Assess for associated nervous system disorders such as depression and mood changes especially anger and irritability (Wilson disease).
- Assess for associated blood sugar problems such as hypoglycemia
- Inquire about and assess for abnormal bleeding or bruising
- Obtain and review laboratory values: Liver function tests, ammonia, bilirubin, fat soluble vitamins (e.g., vitamins A, D, E, K)

Genetics Resources

American Liver Foundation, www.liverfoundation.org

See [Chapter 6, Chart 6-7](#) for components of genetic counseling.

Physical Assessment

The nurse assesses the patient for physical signs that may occur with liver dysfunction, including the pallor often seen with chronic illness and jaundice. The skin, mucosa, and sclerae are inspected for jaundice, and the extremities are assessed for muscle atrophy, edema, and skin excoriation secondary to scratching. The nurse observes the skin for petechiae or ecchymotic areas (bruises), spider angiomas (see [Fig. 43-3](#)), and palmar erythema. The male patient is assessed for unilateral or bilateral gynecomastia and testicular atrophy due to hormonal changes. The patient's cognitive status (recall, memory, abstract thinking) and neurologic status are assessed. The nurse observes for general tremor, **asterixis** (involuntary flapping movements of the hands), weakness, and slurred speech. These symptoms are discussed later.



In some conditions, lipids may accumulate in the hepatocytes, resulting in the abnormal condition called *fatty liver disease*. If unrelated to alcohol, this disease is referred to as nonalcoholic fatty liver disease (NAFLD). A condition known as nonalcoholic steatohepatitis (NASH) represents a more serious condition within the broad spectrum of NAFLDs and may result in damage, fibrotic changes in the liver, and cirrhosis (Hammer & McPhee, 2019).

NAFLD and NASH are two diseases within the spectrum of fatty liver disease to fibrosis and cirrhosis that are strongly associated with obesity (Barrett et al., 2019). Some studies suggest that being overweight and drinking

too much alcohol can cause severe harm to the liver. People who are overweight and people with obesity who are heavy drinkers have a significantly increased risk of developing and dying of chronic liver disease (Bellentani, 2017; Friedman & Martin, 2018; Schiff, Maddrey, & Reddy, 2018). Studies have also identified that there is an increased risk of liver cancer in people with alcoholic cirrhosis who also have fatty liver disease, type 2 diabetes, and are overweight or have obesity (Bellentani, 2017; Schiff et al., 2018). In patients who are overweight, have obesity, or have high alcohol intake, the nurse observes for signs of associated liver dysfunction.



Figure 43-3 • Spider angioma. This vascular (arterial) spider appears on the skin. Beneath the elevated center and radiating branches, the blood vessels are looped and tortuous.



Figure 43-4 • Technique for palpating the liver. The examiner places one hand under the right lower rib cage and presses downward during inspiration with light pressure with the other hand. Reprinted with permission from Bickley, L. S. (2017). *Bates' guide to physical examination and history taking* (12th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

The nurse assesses for the presence of an abdominal fluid wave (discussed later). The abdomen is palpated to assess liver size and to detect any tenderness over the liver. The liver may be palpable in the right upper quadrant. A palpable liver presents as a firm, sharp ridge with a smooth surface (see Fig. 43-4). The nurse estimates the size of the liver by percussing its upper and lower borders. If the liver is not palpable but tenderness is suspected, tapping the lower right thorax briskly may elicit tenderness. For comparison, the nurse then performs a similar maneuver on the left lower thorax.

If the liver is palpable, the nurse notes and records its size, its consistency, any tenderness, and whether its outline is regular or irregular. If the liver is enlarged, the degree to which it descends below the right costal margin is recorded to provide some indication of its size. The nurse determines whether the liver's edge is sharp and smooth or blunt and whether the enlarged liver is nodular or smooth. The liver of a patient with cirrhosis is small and hard in late-stage cirrhosis, whereas the liver of a patient with acute hepatitis is soft and the hand easily moves the edge.

Tenderness of the liver indicates recent acute enlargement with consequent stretching of the liver capsule. The absence of tenderness may imply that the enlargement is of long-standing duration. The liver of a patient with viral hepatitis is tender, whereas that of a patient with alcoholic hepatitis is not.

Enlargement of the liver is an abnormal finding that requires evaluation (Hammer & McPhee, 2019).

Diagnostic Evaluation

A wide range of diagnostic studies may be performed in patients with hepatic disorders. The nurse should educate the patient about the purpose, what to expect, and any possible side effects related to these examinations prior to testing. The nurse should note trends in results because they provide information about disease progression as well as the patient's response to therapy.

Liver Function Tests

More than 70% of the parenchyma of the liver may be damaged before liver function test results become abnormal. Function is generally measured in terms of serum enzyme activity (i.e., serum aminotransferases, alkaline phosphatase, lactic dehydrogenase) and serum concentrations of proteins (albumin and globulins), bilirubin, ammonia, clotting factors, and lipids (Hammer & McPhee, 2019; Mansour & McPherson, 2018; Sanyal et al., 2018; Wendon, Cordoba, Dhawan, et al., 2017). Several of these tests may be helpful for assessing patients with liver disease. However, the nature and extent of hepatic dysfunction cannot be determined by these tests alone, because other disorders can affect test results.

Serum aminotransferases are sensitive indicators of injury to the liver cells and are useful in detecting acute liver disease such as hepatitis. Alanine aminotransferase (ALT), aspartate aminotransferase (AST), and gamma-glutamyl transferase (GGT) (also called gamma-glutamyl transpeptidase [GGTP]) are the most frequently used tests of liver damage (Friedman & Martin, 2018; Maher & Schreibman, 2018; Schiff et al., 2018). ALT levels increase primarily in liver disorders and may be used to monitor the course of hepatitis or cirrhosis or the effects of treatments that may be toxic to the liver. AST is present in tissues that have high metabolic activity; therefore, the level may be increased if there is damage to or death of tissues of organs such as the heart, liver, skeletal muscle, and kidney. Although not specific to liver disease, levels of AST may be increased in cirrhosis, hepatitis, and liver cancer. Increased GGT levels are associated with cholestasis but can also be due to alcoholic liver disease. Although the kidney has the highest level of the enzyme, the liver is considered the source of normal serum activity. The test determines liver cell dysfunction and is a sensitive indicator of cholestasis. Its main value in liver disease is confirming the hepatic origin of an elevated alkaline phosphatase level. Common liver function tests are summarized in

Table 43-1, additional laboratory values can be found in Appendix A on **thePoint**.

Liver Biopsy

Liver biopsy is the removal of a small amount of liver tissue, usually through needle aspiration. It permits examination of liver cells. The most common indication is to evaluate diffuse disorders of the parenchyma and to diagnose space-occupying lesions. Liver biopsy is especially useful when clinical findings and laboratory tests are not diagnostic. Though rare due to the radiologic guidance now available, peritonitis caused by blood or bile after liver biopsy is the most common complication; therefore, coagulation studies are obtained, their values are noted, and abnormal results are treated before liver biopsy is performed (Schiff et al., 2018). Other techniques for liver biopsy are preferred if **ascites** (an accumulation of albumin-rich fluid in the peritoneal cavity) or coagulation abnormalities exist. A liver biopsy can be performed percutaneously with ultrasound guidance or transvenously through the right internal jugular vein to right hepatic vein under fluoroscopic control. Liver biopsy can also be performed laparoscopically.



For the procedural guidelines for assisting with percutaneous liver biopsy, go to thepoint.lww.com/Brunner15e.

TABLE 43-1 Common Laboratory Tests to Assess Liver Function

Test	Normal	Clinical Functions
Pigment Studies		
Serum bilirubin, direct	0.1–0.4 mg/dL	
Serum bilirubin, total	(1.7–3.7 mcmol/L)	These studies measure the ability of the liver to conjugate and excrete bilirubin. Results are abnormal in liver and biliary tract disease and are associated with jaundice clinically.
Urine bilirubin		
Urine urobilinogen	0.3–1 mg/dL (5– 17 mcmol/L)	
Fecal urobilinogen (infrequently used)	<0.25 mg/24 h (<0.42 mcmol/24 h) (Urine urobilinogen) 0.05–2.5 mg/24 h (0.5– 4 Ehrlich U/24 h) (Fecal urobilinogen) 50–300 mg/24 h (100–400 Ehrlich U/100 g)	
Protein Studies		
Total serum protein	7–7.5 g/dL (70– 75 g/L)	Proteins are manufactured by the liver. Their levels may be affected in a variety of liver impairments: albumin is affected in cirrhosis, chronic hepatitis, edema; and ascites; globulins are affected in cirrhosis, liver disease, chronic obstructive jaundice, and viral hepatitis.
Serum albumin	3.5–5.2 g/dL	
Serum globulin	(35–52 g/L)	
Serum protein electrophoresis	2.3–3.5 g/dL	
Albumin	(23–35 g/L)	
α_1 -Globulin	3.5–5.2 g/dL	
α_2 -Globulin	(35–52 g/L)	A/G ratio is reversed in chronic liver disease (decreased albumin and increased globulin).
β -Globulin	0.1–0.3 g/dL (1– 3 g/L)	
γ -Globulin	0.6–1 g/dL (6–10 g/L)	
A/G ratio	0.5–1 g/dL (5–10 g/L)	
	0.6–1.3 g/dL (6– 13 g/L)	
	A > G or 1.5:1– 2.5:1	
Prothrombin Time/International Normalized Ratio (PT/INR)	100% or 11–13 s/the INR is a calculation based on results of the PT. INR levels	Prothrombin time and INR may be prolonged in liver disease. It is an indicator of synthetic hepatic function. It will not return to normal with vitamin K in severe liver cell damage.

	<1.1 are considered normal	
Serum Alkaline Phosphatase	Varies with method: <i>Adults:</i> 52–142 U/L	Serum alkaline phosphatase is manufactured in bones, liver, kidneys, and intestine and excreted through biliary tract. In the absence of bone disease, it is a sensitive measure of biliary tract obstruction. Results may vary because this test is temperature and lab method dependent.
Serum Aminotransferase Studies		
AST	10–40 U/mL	The studies are based on release of enzymes from damaged liver cells. These enzymes are elevated in liver cell damage. Normal values may differ in men and women.
ALT	(0.34–0.68 U/L)	
	8–40 U/mL (0.14–0.68 U/L)	
GGT, GGTP	0–30 U/L IU/L	Values are elevated in alcohol abuse and markers for biliary cholestasis.
LDH	100–200 units (100–225 U/L)	
Ammonia (plasma)	15–45 mcg/dL (11–32 mcmmol/L)	Liver converts ammonia to urea. Ammonia level rises in liver failure.
Cholesterol		
Ester	60–70% of total cholesterol, fraction of total	Cholesterol levels are elevated in biliary obstruction and decreased in parenchymal liver disease.
HDL		
LDL	0.60–0.70 <i>Male:</i> 35–70 mg/dL; <i>Female:</i> 35–85 mg/dL <130 mcg/dL	

A/G, albumin/globulin; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GGT, gamma-glutamyl transferase; GGTP, gamma-glutamyl transpeptidase; HDL, high-density lipoprotein; LDH, lactate dehydrogenase; LDL, low-density lipoprotein.

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

Other Diagnostic Tests

Ultrasonography, computed tomography (CT) scans, and magnetic resonance imaging (MRI) are used to identify normal structures and abnormalities of the liver and biliary tree. A radioisotope liver scan may be performed to assess liver size, blood flow, and obstruction. Noninvasive liver stiffness measurements or elastography uses ultrasound-based vibration and scanning to identify liver fibrosis and determine its extent. Magnetic resonance elastography uses mechanical shear waves to identify stiff tissue (Schiff et al., 2018). Liver fibrosis and other liver diseases can be identified, evaluated and monitored with a variety of other noninvasive studies. These studies may reduce the need for liver biopsy (Friedman & Martin, 2018; Schiff et al., 2018).

Laparoscopy (insertion of a fiberoptic endoscope through a small abdominal incision) is used to examine the liver and other pelvic structures. It is also used to perform guided liver biopsy, to determine the cause of ascites, and to diagnose and stage tumors of the liver and other abdominal organs.

MANIFESTATIONS OF HEPATIC DYSFUNCTION

Hepatic dysfunction results from damage to the liver's parenchymal cells, directly from primary liver diseases, or indirectly from either obstruction of bile flow or derangements of hepatic circulation. Liver dysfunction may be acute or chronic; the latter is far more common.

Chronic liver disease, including cirrhosis, is the 12th leading cause of death in the United States among young and middle-aged adults (Schiff et al., 2018). At least 40% of those deaths are associated with alcohol use. The rate of chronic liver disease for men is twice that for women, and chronic liver disease is more common in Asian and African countries than it is in Europe and the United States. Compensated cirrhosis, in which the damaged liver is still able to perform normal functions, often goes undetected for extended periods, and as many as 1% of people may have subclinical or compensated cirrhosis (Bope & Kellerman, 2018; Schiff et al., 2018). Approximately 80% of patients diagnosed with cirrhosis compensate and remain asymptomatic for the next 10 years (Schiff et al., 2018).

Disease processes that lead to hepatocellular dysfunction may be caused by infectious agents such as bacteria and viruses and by anoxia, metabolic disorders, toxins and medications, nutritional deficiencies, and hypersensitivity states. The most common cause of parenchymal damage is malnutrition, especially that related to alcoholism (Moon, Singal, & Tapper, 2018). It is important to remember that even patients who are overweight or have obesity may suffer from not only malnutrition related to liver disease, but also from sarcopenia, the significant loss of muscle tissue. Sarcopenia is associated with

increased morbidity and mortality in patients with end-stage liver disease (ESLD) (Aby & Saab, 2019).

The parenchymal cells respond to most noxious agents by replacing glycogen with lipids, producing fatty infiltration with or without cell death or necrosis. This is commonly associated with inflammatory cell infiltration and growth of fibrous tissue. Cell regeneration can occur if the disease process is not too toxic to the cells. The result of chronic parenchymal disease is the shrunken, fibrotic liver seen in cirrhosis.

The consequences of liver disease are numerous and varied. Their ultimate effects are often incapacitating or life-threatening, and their presence is ominous. Among the most common and significant manifestations of liver disease are jaundice, portal hypertension, ascites and varices, nutritional deficiencies (resulting from the inability of damaged liver cells to metabolize certain vitamins), and **hepatic encephalopathy** or coma.

Jaundice

The bilirubin concentration in the blood may be increased in the presence of liver disease, if the flow of bile is impeded (e.g., by gallstones in the bile ducts), or if there is excessive destruction of red blood cells. With bile duct obstruction, bilirubin does not enter the intestine; as a consequence, urobilinogen is absent from the urine and decreased in the stool (Hammer & McPhee, 2019).

When the bilirubin concentration in the blood is abnormally elevated, all of the body tissues, including the sclerae and the skin, become tinged yellow or greenish-yellow, a condition known as **jaundice**. Jaundice becomes clinically evident when the serum bilirubin level exceeds 2.0 mg/dL (34 mmol/L) (Hammer & McPhee, 2019). Increased serum bilirubin levels and jaundice may result from impairment of hepatic uptake, conjugation of bilirubin, or excretion of bilirubin into the biliary system. There are several types of jaundice: hemolytic, hepatocellular, and obstructive jaundice, and jaundice due to hereditary hyperbilirubinemia. Hepatocellular and obstructive jaundice are the two types commonly associated with liver disease.

Hemolytic Jaundice

Hemolytic jaundice is the result of an increased destruction of the red blood cells; the effect is that the plasma is rapidly flooded with bilirubin so that the liver, although functioning normally, cannot excrete the bilirubin as quickly as it is formed. This type of jaundice is encountered in patients with hemolytic transfusion reactions and other hemolytic disorders. In these patients, the bilirubin in the blood is predominantly unconjugated or free. Fecal and urine

urobilinogen levels are increased, but the urine is free of bilirubin. Patients with this type of jaundice, unless their hyperbilirubinemia is extreme, do not experience symptoms or complications as a result of the jaundice per se. However, prolonged jaundice, even if mild, predisposes to the formation of pigment stones in the gallbladder, and extremely severe jaundice (levels of free bilirubin exceeding 20 to 25 mg/dL) poses a risk for central nervous system effects (Goldman & Schafer, 2019).

Hepatocellular Jaundice

Hepatocellular jaundice is caused by the inability of damaged liver cells to clear normal amounts of bilirubin from the blood. The cellular damage may be caused by hepatitis viruses, other viruses that affect the liver (e.g., yellow fever virus, Epstein–Barr virus), chemical toxins (e.g., carbon tetrachloride, phosphorus, arsenicals, certain medications), or alcohol. Cirrhosis of the liver is a form of hepatocellular disease that may produce jaundice. It is usually associated with excessive alcohol intake, but it may also be a late result of liver cell necrosis caused by viral infection. In prolonged obstructive jaundice, cell damage eventually develops, and both types of jaundice (i.e., obstructive and hepatocellular jaundice) appear together.

Patients with hepatocellular jaundice may be mildly or severely ill, with lack of appetite, nausea, malaise, fatigue, weakness, and possible weight loss. In some cases of hepatocellular disease, jaundice may not be obvious. The serum bilirubin concentration and the urine urobilinogen level may be elevated. In addition, AST and ALT levels may be increased, indicating cellular necrosis. The patient may report headache, chills, and fever if the cause is infectious. Depending on the cause and extent of the liver cell damage, hepatocellular jaundice may be completely reversible.

Obstructive Jaundice

Obstructive jaundice resulting from extrahepatic obstruction may be caused by occlusion of the bile duct from a gallstone, an inflammatory process, a tumor, or pressure from an enlarged organ (e.g., liver, gallbladder). The obstruction may also involve the small bile ducts within the liver (i.e., intrahepatic obstruction); this may be caused, for example, by pressure on these channels from inflammatory swelling of the liver or by an inflammatory exudate within the ducts themselves. Intrahepatic obstruction resulting from stasis and inspissation (thickening) of bile within the canaliculi may occur after the ingestion of certain medications, which are referred to as cholestatic agents. These include phenothiazines, antithyroid medications, sulfonylureas, tricyclic antidepressant agents, nitrofurantoin, androgens and estrogens, and some antibiotics.

Regardless of whether the obstruction is intra- or extrahepatic, and regardless of its cause, bile cannot flow normally into the intestine and becomes backed up into the liver. It is then reabsorbed into the blood and carried throughout the entire body, staining the skin, mucous membranes, and sclerae. It is excreted in the urine, which becomes deep orange and foamy. Because of the decreased amount of bile in the intestinal tract, the stools become light or clay colored. The skin may itch intensely, requiring repeated soothing baths. Dyspepsia and intolerance to fatty foods may develop because of impaired fat digestion in the absence of intestinal bile. In general, AST, ALT, and GGT levels rise only moderately, but bilirubin and alkaline phosphatase levels are elevated.

Hereditary Hyperbilirubinemia

Hyperbilirubinemia (increased serum bilirubin levels), resulting from any of several inherited disorders, can also produce jaundice. Gilbert syndrome is a familial disorder characterized by an increased level of unconjugated bilirubin that causes jaundice. Although serum bilirubin levels are increased, liver histology and liver function test results are normal, and there is no hemolysis. This syndrome affects 3% to 8% of the population, predominantly males (Bope & Kellerman, 2018).

Other conditions that are probably caused by inborn errors of biliary metabolism include Dubin–Johnson syndrome (chronic idiopathic jaundice, with pigment in the liver) and Rotor syndrome (chronic familial conjugated hyperbilirubinemia, without pigment in the liver); the “benign” cholestatic jaundice of pregnancy, with retention of conjugated bilirubin, probably secondary to unusual sensitivity to the hormones of pregnancy; and benign recurrent intrahepatic cholestasis.

Portal Hypertension

Portal hypertension is the increased pressure throughout the portal venous system that results from obstruction of blood flow into and through the damaged liver. Commonly associated with hepatic cirrhosis, it can also occur with noncirrhotic liver disease. Although splenomegaly (enlarged spleen) with possible hypersplenism is a common manifestation of portal hypertension, the two major consequences of portal hypertension are ascites and varices (Friedman & Martin, 2018).

Ascites

Pathophysiology

The mechanisms responsible for the development of ascites are not completely understood. Portal hypertension and the resulting increase in capillary pressure and obstruction of venous blood flow through the damaged liver are contributing factors. The vasodilation that occurs in the splanchnic circulation (the arterial supply and venous drainage of the GI system from the distal esophagus to the midrectum, including the liver and spleen) is also a suspected causative factor. The failure of the liver to metabolize aldosterone increases sodium and water retention by the kidney. Sodium and water retention, increased intravascular fluid volume, increased lymphatic flow, and decreased synthesis of albumin by the damaged liver all contribute to the movement of fluid from the vascular system into the peritoneal space. The process becomes self-perpetuating; loss of fluid into the peritoneal space causes further sodium and water retention by the kidney in an effort to maintain the vascular fluid volume.

As a result of liver damage, large amounts of albumin-rich fluid, 20 L or more, may accumulate in the peritoneal cavity as ascites (Hammer & McPhee, 2019; Mansour & McPherson, 2018). Ascites may also occur with disorders such as cancer, kidney disease, and heart failure. With the movement of albumin from the serum to the peritoneal cavity, the osmotic pressure of the serum decreases. This, combined with increased portal pressure, results in movement of fluid into the peritoneal cavity (see Fig. 43-5).

Physiology/Pathophysiology

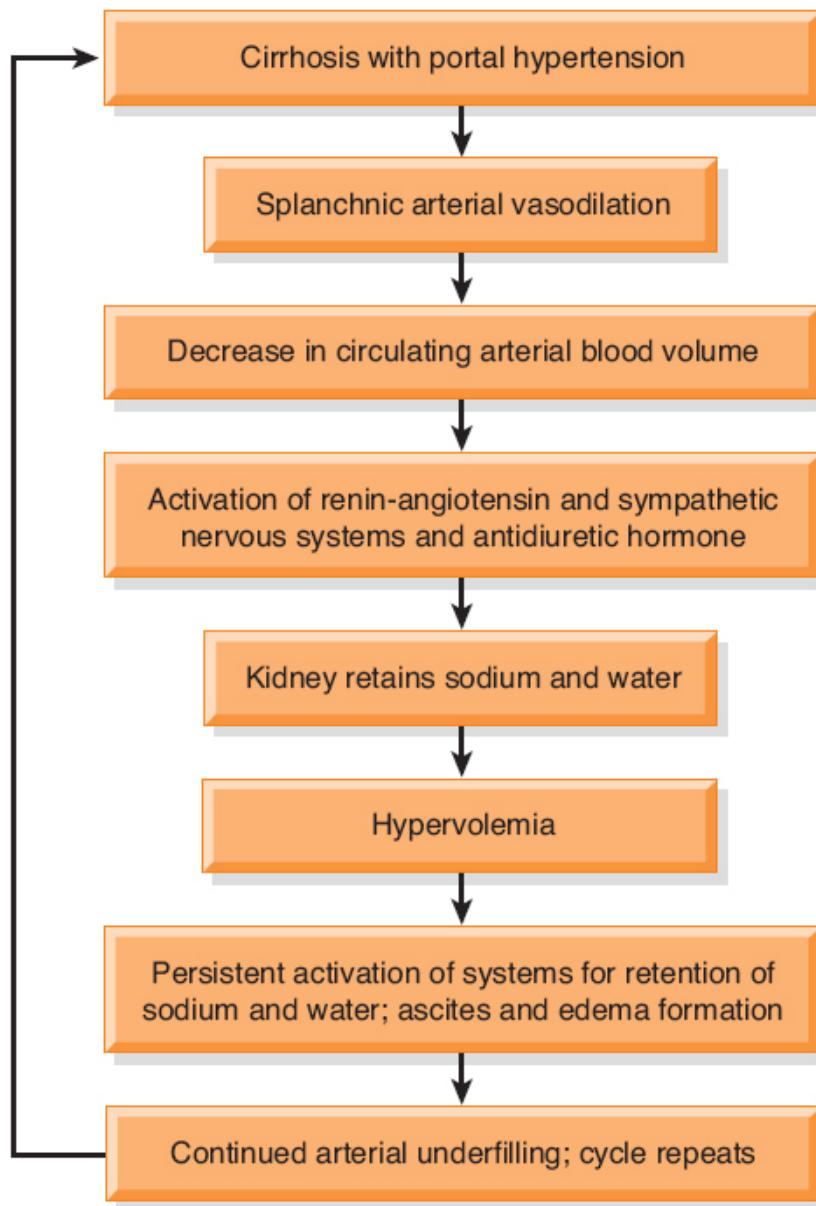


Figure 43-5 • Pathogenesis of ascites (arterial vasodilation theory).

Clinical Manifestations

Increased abdominal girth and rapid weight gain are common presenting symptoms of ascites. The patient may be short of breath and uncomfortable from the enlarged abdomen; striae and distended veins may be visible over the abdominal wall. Umbilical hernias also occur frequently in those patients with cirrhosis. Fluid and electrolyte imbalances are common.

Assessment and Diagnostic Findings

The presence and extent of ascites are assessed by percussion of the abdomen. When fluid has accumulated in the peritoneal cavity, the flanks bulge when the patient assumes a supine position. The presence of fluid can be confirmed by percussing for shifting dullness or by detecting a fluid wave (see Fig. 43-6) (Weber & Kelley, 2018). A fluid wave is likely to be found only if a large amount of fluid is present (Hammer & McPhee, 2019). Daily measurement and recording of abdominal girth and body weight are essential to assess the progression of ascites and its response to treatment.

Medical Management

The medical management of the patient with ascites includes dietary modifications, pharmacologic therapy, bed rest, paracentesis, the use of shunts, and other therapies.

Nutritional Therapy

The goal of treatment for the patient with ascites is a negative sodium balance to reduce fluid retention. Table salt, salty foods, salted butter and margarine, and all canned and frozen foods that are not specifically prepared for low-sodium (2-g sodium) diets should be avoided (Hammer & McPhee, 2019; Simonetto, Liu, & Kamath, 2019). It may take 2 to 3 months for the patient's taste buds to adjust to unsalted foods. In the meantime, the taste of unsalted foods can be improved by using salt substitutes such as lemon juice, oregano, and thyme. Commercial salt substitutes need to be approved by the patient's primary provider, because those that contain ammonia could precipitate hepatic encephalopathy and coma. Most salt substitutes contain potassium and should be avoided if the patient has impaired renal function. The patient should make liberal use of powdered, low-sodium milk and milk products. If fluid accumulation is not controlled with this regimen, the daily sodium allowance may be reduced further to 500 mg, and diuretic agents may be given. However, most patients will not accept such a severe sodium restriction as 500 mg, so clinicians often will not recommend it (Simonetto et al., 2019).



Figure 43-6 • Assessing for abdominal fluid wave. The examiner places the hands along the sides of the patient's flanks, then strikes one flank sharply, detecting any fluid wave with the other hand. An assistant's hand is placed (ulnar side down) along the patient's midline to prevent the fluid wave from being transmitted through the tissues of the abdominal wall.

Dietary control of ascites via strict sodium restriction is difficult to achieve at home. The likelihood that the patient will follow a 2-g sodium diet increases if the patient and the person preparing meals understand the rationale for the diet and receive periodic guidance about selecting and preparing appropriate foods. Approximately 10% of patients with ascites respond to these measures alone. Patients who do not respond and those who find sodium restriction difficult require diuretic therapy (Hammer & McPhee, 2019; Simonetto et al., 2019).

Pharmacologic Therapy

The use of diuretic agents along with sodium restriction is successful in 90% of patients with ascites (Hammer & McPhee, 2019; Mansour & McPherson, 2018). Spironolactone, an aldosterone-blocking agent, is most often the first-line therapy in patients with ascites from cirrhosis. When used with other

diuretic agents, spironolactone helps prevent potassium loss. Oral diuretic agents such as furosemide may be added but should be used cautiously, because long-term use may induce severe hyponatremia (sodium depletion). Ammonium chloride and acetazolamide are contraindicated because of the possibility of precipitating hepatic encephalopathy and coma. Daily weight loss should not exceed 1 kg (2.2 lb) in patients with ascites and peripheral edema or 0.5 to 0.75 kg (1.1 to 1.65 lb) in patients without edema (Bope & Kellerman, 2018; Hammer & McPhee, 2019; Simonetto et al., 2019). Fluid restriction is not attempted unless the serum sodium concentration is very low.

Possible complications of diuretic therapy include fluid and electrolyte disturbances (including hypovolemia, hypokalemia, hyponatremia, and hypochloremic alkalosis) (see [Chapter 10](#)) and encephalopathy. Encephalopathy may be precipitated by dehydration and hypovolemia. In addition, when potassium stores are depleted, the amount of ammonia in the systemic circulation increases, which may cause impaired cerebral functioning and encephalopathy.

Bed Rest

In patients with ascites, an upright posture is associated with activation of the renin–angiotensin–aldosterone system and sympathetic nervous system (Hammer & McPhee, 2019). This causes reduced renal glomerular filtration and sodium excretion and a decreased response to loop diuretics. Therefore, bed rest may be a useful therapy, especially for patients whose condition is refractory to diuretic agents.

Paracentesis

Paracentesis is the removal of fluid (ascites) from the peritoneal cavity through a puncture or a small surgical incision through the abdominal wall under sterile conditions (Hammer & McPhee, 2019; Simonetto et al., 2019). Ultrasound guidance may be indicated in some patients who are at high risk for bleeding because of an abnormal coagulation profile and in those who have had previous abdominal surgery and may have adhesions. Paracentesis was once considered a routine form of treatment for ascites. However, it is now performed primarily for diagnostic examination of ascitic fluid; in treatment for massive ascites that is resistant to nutritional and diuretic therapy and is causing severe problems to the patient; and as a prelude to diagnostic imaging studies, peritoneal dialysis, or surgery. A sample of the ascitic fluid may be sent to the laboratory for cell count, albumin and total protein levels, culture, and other tests.

Large-volume (5 to 6 L) paracentesis is a safe method for treating patients with severe ascites. The use of this therapeutic intervention should not be restricted to patients in whom diuretic therapy has failed but should be

considered the treatment of choice for all patients with large-volume ascites (Hammer & McPhee, 2019; Mansour & McPherson, 2018; Simonetto et al., 2019). This technique, in combination with the IV infusion of salt-poor albumin or other colloid, has become a standard management strategy yielding an immediate effect. Refractive, massive ascites is unresponsive to multiple diuretic agents and sodium restriction for 2 weeks or more and can result in severe sequelae such as respiratory distress, which requires rapid intervention. Albumin infusions help to correct decreases in effective arterial blood volume that lead to sodium retention. The use of this colloid reduces the incidence of postparacentesis circulatory dysfunction with renal dysfunction, hyponatremia, and rapid reaccumulation of ascites associated with decreased effective arterial volume (Hammer & McPhee, 2019; Mansour & McPherson, 2018; Simonetto et al., 2019). The beneficial effects of albumin administration on hemodynamic stability and renal functional status may be related to an improvement in cardiac function as well as a decrease in the degree of arterial vasodilation. Although the patient with cirrhosis has a greatly increased extracellular blood volume, the kidney incorrectly senses that the intravascular volume has decreased. The renin–angiotensin–aldosterone axis is stimulated, and sodium is reabsorbed (Hammer & McPhee, 2019). In addition, antidiuretic hormone secretion increases, which leads to increased retention of free water and sometimes to the development of dilutional hyponatremia. Therapeutic paracentesis provides only temporary removal of fluid; ascites rapidly recurs, necessitating repeated fluid removal.



For the procedural guidelines for assisting with a paracentesis,

go to thepoint.lww.com/Brunner15e.

Transjugular Intrahepatic Portosystemic Shunt Assisting with Paracentesis

Transjugular intrahepatic portosystemic shunt (TIPS) is a method of treating ascites in which a cannula is threaded into the portal vein by the transjugular route (see Fig. 43-7). To reduce portal hypertension, an expandable stent is inserted to serve as an intrahepatic shunt between the portal circulation and the hepatic vein. This is extremely effective in decreasing sodium retention, improving the renal response to diuretic therapy, and preventing recurrence of fluid accumulation (Hammer & McPhee, 2019; Simonetto et al., 2019). TIPS is an effective management strategy for refractive ascites. However, due to a higher risk of encephalopathy and higher cost of TIPS compared with large-volume paracentesis plus albumin, many consider TIPS a second-line therapy.

for refractive ascites that continues to occur despite medical management (Hammer & McPhee, 2019; Hung & Lee, 2019; Simonetto et al., 2019).

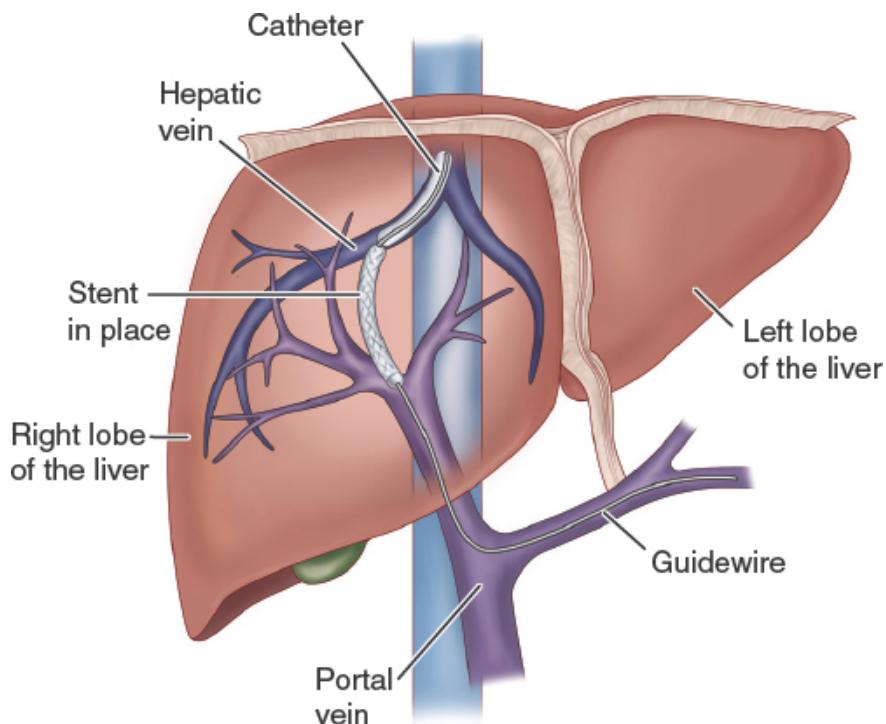


Figure 43-7 • Transjugular intrahepatic portosystemic shunt. A stent is inserted via catheter to the portal vein to divert blood flow and reduce portal hypertension.

Because the development of ascites in patients with cirrhosis is associated with a 50% mortality rate, patients considered candidates for liver transplantation may be referred for TIPS if paracentesis is contraindicated.

Other Methods of Treatment

Ascites can also be treated by the insertion of a peritoneovenous shunt to redirect ascitic fluid from the peritoneal cavity into the systemic circulation via an abdominal and a thoracic catheter that drain into the superior vena cava through a one-way valve (Hammer & McPhee, 2019; Simonetto et al., 2019). However, this procedure is rarely used due to the availability of newer, more effective therapies such as TIPS. In patients with ESLD, some with refractory ascites may be candidates for peritoneal catheters for palliation (Macken, Hashim, Mason, et al., 2019).

Nursing Management

If a patient with ascites from liver dysfunction is hospitalized, nursing measures include assessment and documentation of intake and output (I&O), abdominal girth, and daily weight to assess fluid status. The nurse also closely monitors the respiratory status because large volumes of ascites can compress the thoracic cavity and inhibit adequate lung expansion. The nurse monitors serum ammonia, creatinine, and electrolyte levels to assess electrolyte balance, response to therapy, and indications of hepatic encephalopathy.

Promoting Home, Community-Based, and Transitional Care



Educating Patients About Self-Care

The patient treated for ascites is likely to be discharged with some ascites still present. Before hospital discharge, the nurse educates the patient and family about the treatment plan, including the need to avoid all alcohol intake, adhere to a low-sodium diet, take medications as prescribed, and check with the primary provider before taking any new medications, including OTC and herbal preparations. Additional home care education is summarized in [Chart 43-3](#).

Continuing and Transitional Care

A referral for transitional, home, or community-based care may be warranted, especially if the patient lives alone or cannot provide self-care. The home visit enables the nurse to assess changes in the patient's condition and weight, abdominal girth, skin, and cognitive and emotional status. The nurse assesses the home environment and the availability of resources needed to adhere to the treatment plan (e.g., a scale to obtain daily weights, facilities to prepare and store appropriate foods, resources to purchase needed medications). The nurse also assesses the patient's adherence to the treatment plan and the ability to buy, prepare, and eat appropriate foods. The nurse reinforces previous education and emphasizes the need for regular follow-up and the importance of keeping scheduled health care appointments.

Esophageal Varices

Esophageal varices are present in 30% of patients with compensated cirrhosis and 60% of patients with decompensated cirrhosis at the time of diagnosis (Hammer & McPhee, 2019; Kovacs & Jensen, 2019; Simonetto et al., 2019) (see Clinical Manifestations in the Hepatic Cirrhosis section for further discussion). Varices are varicosities that develop from elevated pressure in the veins that drain into the portal system. They are prone to rupture and often are

the source of massive hemorrhages from the upper GI tract and the rectum. In addition, abnormalities in blood clotting, often seen in patients with severe liver disease, increase the likelihood of bleeding with significant blood loss.

Once esophageal varices form, they increase in size over time and may eventually bleed (Hammer & McPhee, 2019; Kovacs & Jensen, 2019; Simonetto et al., 2019). In cirrhosis, varices are the most significant source of bleeding. The first bleeding episode has a mortality rate of 10% to 30% depending on the severity of the liver disease and is one of the major causes of death in patients with cirrhosis. Overall mortality associated with acute variceal bleeding ranges from 10% to 40%. The mortality rate is related to failure to control a bleeding episode and the occurrence of early rebleeding (Hammer & McPhee, 2019; Kovacs & Jensen, 2019; Simonetto et al., 2019). Patients surviving the first episode of variceal bleeding are at very high risk for recurrent bleeding (approximately 70%) and death (30% to 50%) (Hammer & McPhee, 2019; Kovacs & Jensen, 2019; Simonetto et al., 2019).

Pathophysiology

Esophageal varices are dilated, tortuous veins that are usually found in the submucosa of the lower esophagus but may develop higher in the esophagus or extend into the stomach. This condition is almost always caused by portal hypertension, which results from obstruction of the portal venous circulation within the damaged liver.

Chart 43-3



HOME CARE CHECKLIST

Management of Ascites

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

- State the impact of ascites and treatment on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- State the name, dose, side effects, frequency, and schedule for all medications.
- Describe effects, side effects, and monitoring parameters for diuretic therapy.
- Discuss importance of avoiding nonsteroidal anti-inflammatory agents, medications containing alcohol (e.g., cough mixtures), antibiotics, or antacids containing salt.
- Make appropriate dietary choices consistent with dietary prescription and recommendations.
 - Explain the use of salt substitutes must be approved by primary provider.
- State the importance of weighing self-daily and keeping a daily record of weight.
 - Maintain record of daily weight, and identify daily weight-loss goals.
 - List weight changes (loss or gain) that should be reported to the primary provider.
- Identify rationale for fluid restrictions (if needed), and for monitoring and keeping a daily record intake and output.
 - Maintain a record of daily intake and output.
 - Identify changes in output that should be reported to primary provider (e.g., decreasing urine output).
- Identify need to stop all alcohol intake as critical to well-being.
- Explain how to contact Alcoholics Anonymous or alcohol counselors in related organizations if indicated.
- Demonstrate how to inspect and care for skin, alleviate pressure over bony prominences by turning when in bed or chair, and decrease edema by position changes.
- Identify early signs and symptoms of complications (encephalopathy, spontaneous bacterial peritonitis, dehydration, electrolyte abnormalities, azotemia).
- Relate how to reach primary provider with questions or complications.
- State time and date of follow-up medical appointments, therapy, and testing.
- Identify sources of support (e.g., friends, relatives, faith community).
- Identify the contact details for support services for patients and their caregivers/families.

- Identify the need for health promotion, disease prevention, and screening activities.

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

Physiology/Pathophysiology

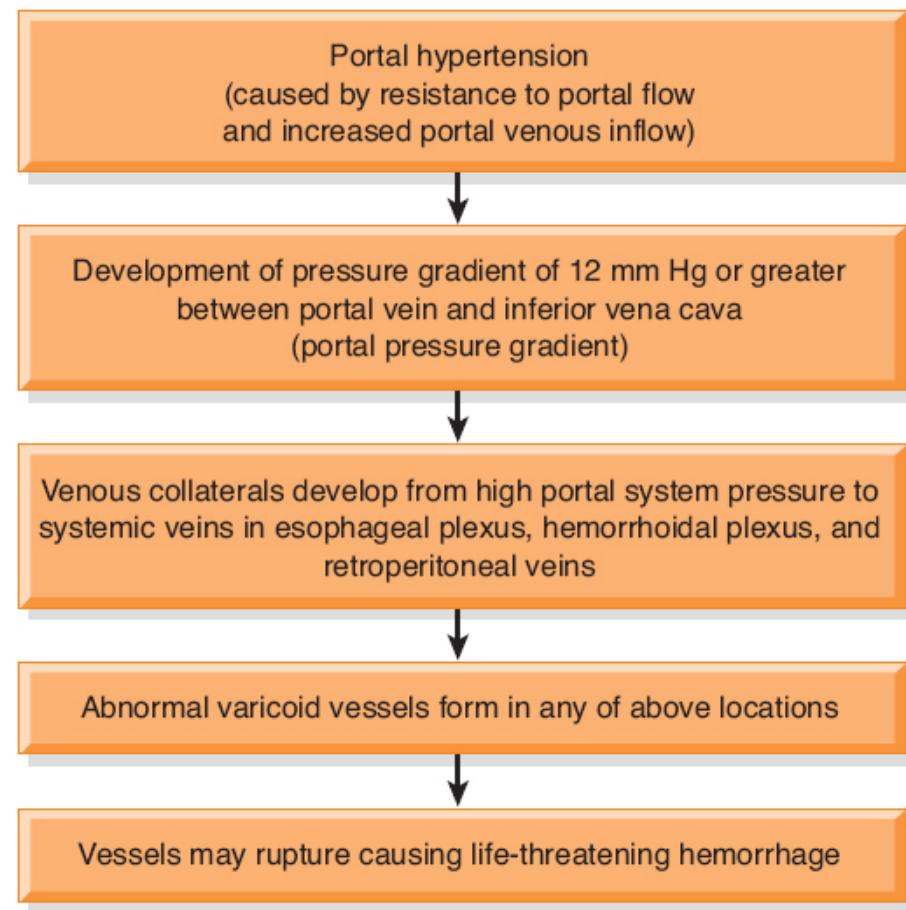


Figure 43-8 • Pathogenesis of bleeding esophageal varices.

Because of increased obstruction of the portal vein, venous blood from the intestinal tract and spleen seeks an outlet through collateral circulation (new pathways for return of blood to the right atrium). The effect is increased pressure, particularly in the vessels in the submucosal layer of the lower esophagus and upper part of the stomach. These collateral vessels are not very elastic; rather, they are tortuous and fragile, and they bleed easily (see Fig. 43-8). Less common causes of varices are abnormalities of the circulation in the splenic vein or superior vena cava and hepatic venothrombosis.

Bleeding esophageal varices are life-threatening and can result in hemorrhagic shock that produces decreased cerebral, hepatic, and renal

perfusion. In turn, there is an increased nitrogen load from bleeding into the GI tract and an increased serum ammonia level, increasing the risk of encephalopathy. Usually, the dilated veins cause no symptoms. However, if the portal pressure increases sharply and the mucosa or supporting structures become thin, massive hemorrhaging occurs.

Factors that contribute to hemorrhage are muscular exertion from lifting heavy objects; straining at stool; sneezing, coughing, or vomiting; esophagitis; irritation of vessels by poorly chewed foods or irritating fluids; and reflux of stomach contents (especially alcohol). Salicylates and any medication that erodes the esophageal mucosa or interferes with cell replication also may contribute to bleeding.

Clinical Manifestations

The patient with bleeding esophageal varices may present with hematemesis, melena, or general deterioration in mental or physical status and often has a history of alcohol abuse. Signs and symptoms of shock (cool clammy skin, hypotension, tachycardia) may be present (see [Chapter 11](#)).

Assessment and Diagnostic Findings

Endoscopy is used to identify the bleeding site, along with ultrasonography, CT scanning, and angiography. Another diagnostic tool, the endoscopic video capsule, can detect esophageal varices but does not substitute for endoscopy unless this test cannot be performed. Standard endoscopy is superior to video capsule for the diagnosis of esophageal varices (Hammer & McPhee, 2019; Kovacs & Jensen, 2019; Simonetto et al., 2019). Because varices are present in 50% of patients with cirrhosis, it is recommended that patients who have been diagnosed with cirrhosis undergo screening endoscopy. If no varices are detected on initial endoscopy, the test should be repeated in 2 to 3 years in an effort to identify and treat large varices, which are the ones most likely to bleed. If small varices are identified on initial endoscopy, the test should be repeated in 1 to 2 years (Hammer & McPhee, 2019; Kovacs & Jensen, 2019; Simonetto et al., 2019).

Endoscopy

Immediate endoscopy (see [Chapter 38](#)) is indicated to identify the cause and the site of bleeding; approximately 40% of patients with suspected bleeding from esophageal varices are actually bleeding from another source (gastritis, ulcer) (Hammer & McPhee, 2019; Kovacs & Jensen, 2019; Simonetto et al., 2019). Nursing support is essential during this often stressful experience. Careful monitoring can detect early signs of cardiac arrhythmias, perforation, and hemorrhage.

After the examination, fluids are not given until the patient's gag reflex returns. Lozenges and gargles may be used to relieve throat discomfort if the patient's physical condition and mental status permit. If the patient is actively bleeding, oral intake will not be permitted, and the patient will be prepared for further diagnostic and therapeutic procedures.

Portal Hypertension Measurements

Portal hypertension may be suspected if dilated abdominal veins and hemorrhoids are detected. Splenomegaly and ascites may also be present. Portal venous pressure can be measured directly or indirectly. Indirect measurement of the hepatic vein pressure gradient is the most common procedure. The measurement requires insertion of a catheter with a balloon into the antecubital or femoral vein. The catheter is advanced under fluoroscopy to a hepatic vein. Fluid is infused once the catheter is in position to inflate the balloon. A "wedged" pressure (similar to pulmonary artery wedge pressure) is obtained by occluding the blood flow in the blood vessel; pressure in the unoccluded vessel is also measured and the hepatic venous pressure gradient (HVPG) is obtained. An HVPG of over 10 mm Hg is indicative of clinically significant portal hypertension (Kovacs & Jensen, 2019). Although the values obtained may underestimate portal pressure, this measurement may be taken several times to evaluate the results of therapy (Kovacs & Jensen, 2019; Schiff et al., 2018).

Direct measurement of portal vein pressure can be obtained by several methods. One direct measurement requires insertion of a catheter into the portal vein or one of its branches (Kovacs & Jensen, 2019; Schiff et al., 2018). Endoscopic measurement of pressure within varices is used only in conjunction with endoscopic sclerotherapy (see later discussion).

Laboratory Tests

Laboratory studies may include various liver function tests, such as serum aminotransferases, bilirubin, alkaline phosphatase, and serum proteins. Splenoportography, which involves serial or segmental x-rays, is used to detect extensive collateral circulation in esophageal vessels, which would indicate varices. Other tests are hepatoportography and celiac angiography. These are usually performed in the operating room or x-ray department.



Medical Management

Bleeding from esophageal varices is an emergency that can quickly lead to hemorrhagic shock. The patient is critically ill, requiring aggressive medical care and expert nursing care, and is usually transferred to the intensive care unit (ICU) for close monitoring and management. See [Chapter 11](#) for a

discussion of care of the patient in shock. The extent of bleeding is evaluated, and vital signs are monitored continuously if hematemesis and melena are present.

Because patients with bleeding esophageal varices have intravascular volume depletion and are subject to electrolyte imbalance, IV fluids, electrolytes, and volume expanders are provided to restore fluid volume and replace electrolytes. Transfusion of blood components also may be required.

Caution must be taken with volume resuscitation so that overhydration does not occur, because this would raise portal pressure and increase bleeding. An indwelling urinary catheter is usually inserted to permit frequent monitoring of urine output.

Although a variety of pharmacologic, endoscopic, and surgical approaches are used to treat bleeding esophageal varices, none is ideal, and most are associated with considerable risk to the patient. Nonsurgical treatment of bleeding esophageal varices is preferable because of the high mortality rate of emergency surgery to control bleeding esophageal varices and because of the poor physical condition that is typical of the patient with severe liver dysfunction.

Pharmacologic Therapy

In suspected variceal bleeding, vasoactive drugs such as octreotide or vasopressin need to be given as soon as possible and before endoscopy (Kovacs & Jensen, 2019; Simonetto et al., 2019). In a patient who is actively bleeding, medications are given initially because they can be obtained and given more quickly than other therapies. Octreotide, a synthetic analog of the hormone somatostatin, is effective in decreasing bleeding from esophageal varices, and lacks the vasoconstrictive effects of vasopressin. Because of this safety and efficacy profile, octreotide is considered the preferred treatment regimen for immediate control of variceal bleeding. These medications cause selective splanchnic vasoconstriction by inhibiting glucagon release and are used mainly in the management of active hemorrhage. Adverse effects are rare with octreotide but mild hypoglycemia and abdominal cramping can occur (Kovacs & Jensen, 2019; Simonetto et al., 2019).

Vasopressin may be the initial mode of therapy in urgent situations because it produces constriction of the splanchnic arterial bed and decreases portal pressure. As described previously, splanchnic circulation comprises the arterial blood supply and venous drainage of the entire GI tract from the distal esophagus to the midrectum, including the liver and spleen. Vasopressin constricts distal esophageal and proximal gastric veins, thus reducing the inflow into the portal system and therefore the portal pressure. Vital signs and the presence or absence of blood in the gastric aspirate indicate the effectiveness of vasopressin. Monitoring of I&O and electrolyte levels is

necessary because hyponatremia may develop, and vasopressin may have an antidiuretic effect.

Coronary artery disease is a contraindication to the use of vasopressin because coronary vasoconstriction is a side effect that may precipitate myocardial infarction. The combination of vasopressin with nitroglycerin (given by the IV, sublingual, or transdermal route) has been effective in reducing or preventing the side effects (constriction of coronary vessels and angina) caused by vasopressin alone. Side effects of vasopressin include myocardial and extremity ischemia as well as cardiac arrhythmias; therefore, vasopressin is used only in urgent situations or when other agents such as octreotide are not available. Vasopressin must be given with close monitoring (Kovacs & Jensen, 2019; Simonetto et al., 2019).

Beta-blocking agents such as propranolol, nadolol, or carvedilol that decrease portal pressure are the most common medications used both to prevent a first bleeding episode in patients with known varices and to prevent rebleeding (Bunchorntavakul & Reddy, 2019; Hammer & McPhee, 2019; Simonetto et al., 2019). Beta-blockers have been shown to effectively reduce the risk of variceal bleeding and its associated mortality. Beta-blockers should not be used in acute variceal hemorrhage, but they are effective prophylaxis against initial and recurrent bleeding episodes (Bunchorntavakul & Reddy, 2019; Hammer & McPhee, 2019; Simonetto et al., 2019). Nitrates such as isosorbide lower portal pressure by venodilation and decreased cardiac output and may be used in combination with beta-blockers to reduce the risk of recurrent variceal bleeding (Bunchorntavakul & Reddy, 2019; Kovacs & Jensen, 2019).



Balloon Tamponade

Although used infrequently today, balloon tamponade therapy may be used to temporarily control hemorrhage and to stabilize a patient with massive bleeding prior to other definitive management (Kovacs & Jensen, 2019; Simonetto et al., 2019). This procedure involves the insertion of a tube from the nose into the stomach. This tube has two inflatable balloons, one esophageal and one gastric. When inflated from a port external to the patient, these balloons compress bleeding varices in the stomach or esophagus to inhibit bleeding.

When indicated, balloon tamponade can be successful; however, there are risks. Displacement of the tube and the inflated balloon into the oropharynx can cause life-threatening obstruction of the airway and asphyxiation. This may occur if the patient pulls on the tube because of confusion or discomfort. It may also result from rupture of the gastric balloon, which causes the

esophageal balloon to move into the oropharynx. Sudden rupture of the balloon causes airway obstruction and aspiration of gastric contents into the lungs. Therefore, the tube must be tested before insertion to minimize this risk by ensuring that the balloons can attain and maintain inflation. Aspiration of blood and secretions into the lungs is frequently associated with balloon tamponade, especially in the stuporous or comatose patient. Endotracheal intubation before insertion of the tube protects the airway and minimizes the risk of aspiration. Ulceration and necrosis of the nose, the mucosa of the stomach, or the esophagus may occur if the tube is left in place too long, inflated too long, or inflated at too high a pressure. The therapy is used for as short a time as possible to control bleeding while emergency treatment is completed and definitive therapies are instituted (no longer than 12 hours, preferably less) (Kovacs & Jensen, 2019; Mansour & McPherson, 2018; Simonetto et al., 2019).



Quality and Safety Nursing Alert

The patient being treated with balloon tamponade must remain under close observation in the ICU and must be monitored continuously because of the risk of serious complications such as aspiration, esophageal ulcer formation, and perforation. Precautions must be taken to ensure that the patient does not pull on or inadvertently displace the tube.

Nursing measures include frequent mouth and nasal care. For secretions that accumulate in the mouth, tissues should be within easy reach of the patient. Oral suction may be necessary to remove secretions.

Although balloon tamponade stops the bleeding in 90% of patients, bleeding recurs in 60% to 70%, necessitating other treatment modalities, such as endoscopic therapies (see later discussion). Once the balloons are deflated or the tube is removed, the patient must be assessed frequently because of the high risk of recurrent bleeding (Kovacs & Jensen, 2019; Mansour & McPherson, 2018; Simonetto et al., 2019).

Endoscopic Sclerotherapy

In endoscopic **sclerotherapy** (see Fig. 43-9), also referred to as injection sclerotherapy, a sclerosing agent (i.e., sodium morrhuate, ethanolamine oleate, sodium tetradecyl sulfate, or ethanol) is injected through a fiberoptic endoscope into or adjacent to the bleeding esophageal varices to promote thrombosis and eventual sclerosis (Kovacs & Jensen, 2019; Mansour & McPherson, 2018). The process of sclerotherapy causes inflammation of the involved vein with eventual thrombosis and loss of the lumen of the vessel.

The procedure has been used successfully to treat acute GI hemorrhage but is not recommended for prevention of first and subsequent variceal bleeding episodes where **endoscopic variceal ligation (EVL)**, also known as esophageal banding therapy (discussed later), is the first-line treatment (Kovacs & Jensen, 2019; Mansour & McPherson, 2018).

After treatment for acute hemorrhage, the patient must be observed for bleeding, perforation of the esophagus, aspiration pneumonia, and esophageal stricture. Antacids, histamine-2 (H_2) antagonists such as cimetidine, or proton pump inhibitors such as pantoprazole may be given after the procedure to counteract the chemical effects of the sclerosing agent on the esophagus and the acid reflux associated with the therapy.

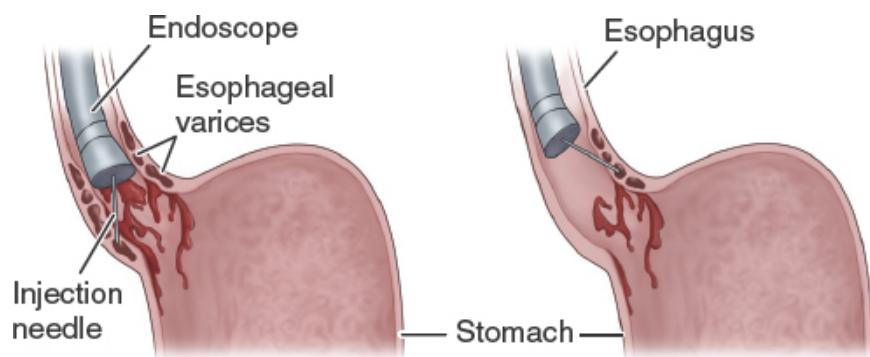


Figure 43-9 • Endoscopic or injection sclerotherapy. Injection of sclerosing agent into esophageal varices through an endoscope promotes thrombosis and eventual sclerosis, thereby obliterating the varices.

Endoscopic Variceal Ligation (Esophageal Banding Therapy)

In EVL (see Fig. 43-10), also referred to as variceal banding, a modified endoscope loaded with an elastic rubber band is passed through an overtube directly onto the varix (or varices) to be banded. After the bleeding varix is suctioned into the tip of the endoscope, the rubber band is slipped over the tissue, causing necrosis, ulceration, and eventual sloughing of the varix.

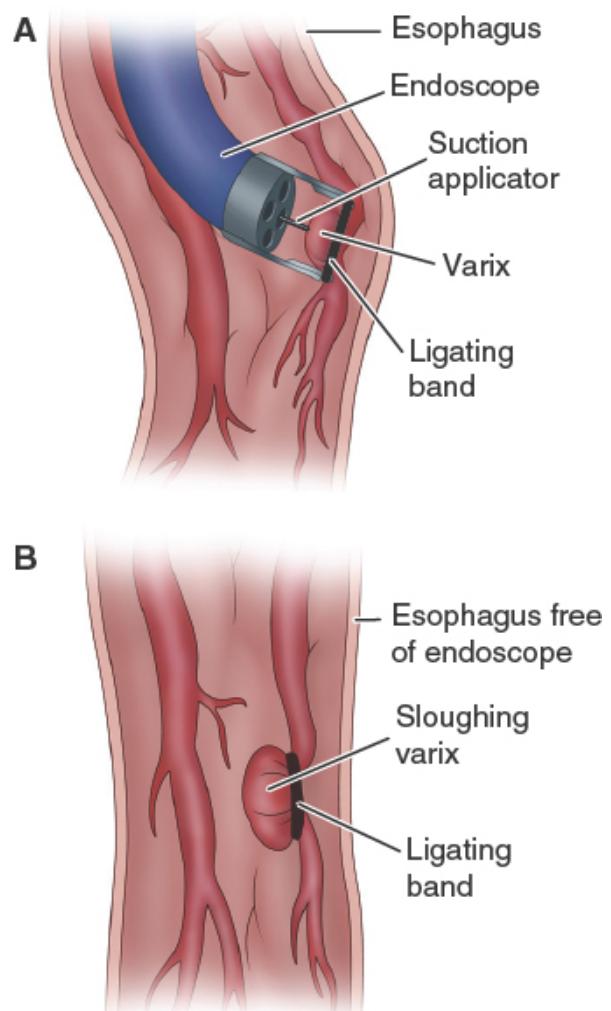


Figure 43-10 • Endoscopic variceal ligation. **A.** A rubber band–like ligature is slipped over an esophageal varix via an endoscope. **B.** Necrosis results, and the varix eventually sloughs off.

An EVL procedure is effective in controlling acute bleeding. Compared with sclerotherapy, EVL also significantly reduces the rebleeding rate, mortality, procedure-related complications, as well as number of sessions needed to eradicate varices and thus has replaced sclerotherapy as the treatment of choice in the management of esophageal varices (Kovacs & Jensen, 2019; Simonetto et al., 2019). Potential complications include superficial ulceration and dysphagia, transient chest discomfort, and, rarely, esophageal strictures. An EVL procedure in combination with pharmacologic therapy may be more effective than monotherapy (i.e., a single mode of therapy) in the treatment of acute hemorrhage. EVL is also recommended for patients who have experienced variceal bleeding while receiving beta-blocker therapy and for those who cannot tolerate beta-blocking agents (Kovacs & Jensen, 2019; Simonetto et al., 2019).

Transjugular Intrahepatic Portosystemic Shunt

A TIPS procedure (see Fig. 43-9) is indicated for the treatment of an acute episode of uncontrolled variceal bleeding refractory to pharmacologic or endoscopic therapy. In 10% to 20% of patients for whom urgent band ligation or sclerotherapy and medications are not successful in eradicating bleeding, a TIPS procedure can effectively control acute variceal hemorrhage by rapidly lowering portal pressure. Polytetrafluoroethylene-covered TIPS placement should be considered in high-risk patients with cirrhosis and active bleeding at the time of endoscopy (Kovacs & Jensen, 2019; Simonetto et al., 2019). Potential complications of TIPS include bleeding, sepsis, heart failure, organ perforation, shunt thrombosis, and progressive liver failure.

Additional Therapies

The use of endoscopically placed tissue adhesives and fibrin glue has been successful in the treatment of gastric and esophageal varices. Coated expandable stents (placed via endoscope) have also been used effectively for the same purpose (Kovacs & Jensen, 2019; Simonetto et al., 2019). Portosystemic shunting into lower resistance vessels (those vessels not affected by the high pressure in the portal system) and the end-organ collateral variceal formation that results in bleeding may also be treated by a variety of embolization procedures, including balloon-occluded retrograde transvenous obliteration (BRTO) (Brunicardi, 2019; Philips, Rajesh, Augustine, et al., 2019).

Surgical Management

Several surgical procedures have been developed to treat esophageal varices and to minimize rebleeding, but these procedures have significant risk. Procedures that may be used for esophageal varices are direct surgical ligation of varices; splenorenal, mesocaval, and portacaval venous shunts to relieve portal pressure; and esophageal transection with devascularization. These procedures are rarely used and remain controversial, as studies regarding their effectiveness and outcomes continue. What is known is that these procedures are very effective in controlling variceal bleeding. They may be considered as second-line management (rescue therapy) in those patients for whom all other treatments have failed, those who are not candidates for liver transplantation, and those who require a bridge to transplantation. There is a high incidence of encephalopathy after the surgical shunting procedures, and morbidity and mortality statistics remain high (Kovacs & Jensen, 2019; Philips et al., 2019). The TIPS procedure has largely replaced the use of surgical decompression shunts and ligation procedures but these interventions may still be used in some cases to manage esophageal varices.

Surgical Bypass Procedures

Surgical decompression (shunt surgery) of the portal circulation may be used with the advent of a variceal bleeding episode. Although effective in eradicating bleeding, survival statistics and encephalopathy are worse than other preventative measures such as a TIPS procedure when this method is employed for prophylaxis, and shunt surgery for this purpose has largely been abandoned worldwide (Brunicardi, 2019; Kovacs & Jensen, 2019). The current recommendation is that surgical shunts be considered only in patients who have Model for End-Stage Liver Disease (MELD) scores of <15, who are not candidates for hepatic transplantation, or who have limited access to TIPS therapy and needed follow-up. (See later discussion of MELD classification in Liver Transplantation section.)

An aim of a surgical shunt is to reduce portal venous pressure (Brunicardi, 2019). One surgical shunting procedure (see Fig. 43-11) is the distal splenorenal shunt, which is made between the splenic vein and the left renal vein after splenectomy. A mesocaval shunt is created by anastomosing the superior mesenteric vein to the proximal end of the vena cava or to the side of the vena cava using grafting material. The goal of distal splenorenal and mesocaval shunts is to decrease portal pressure by draining only a portion of venous blood from the portal bed; therefore, they are considered selective shunts. The liver continues to receive some portal flow, and the incidence of encephalopathy may be reduced. Portacaval shunts are considered nonselective shunts because they divert all portal flow to the vena cava via end-to-side or side-to-side approaches.

These procedures are extensive and are not always successful because of secondary thrombosis in the veins used for the shunt and because of complications (e.g., encephalopathy, accelerated liver failure). The effectiveness of these procedures has been studied extensively. All shunt procedures are equally effective in preventing recurrent variceal bleeding but may cause further impairment of liver function and encephalopathy. Partial portacaval shunts with interposition grafts are as effective as other shunts but are associated with a lower rate of encephalopathy (Cameron & Cameron, 2020; Schiff et al., 2018). The severity of the disease (by a classification such as the Child–Pugh system, discussed later) and the potential for future liver transplantation guide the treatment decision. If the cause of portal hypertension is the rare Budd–Chiari syndrome (which is manifested by noncirrhotic portal hypertension caused by hepatic vein thrombosis) or other venous obstructive disease, a portacaval or a mesoatrial shunt may be performed (see Fig. 43-11). The mesoatrial shunt is required when the infrahepatic vena cava is thrombosed and must be bypassed.

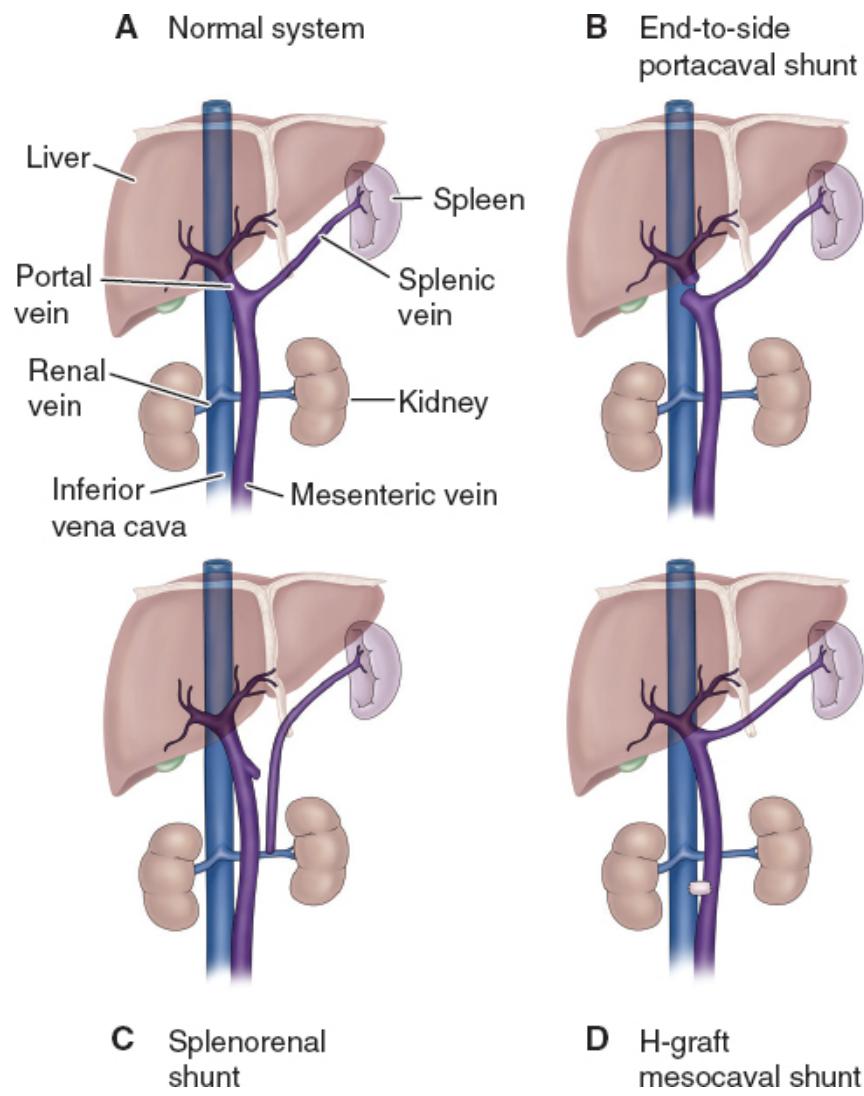


Figure 43-11 • Portosystemic shunts. **A.** Normal portal system. **B–D.** Examples of portal shunts to reduce portal pressure.

Devascularization and Transection

Devascularization and staple-gun transection procedures to separate the bleeding site from the high-pressure portal system have been used in the emergency management of variceal bleeding. The lower end of the esophagus is reached through a small gastrostomy incision; a staple gun permits anastomosis of the transected ends of the esophagus. Rebleeding is a risk, and the outcomes of these procedures vary among patient populations.



Quality and Safety Nursing Alert

The surgical procedures used to treat esophageal varices do not alter the course of the progressive liver disease, and bleeding may recur as new collateral vessels develop. The risk of complications (hypovolemic or hemorrhagic shock, hepatic encephalopathy, electrolyte imbalance, metabolic and respiratory alkalosis, alcohol withdrawal syndrome, and seizures) is high.

Nursing Management

Nursing assessment includes monitoring the patient's physical condition and evaluating emotional responses and cognitive status. The nurse monitors and records vital signs and assesses the patient's nutritional and neurologic status. This assessment assists in identifying hepatic encephalopathy (see later discussion).

If complete rest of the esophagus is indicated because of bleeding, parenteral nutrition is initiated. Gastric suction usually is initiated to keep the stomach as empty as possible and to prevent straining and vomiting. The patient often complains of severe thirst, which may be relieved by frequent oral hygiene and moist sponges to the lips. The nurse closely monitors the blood pressure. Vitamin K therapy and multiple blood transfusions often are indicated because of blood loss. A quiet environment and calm reassurance may help to relieve the patient's anxiety and reduce agitation.

Bleeding anywhere in the body is anxiety provoking, resulting in a crisis for the patient and family. If the patient has been a heavy user of alcohol, delirium secondary to alcohol withdrawal can complicate the situation. The nurse provides support and explanations about medical and nursing interventions to prepare both the patient and the family, because these procedures can be difficult to undergo and observe. Close monitoring of the patient helps in detecting and managing complications. Management modalities and nursing care of the patient with bleeding esophageal varices are summarized in [Table 43-2](#).

TABLE 43-2 Select Modalities and Nursing Care for the Patient with Bleeding Esophageal Varices

Treatment Modality ^a	Nursing Interventions
Nonsurgical Modalities	
Pharmacologic agents	Observe response to therapy.
Propranolol	Monitor for side effects: <i>propranolol, carvedilol, nadolol</i> —decreased pulse pressure, impaired cardiovascular response to hemorrhage; <i>vasopressin</i> —angina (nitroglycerin may be prescribed to prevent or treat angina).
Carvedilol	
Nadolol	
Vasopressin	
Octreotide	Support patient during treatment.
Balloon tamponade	Explain procedure to patient briefly to obtain cooperation with insertion and maintenance of esophageal/gastric tamponade tube and reduce patient's fear of the procedure. Monitor closely to prevent inadvertent removal or displacement of tube, subsequent airway obstruction, and aspiration. Provide frequent oral hygiene.
Endoscopic sclerotherapy	Observe for aspiration, perforation of the esophagus, and recurrence of bleeding after treatment.
Endoscopic variceal ligation	Observe for recurrence of bleeding, esophageal perforation.
Transjugular intrahepatic portosystemic shunt (TIPS)	Observe for rebleeding and signs of infection.
Balloon-occluded retrograde transvenous obliteration (BRTO) procedure	Observe for rebleeding, signs of infection, or changes in mental status.
Surgical Modalities	
Portal-systemic shunt	Observe for development of portal-systemic encephalopathy (altered mental status, neurologic dysfunction), hepatic failure, and rebleeding. Requires intensive, expert nursing care for prolonged period.
Surgical ligation of varices	Observe for rebleeding.
Esophageal transection and devascularization	Observe for rebleeding. Provide postthoracotomy care.

^aSeveral modalities may be used concurrently or in sequence.

Adapted from Brunicardi, F. C. (2019). *Schwartz's principles of surgery* (11th ed.). New York: McGraw-Hill Education; Feldman, M., Friedman, L. S., & Brandt, L. J. (2016).

Hepatic Encephalopathy and Coma

Hepatic encephalopathy, or portosystemic encephalopathy, is a life-threatening complication of liver disease that occurs with profound liver failure. Patients with this condition may have no overt signs of the illness but have abnormalities on neuropsychological testing (Hammer & McPhee, 2019; Simonetto et al., 2019; Yanny, Winters, Boutros, et al., 2019). Hepatic encephalopathy is the neuropsychiatric manifestation of hepatic failure associated with portal hypertension and the shunting of blood from the portal venous system into the systemic circulation (Mansour & McPherson, 2018; Yanny et al., 2019). This reversible metabolic form of encephalopathy can improve with recovery of liver function. The onset is often insidious and subtle, and initially the disease is termed *subclinical* or *minimal hepatic encephalopathy*.

Pathophysiology

Despite the frequency with which hepatic encephalopathy occurs, the precise pathophysiology is not fully understood (Yanny et al., 2019). Two major alterations underlie its development in acute and chronic liver disease. First, hepatic insufficiency may result in encephalopathy because of the inability of the liver to detoxify toxic by-products of metabolism. Second, portosystemic shunting, in which collateral vessels develop as a result of portal hypertension, allows elements of the portal blood (laden with potentially toxic substances usually extracted by the liver) to enter the systemic circulation (Yanny et al., 2019). Ammonia is considered the major etiologic factor in the development of encephalopathy. Ammonia enters the brain and excites peripheral benzodiazepine-type receptors on astrocyte cells, increasing neurosteroid synthesis, and stimulating gamma-aminobutyric acid (GABA) neurotransmission. GABA causes depression of the central nervous system that inhibits neurotransmission and synaptic regulation (Yanny et al., 2019), producing sleep and behavior patterns associated with hepatic encephalopathy.

Circumstances that increase serum ammonia levels tend to aggravate or precipitate hepatic encephalopathy. The largest source of ammonia is the enzymatic and bacterial digestion of dietary and blood proteins in the GI tract. Ammonia from these sources increases as a result of GI bleeding (i.e., bleeding esophageal varices, chronic GI bleeding), a high-protein diet, bacterial infection, or uremia. The ingestion of ammonium salts also increases the blood ammonia level. In the presence of alkalosis or hypokalemia, increased amounts of ammonia are absorbed from the GI tract and from the

renal tubular fluid. Conversely, serum ammonia is decreased by elimination of protein from the diet and by the administration of antibiotics, such as neomycin sulfate, which reduce the number of intestinal bacteria capable of converting urea to ammonia (Hammer & McPhee, 2019; Yanny et al., 2019).

Other factors unrelated to increased serum ammonia levels that can cause hepatic encephalopathy in susceptible patients include excessive diuresis, dehydration, infections, surgery, fever, and some medications (sedatives, tranquilizers, analgesics, and diuretics that cause potassium loss). Additional causes include elevated levels of serum manganese (Schiff et al., 2018), as well as changes in the types of circulating amino acids, mercaptans, and levels of dopamine and other neurotransmitters in the central nervous system (Schiff et al., 2018). Mercaptans are toxic metabolites of sulfur-containing compounds that are excreted by the liver under normal conditions. Mercaptans and these other so-called “false” neurotransmitters may be generated from an intestinal source or from metabolism of protein by the liver and, with defective hepatic clearance, may precipitate encephalopathy.

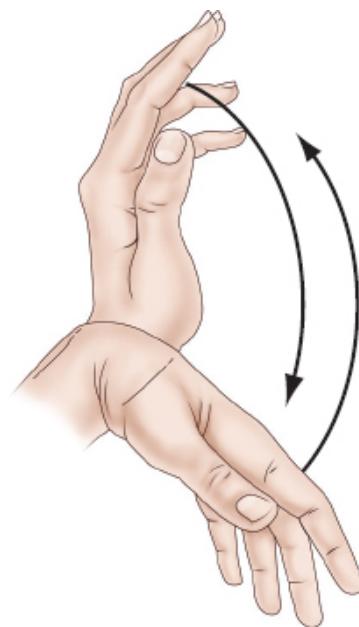


Figure 43-12 • Asterixis (“liver flap”) may occur in hepatic encephalopathy. The patient is asked to hold the arm out with the hand held upward (dorsiflexed). Within a few seconds, the hand falls forward involuntarily and then quickly returns to the dorsiflexed position.

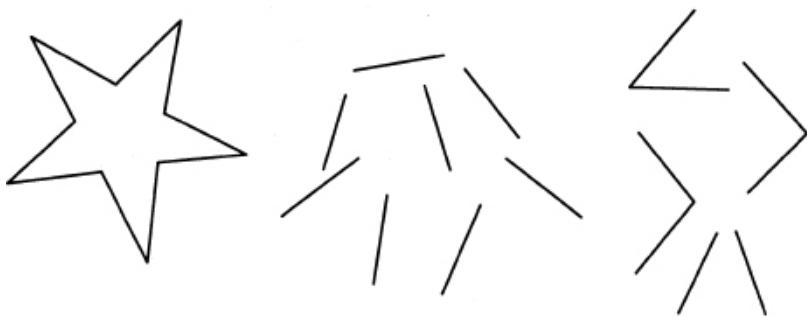
Clinical Manifestations

The earliest symptoms of hepatic encephalopathy include mental status changes and motor disturbances. The patient appears confused and unkempt

and has alterations in mood and sleep patterns. The patient tends to sleep during the day and has restlessness and insomnia at night. As hepatic encephalopathy progresses, the patient may become difficult to awaken and completely disoriented with respect to time and place. With further progression, the patient lapses into frank coma and may have seizures.

Asterixis, an involuntary flapping of the hands, may be seen in stage II encephalopathy (see Fig. 43-12). Simple tasks, such as handwriting, become difficult. A handwriting or drawing sample (e.g., star figure), taken daily, may provide graphic evidence of progression or reversal of hepatic encephalopathy. Inability to reproduce a simple figure in two or three dimensions (see Fig. 43-13) is referred to as **constructional apraxia**. In the early stages of hepatic encephalopathy, the deep tendon reflexes are hyperactive; with worsening of the encephalopathy, these reflexes disappear and the extremities may become flaccid.

Occasionally, **fetor hepaticus**, a sweet, slightly fecal odor to the breath that is presumed to be of intestinal origin, may be noticed. The odor has also been described as similar to that of freshly mowed grass, acetone, or old wine. Fetor hepaticus is prevalent with extensive collateral portal circulation in chronic liver disease.



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Figure 43-13 • Effects of constructional apraxia. Deterioration of handwriting and inability to draw a simple star figure occurs with progressive hepatic encephalopathy. Reprinted with permission from Morgan, M. (2018). Hepatic encephalopathy in patients with cirrhosis. In J. Dooley, A. S. Lok, G. Garcia-Tsao, & P. Massimo (Eds.). *Sherlock's diseases of the liver and biliary system* (13th ed.). Oxford, UK: John Wiley & Sons.

Assessment and Diagnostic Findings

Several diagnostic algorithms and a variety of psychometric tests are used in determining the presence and severity of hepatic encephalopathy. The electroencephalogram shows generalized slowing, an increase in the amplitude of brain waves, and characteristic triphasic waves. The survival rate after a first episode of overt hepatic encephalopathy in patients with cirrhosis is approximately 40% at 1 year. Eligible patients should be referred for liver transplantation after this initial episode (Mansour & McPherson, 2018; Yanny et al., 2019).

Medical Management

Medical management focuses on identifying and eliminating the precipitating cause, if possible, initiating ammonia-lowering therapy, minimizing potential medical complications of cirrhosis and depressed consciousness, and reversing the underlying liver disease, if possible. Correction of the possible reasons for the deterioration such as bleeding, electrolyte abnormalities, sedation, or azotemia is essential. Lactulose is given to reduce serum ammonia levels. It acts by trapping and expelling the ammonia in the feces (Hammer & McPhee, 2019; Mansour & McPherson, 2018; Yanny et al., 2019). Two or three soft stools per day are desirable; this indicates that lactulose is performing as intended.



Quality and Safety Nursing Alert

The patient receiving lactulose is monitored closely for the development of watery diarrhea stools, because they indicate a medication overdose. Serum ammonia levels are closely monitored as well.

Possible side effects of lactulose include intestinal bloating and cramps, which usually disappear within a week. To mask the sweet taste, which some patients dislike, it can be diluted with fruit juice. The patient is closely monitored for hypokalemia and dehydration. Other laxatives are not prescribed during lactulose administration because their effects disturb dosage regulation. Lactulose may be given by nasogastric tube or enema for patients who are comatose or for those in whom oral administration is contraindicated or not possible (Yanny et al., 2019).

Other management strategies include IV administration of glucose to minimize protein breakdown, administration of vitamins to correct deficiencies, and correction of electrolyte imbalances (especially potassium). Antibiotics may also be added to the treatment regimen. Neomycin, metronidazole, and rifaximin have been used to reduce levels of ammonia-

forming bacteria in the colon. However, no benefit has been shown for long-term treatment with these antibiotics (Mansour & McPherson, 2018; Yanny et al., 2019). Additional management strategies for hepatic encephalopathy include the following:

- Neurologic status is assessed frequently.
- Mental status is monitored by keeping a daily record of handwriting and arithmetic performance.
- I&O and body weight are recorded each day.
- Vital signs are measured and recorded every 4 hours.
- Potential sites of infection (peritoneum, lungs) are assessed frequently, and abnormal findings are reported promptly.
- Serum ammonia level is monitored daily.
- Dietary protein intake should not be restricted in hepatic encephalopathy as recommended in the past. Protein intake should be maintained at 1.2 to 1.5 g/kg/day (European Association for the Study of the Liver [EASL], 2019; Styskel, Natarajan, & Kanwal, 2019; Yanny et al., 2019; Yao, Fung, Chu, et al., 2018) (see [Chart 43-4](#)). The danger of protein malnutrition far outweighs the risk of worsening hepatic encephalopathy caused by increased protein intake (Styskel et al., 2019; Yanny et al., 2019; Yao et al., 2018).
- Enteral feeding is provided for patients whose encephalopathic state persists.
- Reduction in the absorption of ammonia from the GI tract is accomplished by the use of gastric suction, enemas, or oral antibiotics.
- Electrolyte status is monitored and corrected if abnormal.
- Sedatives, tranquilizers, and analgesic medications are discontinued.
- Benzodiazepine antagonists such as flumazenil may be administered IV to improve encephalopathy, whether or not the patient has previously taken benzodiazepines. This action may have short-term efficacy because patients with hepatic encephalopathy have an increased concentration of benzodiazepine receptors (Friedman & Martin, 2018).

Chart 43-4

Nutritional Management of Hepatic Encephalopathy

- Minimize the formation and absorption of toxins, principally ammonia, from the intestine.
- Keep daily protein intake between 1.2 and 1.5 g/kg body weight per day.
- Avoid protein restriction if possible, even in those with encephalopathy.
- For patients who are truly protein intolerant, provide additional nitrogen in the form of an amino acid supplement. The use of branched-chain amino acids should be a consideration in patients with cirrhosis. It has improved outcomes in varied populations with the disease.
- Provide small, frequent meals and 3 small snacks per day in addition to a late-night snack before bed.

Adapted from European Association for the Study of the Liver (EASL). (2019). EASL clinical practice guidelines on nutrition in chronic liver disease. *Journal of Hepatology*, 70(1), 172–193; Styskel, B., Natarajan, Y., & Kanwal, F. (2019). Nutrition in alcoholic liver disease: An update. *Clinics in Liver Disease*, 23(1), 99–114; Yanny, B., Winters, A., Boutros, S., et al. (2019). Hepatic encephalopathy challenges, burden, and diagnostic and therapeutic approach. *Clinics in Liver Disease*, 23(4), 607–623; Yao, C. K., Fung, J., Chu, N. H. S., et al. (2018). Dietary interventions in liver cirrhosis. *Journal of Clinical Gastroenterology*, 52(8), 663–673.



Nursing Management

Table 43-3 presents the stages of hepatic encephalopathy, common signs and symptoms, and potential nursing diagnoses for each stage. The nurse is responsible for maintaining a safe environment to prevent injury, bleeding, and infection. The nurse administers the prescribed treatments and monitors the patient for the numerous potential complications. The potential for respiratory compromise is great given the patient's depressed neurologic status. The nurse encourages deep breathing and position changes to prevent the development of atelectasis, pneumonia, and other respiratory complications. Despite aggressive pulmonary care, patients may develop respiratory compromise. They may require intubation and mechanical ventilation to protect the airway, and they are frequently admitted to the ICU.

The nurse communicates with the patient's family to inform them about the patient's status and supports them by explaining the procedures and treatments that are part of the patient's care. If the patient recovers from hepatic encephalopathy and coma, rehabilitation is likely to be prolonged. Therefore, the patient and family will require assistance to understand the causes of this severe complication and to recognize that it may recur.

Promoting Home, Community-Based and Transitional Care



Educating Patients About Self-Care

If the patient has recovered from hepatic encephalopathy and is to be discharged home, the nurse educates the family about subtle signs of recurrent encephalopathy. The goals for caloric intake and protein intake should be 35 to 40 kcal/kg body weight per day and 1.2 to 1.5 g/kg body weight per day (see [Chart 43-4](#)) (Styskel et al., 2019; Yanny et al., 2019; Yao et al., 2018). Protein intake should not be limited too severely, because doing so worsens nutritional status and increases mortality (Styskel et al., 2019; Yanny et al., 2019; Yao et al., 2018). Continued use of lactulose after discharge is not uncommon, and the patient and family should closely monitor its efficacy and side effects. They should also be cautioned that constipation can precipitate encephalopathy and may be prevented through the prescribed use of lactulose.

Continuing and Transitional Care

Referral for transitional, home, or community-based care is warranted for the patient who returns home after recovery from hepatic encephalopathy. The nurse assesses the patient's physical and mental status and collaborates closely with the primary provider. The home visit provides an opportunity for the nurse to assess the home environment and the ability of the patient and family to monitor signs and symptoms and follow the treatment regimen. The nurse must evaluate the patient's fluid volume status and be alert for changes indicative of hypovolemia due to decreased intake and for decreased urine output associated with hepatorenal syndrome (see later discussion). Monitoring of laboratory values continues to be important, and the nurse must obtain prescriptions to correct abnormalities, especially electrolyte imbalances, which also can worsen encephalopathy.

The safety of the home environment is assessed closely to identify areas of risk for falls and other injuries. Home or transitional care visits are especially important if the patient lives alone because encephalopathy may affect the patient's ability to remember or follow the treatment regimen. The nurse reinforces previous education and reminds the patient and family about the importance of dietary restrictions, close monitoring, and follow-up. In addition, the nurse must observe the patient for subtle behavior changes of worsening hepatic encephalopathy. Patients with all types and stages of hepatic encephalopathy should have periodic neurologic evaluations to determine their cognitive function so that they do not engage in potentially harmful activities. Even subtle neuropsychiatric abnormalities may preclude patients from

driving, operating machinery, or participating in other activities that require psychomotor coordination.

Patients and families may need additional support during those times that the patient exhibits mood disturbances and sleep disorders. Patients should be as active as possible during the day and develop a normal sleep-wake pattern. Sedating medications should be avoided because they may precipitate encephalopathy. Patients and families may require assistance in developing plans to cope with changes in mood and mental status changes. This plan should identify support persons to attend to the patient in the home if needed. Social workers and case managers may make appropriate referrals for assistance with physical and psychosocial support and care. Referrals to psychologists, psychiatric liaison nurses, case managers, social workers, or therapists may assist family members with coping. Spiritual advisors may also provide another outlet for communication and guidance. If alcohol played a role in the development of the liver disease and encephalopathy, referral to Alcoholics Anonymous or Al-Anon may provide needed support and education.

TABLE 43-3 Stages of Hepatic Encephalopathy and Applicable Nursing Diagnoses

Stage	Clinical Symptoms	Clinical Signs and EEG Changes	Select Potential Nursing Diagnoses ^a
1	Normal level of consciousness with periods of lethargy and euphoria; reversal of day–night sleep patterns	Impaired writing and ability to draw line figures. Normal EEG.	Activity intolerance Impaired ability to manage regime Impaired sleep pattern
2	Increased drowsiness; disorientation; inappropriate behavior; mood swings; agitation	Asterixis; fetor hepaticus. Abnormal EEG with generalized slowing.	Impaired socialisation Impaired role performance Risk for injury Acute confusion
3	Stuporous; difficult to rouse; sleeps most of time; marked confusion; incoherent speech	Asterixis; increased deep tendon reflexes; rigidity of extremities. EEG markedly abnormal.	Impaired nutritional intake Impaired mobility Impaired verbal communication
4	Comatose; may not respond to painful stimuli	Absence of asterixis; absence of deep tendon reflexes; flaccidity of extremities. EEG markedly abnormal.	Risk for aspiration Impaired gas exchange Impaired tissue integrity

EEG, electroencephalogram.

^aNursing diagnoses are likely to progress; thus, most nursing diagnoses present at earlier stages will occur during later stages as well.

Adapted from information in Feldman, M., Friedman, L. S., & Brandt, L. J. (2016). *Sleisinger & Fordtran's gastrointestinal & liver disease* (10th ed.). Philadelphia, PA: Saunders Elsevier.

Other Manifestations of Hepatic Dysfunction

Edema and Bleeding

Many patients with liver dysfunction develop generalized edema caused by hypoalbuminemia due to decreased hepatic production of albumin. The production of blood clotting factors by the liver is also reduced, leading to an

increased incidence of bruising, epistaxis, bleeding from wounds, and, as described previously, GI bleeding. Abnormalities in the number and effectiveness of platelets also contribute to the bleeding in liver dysfunction. Congestion of the spleen secondary to portal hypertension causes hypersplenism (increased pooling of platelets in the organ). The resultant thrombocytopenia generally correlates with spleen size. In patients who abuse alcohol, suppression of bone marrow by the acute toxic effects of alcohol or folate deficiency may contribute to the thrombocytopenia (Goldman & Schafer, 2019). These factors predispose patients to easy bruising, petechiae formation, and bleeding from a variety of sources such as the GI or genitourinary tract (Goldman & Shafer, 2019).

Vitamin Deficiency

Decreased production of several clotting factors may be partially due to deficient absorption of vitamin K from the GI tract. This probably is caused by the inability of liver cells to use vitamin K to make prothrombin (Barrett et al., 2019; Hammer & McPhee, 2019). Absorption of the other fat-soluble vitamins (vitamins A, D, and E) as well as dietary fats may also be impaired because of decreased secretion of bile salts into the intestine.

Another group of problems common to patients with severe chronic liver dysfunction results from inadequate intake of sufficient vitamins. These include the following:

- Vitamin A deficiency, resulting in night blindness and eye and skin changes
- Thiamine deficiency, leading to beriberi, polyneuritis, and Wernicke–Korsakoff psychosis
- Riboflavin deficiency, resulting in characteristic skin and mucous membrane lesions
- Pyridoxine deficiency, resulting in skin and mucous membrane lesions and neurologic changes
- Vitamin C deficiency, resulting in the hemorrhagic lesions of scurvy
- Vitamin K deficiency, resulting in hypoprothrombinemia, characterized by spontaneous bleeding and ecchymoses
- Folic acid deficiency, resulting in macrocytic anemia

Because of these potential vitamin deficiencies, the diet of every patient with chronic liver disease (especially if alcohol related) is supplemented with vitamins A, B complex, C, K, and folic acid (EASL, 2019; Styskel et al., 2019; Yao et al., 2018).

Metabolic Abnormalities

Abnormalities of glucose metabolism also occur; the blood glucose level may be abnormally high shortly after a meal (similar to that when diabetes is present), but hypoglycemia may occur during fasting because of decreased hepatic glycogen reserves and decreased gluconeogenesis. Medications must be used cautiously and in reduced dosages because the ability to metabolize medications is decreased in the patient with liver failure.

Many endocrine abnormalities also occur with liver dysfunction because the liver cannot properly metabolize hormones, including androgens and sex hormones. Failure of the damaged liver to inactivate estrogens normally can cause gynecomastia, amenorrhea, testicular atrophy, loss of pubic hair in the male, menstrual irregularities in the female, and other disturbances of sexual function and sex characteristics.

Pruritus and Other Skin Changes

Patients with liver dysfunction resulting from biliary obstruction commonly develop severe pruritus due to retention of bile salts. Patients may develop vascular (or arterial) spider angiomas on the skin (see Fig. 43-3), usually above the waistline. These are numerous small vessels resembling a spider's legs. They are most often associated with cirrhosis, especially in alcoholic liver disease. Patients may also develop palmar erythema ("liver palms" or reddened palms).

VIRAL HEPATITIS

Viral hepatitis is a systemic, viral infection in which necrosis and inflammation of liver cells produce a characteristic cluster of clinical, biochemical, and cellular changes. To date, five definitive types of viral hepatitis that cause liver disease have been identified: hepatitis A, B, C, D, and E. Hepatitis A and E are similar in mode of transmission (fecal–oral route), whereas hepatitis B, C, and D share many other characteristics.

Hepatitis is easily transmitted and causes high morbidity and prolonged loss of time from school or employment. Acute viral hepatitis affects 0.5% to 1% of people in the United States each year. Hepatitis A virus (HAV) was responsible for 3366 cases in the United States in 2017. Incidence rates decreased more than 95% from 1995 to 2011, then increased by 140% from 2011 to 2017. In 2017, large person-to-person outbreaks began occurring, among persons who use drugs and persons experiencing homelessness (Centers for Disease Control and Prevention [CDC], 2017). During the same year, the hepatitis B virus (HBV) was the offending agent in a total of 3407 cases of acute viral hepatitis nationwide. The occurrence rate of viral hepatitis C (HCV) in 2017 was 3186 cases, with an incidence of 1.0 cases per 100,000 population, which represents an increase since 2013. Rates have been

influenced by the opioid crisis. An estimated 2.4 million people in the United States are living with HCV infection (CDC, 2017).

The number of reported acute hepatitis B cases has remained stable with a slight increase in 2017. The increase is most likely due to increasing injection drug use related to the opioid crisis, and improved surveillance (CDC, 2017). The overall decrease in HBV rates since 1990 is largely due to the use of hepatitis A and B vaccines, the introduction of universal precautions and blood supply safety measures as well as public health education regarding high-risk behaviors (Goldman & Schafer, 2019). Conversely, the incidence of HAV and HCV infections has been on the rise. It is estimated that 60% to 90% of viral hepatitis cases go unreported (CDC, 2017). The occurrence of subclinical cases, failure to recognize mild cases, and misdiagnosis are thought to contribute to the underreporting. **Table 43-4** compares the major forms of viral hepatitis.

The clinical presentation of hepatitis varies with individual patients as well as with the specific causative virus. Four phases of infectious hepatitis describe the clinical presentation. Phase 1 is the viral replication phase in which patients are asymptomatic but laboratory studies will reveal markers of hepatitis. Phase 2 is the preicteric or prodromal phase when those affected may experience anorexia, nausea, vomiting, fatigue and pruritus. Phase 3 is the icteric phase which is characterized by jaundice and dark urine. Some patients experience abdominal pain from an enlarged liver. Phase 4 is the convalescent phase when signs and symptoms resolve and laboratory values return to normal. Not all patients will experience all phases, especially those with a mild form of the disease (Chi, Cleary, & Bocchini, 2018; Shin, 2018).

TABLE 43-4 Comparison of Major Forms of Viral Hepatitis

	Hepatitis A	Hepatitis B	Hepatitis C	Hepatitis D	Hepatitis E
Previous Names	Infectious Hepatitis	Serum Hepatitis	Non-A, non-B Hepatitis		
Epidemiology					
Cause	Hepatitis A virus (HAV)	Hepatitis B virus (HBV)	Hepatitis C virus (HCV)	Hepatitis D virus (HDV)	Hepatitis E virus (HEV)
Immunity	Average: 30 days Homologous	Average: 70–80 days Homologous	Average: 50 days Second attack may indicate weak immunity or infection with another agent.	Average: 35 days Homologous	Average: 31 days Unknown
Nature of Illness					
Signs and symptoms	May occur with or without symptoms; flu-like illness <i>Preicteric phase:</i> Headache, malaise, fatigue, anorexia, fever <i>Icteric phase:</i> Dark urine, jaundice of sclera and skin, tender liver	May occur without symptoms May develop arthralgias, rash	Similar to HBV; less severe and anicteric	Similar to HBV	Similar to HAV; very severe in pregnant women
Outcome	Usually mild with recovery. No carrier state or increased risk of chronic hepatitis, cirrhosis, or hepatic cancer.	May be severe. Carrier state possible. Increased risk of chronic hepatitis, cirrhosis, and hepatic cancer.	Frequent occurrence of chronic carrier state and chronic liver disease, but effective therapies that provide a sustained virologic response (SVR) are available. SVR is indicative of a cure of HCV infection. Increased risk of hepatic cancer if disease is not treated.	Similar to HBV but greater likelihood of carrier state, chronic active hepatitis, and cirrhosis	Similar to HAV except very severe in pregnant women

Adapted from Goldman, L., & Schafer, A. I. (2019). *Goldman's Cecil medicine* (26th ed.). Philadelphia, PA: Saunders Elsevier.

Hepatitis A Virus

The HAV accounts for 20% to 25% of cases of clinical hepatitis in the United States (CDC, 2017). Hepatitis A, formerly called *infectious hepatitis*, is caused by an RNA virus of the enterovirus family. In the United States, the disease is seen mainly in the adult population. HAV is transmitted primarily through the fecal–oral route, by the ingestion of food or liquids infected with the virus. It is more prevalent in countries with overcrowding and poor sanitation. The virus has been found in the stool of infected patients before the onset of symptoms and during the first few days of illness.

Typically, a child or a young adult acquires the infection at school through poor hygiene, hand-to-mouth contact, or other close contact. The virus is carried home, where haphazard sanitary habits spread it through the family. An infected food handler can spread the disease, and people can contract it by consuming water or shellfish from sewage-contaminated waters. Outbreaks have occurred in day care centers and institutions as a result of poor hygiene among people with developmental disability. Hepatitis A can be transmitted during sexual activity; this is more likely with oral–anal contact or anal intercourse and with multiple sex partners (Chi et al., 2018; Goldman & Schafer, 2019; Shin & Jeong, 2018). Hepatitis A is not transmitted by blood transfusions.

The incubation period is estimated to be between 2 and 6 weeks, with a mean of approximately 4 weeks (CDC, 2017; Chi et al., 2018; Shin & Jeong, 2018). The illness may be prolonged, lasting 4 to 8 weeks. It usually lasts longer and is more severe in those older than 40 years. Most patients recover from hepatitis A; it rarely progresses to acute liver necrosis or acute hepatic failure resulting in cirrhosis of the liver or death. The mortality rate of hepatitis A is approximately 0.5% for those younger than 40 years and 1% to 2% for older adults. In patients with underlying chronic liver disease, morbidity and mortality are increased in the presence of an acute hepatitis A infection. No carrier state exists, and no chronic hepatitis is associated with the HAV. The virus is present only briefly in the serum; by the time jaundice occurs, the patient is likely to be noninfectious. Although hepatitis A confers immunity against itself, the person may contract other forms of hepatitis.

Clinical Manifestations

Many patients are anicteric (without jaundice) and symptomless. When symptoms appear, they resemble those of a mild, flu-like upper respiratory tract infection, with low-grade fever. Anorexia, an early symptom, is often severe. It is thought to result from release of a toxin by the damaged liver or from failure of the damaged liver cells to detoxify an abnormal product. Later, jaundice and dark urine may become apparent. Indigestion is present in

varying degrees, marked by vague epigastric distress, nausea, heartburn, and flatulence. The patient may also develop a strong aversion to the taste of cigarettes or the presence of cigarette smoke and other strong odors (Papadakis & McPhee, 2020; Shin & Jeong, 2018). These symptoms tend to clear as soon as the jaundice reaches its peak, perhaps 10 days after its initial appearance. Symptoms may be mild in children; in adults, they may be more severe and the course of the disease prolonged.

Assessment and Diagnostic Findings

The liver and spleen are often moderately enlarged for a few days after onset; other than jaundice, there are few other physical signs. An HAV antigen may be found in the stool 7 to 10 days before illness and for 2 to 3 weeks after symptoms appear. HAV antibodies are detectable in the serum, although usually not until symptoms appear. Analysis of subclasses of immunoglobulins can help determine whether the antibody represents acute or past infection.

Prevention

A number of strategies exist to prevent transmission of HAV. Patients and their families are encouraged to follow general precautions that can prevent transmission of the virus. Scrupulous hand hygiene, safe water supplies, and proper control of sewage disposal are just a few of these prevention strategies.

Effective (95% to 100% after two to three doses) and safe HAV vaccines are available (Link-Gelles, Hofmeister, & Nelson, 2018). It is recommended that the two-dose vaccine be given to adults 18 years of age or older, with the second dose given 6 to 12 months after the first. Protection against HAV develops within several weeks after the first dose of the vaccine. Children and adolescents 1 to 18 years of age receive three doses; the second dose is given 1 month after the first, and the third dose is given 6 to 12 months later. HAV routine immunization of young children has proved to be effective in reducing disease incidence and maintaining very low incidence levels among vaccine recipients and across all age groups in many settings (Chi et al., 2018; Goldman & Schafer, 2019). As a result of its effectiveness in decreasing HAV, the hepatitis A vaccination recommendations have been expanded to include all children at 1 year of age. Hepatitis A vaccine is also recommended for people traveling to locations where sanitation and hygiene are unsatisfactory. Vaccination is also recommended for those from high-risk groups, such as men who have sex with men, people who use IV or injection drugs, staff of day care centers, health care personnel and those who work with the virus in research or animal care settings (Chi et al., 2018). The vaccine has also been used to interrupt community-wide outbreaks. A combined HAV and HBV vaccine is available for vaccination of people 18 years of age and older with indications

for both HAV and HBV vaccination. Vaccination consists of three doses, given on the same schedule as that used for single-antigen HBV vaccine.

For people who have not been previously vaccinated, HAV can be prevented by intramuscular administration of globulin during the incubation period, if given within 2 weeks of exposure. This bolsters the person's antibody production and provides 6 to 8 weeks of passive immunity. Immune globulin may suppress overt symptoms of the disease; the resulting subclinical case of HAV would produce immunity to subsequent episodes of the virus.

Immune globulin is also recommended for household members and sexual contacts of people with HAV. Susceptible people in the same household as the patient are usually also infected by the time the diagnosis is made and should receive immune globulin. Institutional contacts of patients with HAV should also receive post-exposure prophylaxis with immune globulin. Prophylaxis is not necessary for casual contacts of an infected person, such as classmates, coworkers, or hospital employees (Link-Gelles et al., 2018). Although rare, systemic reactions to immune globulin do occur. Caution is required when anyone who has previously had angioedema, hives, or other allergic reactions is treated with any human immune globulin. Epinephrine should be available in case of systemic, anaphylactic reaction.

Pre-exposure prophylaxis is recommended for those traveling to developing countries or settings with poor or uncertain sanitation conditions who do not have sufficient time to acquire protection by administration of hepatitis A vaccine (Chi et al., 2018). Prevention strategies for HAV are outlined in [Chart 43-5](#).

Medical Management

Bed rest during the acute stage and a nutritious diet are important aspects of treatment. During the period of anorexia, the patient should receive frequent small feedings, supplemented if necessary by IV fluids with glucose. Because the patient often has an aversion to food, gentle persistence, and creativity may be required to stimulate appetite. Optimal food and fluid levels are necessary to counteract weight loss and to speed recovery. Even before the icteric phase, however, many patients recover their appetites (see [Chart 43-6](#)).

The patient's sense of well-being and laboratory test results are generally appropriate guides to bed rest and restriction of physical activity. Gradual but progressive ambulation hastens recovery.

Chart 43-5  HEALTH PROMOTION

Prevention of Hepatitis

Hepatitis A

- Educate patients regarding safe practices for preparing and dispensing food.
- Encourage conscientious individual hygiene.
- Encourage proper community and home sanitation.
- Facilitate mandatory reporting of viral hepatitis to local health departments.
- Promote community health education programs.
- Promote vaccination to interrupt community-wide outbreaks.
- Recommend pre-exposure vaccination for all children 12–23 months of age. Continue existing immunization programs for children 1–18 years of age.
- Recommend vaccination for travelers to developing countries, illegal drug users (injection and noninjection drug users), men who have sex with men, people with chronic liver disease, people who work with HAV-infected animals or work with HAV in research facilities and recipients (e.g., hemophiliacs) of pooled plasma products for clotting factor disorders.
- Support effective health supervision of schools, dormitories, extended care facilities, barracks, and camps.

Hepatitis B

- Advise avoidance of high-risk behaviors.
- Avoid multidose vials in patient care settings.
- Monitor cleaning, disinfection, and sterilization of reusable devices in patient care settings.
- Recommend vaccination for international travelers to regions with high or intermediate levels of endemic hepatitis B virus infection and for persons with chronic liver disease or with human immune deficiency virus infection.
- Recommend vaccination for persons at risk for infection by sexual exposure, by percutaneous or mucosal exposure to blood.
- Recommend vaccination of all infants in the United States regardless of the mother's hepatitis B.
- Use barrier precautions in situations of contact with blood or body fluids.
- Use needleless IV and injection systems in health care.
- Use standard precautions in clinical care.

Hepatitis C

- Advise avoidance of high-risk behaviors such as IV drug use.
- Avoid multidose vials in patient care settings.

- Monitor cleaning, disinfection, and sterilization of reusable devices in patient care settings.
- Use barrier precautions in situations of contact with blood or body fluids.
- Use needleless IV and injection systems in health care.
- Use standard precautions in clinical care.

Adapted from Ferri, F. F. (Ed.). (2014). *Practical guide to the care of the medical patient* (9th ed.). Philadelphia, PA: Mosby Elsevier.

Chart 43-6

Dietary Management of Hepatitis

- Advise patient to avoid substances (medications, herbs, illicit drugs, and toxins) that may affect liver function, such as St. John's wort in patients taking hepatitis C virus protease inhibitors.
- Be aware that enteral feedings may be necessary if anorexia, nausea, and vomiting persist.
- Carefully monitor fluid balance.
- Instruct patient to abstain from alcohol during acute illness and for at least 6 months after recovery.
- Provide intake of 25–30 kcal/kg/day.
- Provide protein intake of 1.2–1.5 g/kg/day.
- Recommend small, frequent meals; minimize periods without food intake.

Adapted from European Association for the Study of the Liver (EASL). (2019). EASL clinical practice guidelines on nutrition in chronic liver disease. *Journal of Hepatology*, 70(1), 172–193; Styskel, B., Natarajan, Y., & Kanwal, F. (2019). Nutrition in alcoholic liver disease: An update. *Clinics in Liver Disease*, 23(1), 99–114.

Nursing Management

Management usually occurs in the home unless symptoms are severe. Therefore, the nurse assists the patient and family in coping with the temporary disability and fatigue that are common with HAV and educates them to seek additional health care if the symptoms persist or worsen. The patient and family also need specific guidelines about diet, rest, follow-up blood work, and the importance of avoiding alcohol, as well as sanitation and hygiene measures (particularly hand hygiene) to prevent spread of the disease to other family members.

Specific education for patients and families about reducing the risk of contracting HAV includes good personal hygiene, stressing careful hand

hygiene (after bowel movements and before eating) and environmental sanitation (safe food and water supply, effective sewage disposal).

Hepatitis B Virus

Unlike HAV, the HBV is transmitted primarily through blood (percutaneous and permucosal routes). HBV can be found in blood, saliva, semen, and vaginal secretions and can be transmitted through mucous membranes and breaks in the skin. HBV is also transferred from carrier mothers to their infants, especially in areas with a high incidence (e.g., Southeast Asia). The infection usually is not transmitted via the umbilical vein but from the mother at the time of birth and during close contact afterward.

HBV has a long incubation period. It replicates in the liver and remains in the serum for relatively long periods, allowing transmission of the virus. Risk factors for HBV infection are summarized in [Chart 43-7](#). Screening of blood donors has greatly reduced the occurrence of HBV after blood transfusion.

Chart 43-7



RISK FACTORS

Hepatitis B

- Close contact with carrier of hepatitis B virus
- Frequent exposure to blood, blood products, or other body fluids
- Health care workers: hemodialysis staff, oncology and chemotherapy nurses, personnel at risk for needlesticks, operating room staff, respiratory therapists, surgeons, dentists
- Hemodialysis
- IV/injection drug use
- Gay men and bisexual activity
- Mother-to-child transmission
- Multiple sexual partners
- Receipt of blood or blood products (e.g., clotting factor concentrate)
- Recent history of sexually transmitted infection
- Tattooing
- Travel to or residence in area with uncertain sanitary conditions

Adapted from Bope, E. T., & Kellerman, R. D. (Eds.). (2018). *Conn's current therapy*. Philadelphia, PA: Saunders.

Most people (more than 90%) who contract HBV infection develop antibodies and recover spontaneously in 6 months. The mortality rate from acute HBV has been reported to be as high as 1%. Another 10% of patients

who have HBV progress to a carrier state or develop chronic hepatitis with persistent HBV infection and hepatocellular injury and inflammation. It remains a major worldwide cause of cirrhosis and hepatocellular carcinoma (HCC) with higher mortality rates (Papadakis & McPhee, 2020; Sedhom, 2018; Schiff et al., 2018). In fact, approximately 15% of those who develop chronic hepatitis B during adulthood die of cirrhosis or liver cancer. Mortality rates are even higher (25%) for those whose chronic infection occurs during childhood (Chi et al., 2018). An estimated 730,000 adult residents of the United States are afflicted with chronic hepatitis B infection; however, there has been a small but significant decrease in the prevalence among U.S.-born adults who are 20 to 49 years of age (CDC, 2017; Sedhom, 2018).

Gerontologic Considerations

The immune system is altered in older adults. A less responsive immune system may be responsible for the increased incidence and severity of HBV among older adults and the increased incidence of liver abscesses secondary to decreased phagocytosis by the Kupffer cells. The older patient with HBV has a serious risk of severe liver cell necrosis or acute hepatic failure, particularly if other illnesses are present. With the advent of an HBV vaccine as the standard for prevention, the incidence of hepatic diseases may decrease in the future.

Clinical Manifestations

Clinically, HBV closely resembles HAV, but the incubation period is much longer (1 to 6 months). Signs and symptoms of HBV may be insidious and variable. Fever and respiratory symptoms are rare; some patients have arthralgias and rashes. The patient may have loss of appetite, dyspepsia, abdominal pain, generalized aching, malaise, and weakness. Jaundice may or may not be evident. If jaundice occurs, light-colored stools and dark urine accompany it. The liver may be tender and enlarged to 12 to 14 cm vertically. The spleen is enlarged and palpable in a few patients; the posterior cervical lymph nodes may also be enlarged. Subclinical episodes also occur frequently.

Assessment and Diagnostic Findings

HBV is a deoxyribonucleic acid (DNA) virus composed of the following antigenic particles:

- HBcAg—hepatitis B core antigen (antigenic material in an inner core)
- HBsAg—hepatitis B surface antigen (antigenic material on the viral surface, a marker of active replication and infection)

- HBeAg—an independent protein circulating in the blood
- HBxAg—gene product of X gene of HBV DNA

Each antigen elicits its specific antibody and is a marker for different stages of the disease process:

- anti-HBc—antibody to core antigen of HBV; persists during the acute phase of illness; may indicate continuing HBV in the liver
- anti-HBs—antibody to surface determinants on HBV; detected during late convalescence; usually indicates recovery and development of immunity
- anti-HBe—antibody to hepatitis B e-antigen; usually signifies reduced infectivity
- anti-HBxAg—antibody to the hepatitis B x-antigen; may indicate ongoing replication of HBV

HBsAg appears in the circulation in 80% to 90% of infected patients 1 to 10 weeks after exposure to HBV and 2 to 8 weeks before the onset of symptoms or an increase in transferase levels. Patients with HBsAg that persists for 6 months or longer after acute infection are considered to be HBsAg carriers (Chi et al., 2018; Sedhom, 2018). HBeAg is the next antigen of HBV to appear in the serum. It usually appears within 1 week of the appearance of HBsAg but before changes in aminotransferase levels; it disappears from the serum within 2 weeks. HBV DNA, detected by polymerase chain reaction testing, appears in the serum at about the same time as HBeAg. HBcAg is not always detected in the serum in HBV infection.

In the United States, the number of cases of chronic HBV is estimated to be 0.8 to 1.4 million persons. However, an accurate estimate is difficult to obtain because there is no national chronic-hepatitis surveillance program (CDC, 2017; Younossi, Stepanova, Younossi, et al., 2019). In 2013, males who died with HBV had a mortality rate that was nearly three times the mortality rate of females who died with HBV (0.8 deaths/100,000 population compared to 0.3 deaths/100,000 population). From 2009 to 2013, HBV-related mortality remained relatively stable for males and females (CDC, 2017). In 2015, hepatitis B resulted in an estimated 887,000 deaths worldwide, mostly from cirrhosis and HCC (World Health Organization [WHO], 2019).

Prevention

Prevention of hepatitis B transmission requires a multifaceted approach, including public health interventions and education as well as programs to foster immunization against this virulent virus in an effort to reduce the disease burden.

Preventing Transmission

Continued screening of blood donors for the presence of hepatitis B antigen (HBsAg) further decreases the risk of transmission by blood transfusion. The use of disposable syringes, needles, and lancets and the introduction of needleless IV administration systems have reduced the risk of spreading this infection from one patient to another or to health care personnel during the collection of blood samples or the administration of parenteral therapy. In the clinical laboratory and the hemodialysis unit, work areas are disinfected daily. Gloves are worn when handling all blood and body fluids, as well as HBsAg-positive specimens, or when there is potential exposure to blood (e.g., blood drawing) or to patients' secretions. Eating is prohibited in the laboratory and in other areas exposed to secretions, blood, or blood products. Patient education regarding the nature of the disease, its infectiousness, and prognosis is a critical factor in preventing transmission and protecting contacts (see [Chart 43-5](#)).

Active Immunization: HBV

Active immunization is recommended for people who are at high risk for HBV (e.g., health care personnel, patients undergoing hemodialysis). In addition, people with HCV and other chronic liver diseases should receive the vaccine. In 2018, the **Advisory Committee on Immunization Practices (ACIP)** recommended the use of a newly licensed hepatitis B vaccine, HEPLISAV-B, for people over the age of 18. This new vaccine is administered in two doses given 1 month apart. The decreased number of doses and abbreviated time period between doses may increase the rates of completion of the full vaccine series (Chi et al., 2018).

Prior to 2019, a yeast-recombinant hepatitis B vaccine was used to provide active immunity and has shown rates of protection greater than 90% in healthy people (Chan, Wong, Qin, et al., 2016; Terrault, Lok, McMahon, et al., 2018). Although antibody levels may become low or undetectable, immunologic memory may remain intact for at least 5 to 10 years. Measurable levels of antibodies may not be essential for protection. In general, in those with normal immune systems, booster doses are not required, and no data support the use of booster doses of hepatitis B vaccine among people who are immunocompetent and have responded to the vaccination series. However, booster doses are recommended for people who are immunocompromised (Chan et al., 2016; Terrault et al., 2018). A hepatitis B vaccine prepared from plasma of humans chronically infected with HBV is used only rarely in patients who are immunodeficient or allergic to recombinant yeast-derived vaccines.

Hepatitis B vaccines should be administered to adults in the deltoid muscle. Antibody response may be measured by anti-HBs levels 1 to 3 months after completion of the basic course of vaccine, but this testing is not routine and is not currently recommended. People who do not respond may benefit from additional doses (Terrault et al., 2018).

People at high risk, including nurses and other health care personnel exposed to blood or blood products, should receive active immunization. Health care workers who have had frequent contact with blood are screened for anti-HBs to determine whether immunity is already present from previous exposure. The vaccine produces active immunity to HBV in 90% to 95% of healthy people (Chi et al., 2018; Friedman & Martin, 2018; Terrault et al., 2018). It does not provide protection to those already exposed to HBV nor does it provide protection against other types of viral hepatitis.

Because HBV infection is frequently transmitted sexually, hepatitis B vaccination is recommended for all people who are unvaccinated and are being evaluated for a sexually transmitted infection (STI). It is also recommended for those with a history of an STI, people with multiple sex partners, people who have sex with people who use injection drugs, and men who are sexually active and have sex with other men (CDC, 2017; Chi et al., 2018; Terrault et al., 2018).

Universal childhood vaccination for hepatitis B prevention has been instituted in the United States, and universal vaccination of all infants is encouraged. Catch-up vaccination is recommended for all children and prepubertal adolescents up to the age of 19 years who have not been previously immunized (Feldman et al., 2016). Development of chronic carrier states has not been reported in adult responders to the vaccine.

Passive Immunity: Hepatitis B Immune Globulin

Hepatitis B immune globulin (HBIG) provides passive immunity to HBV and is indicated for people exposed to HBV who have never had hepatitis B and have never received hepatitis B vaccine. Specific indications for postexposure vaccine with HBIG include inadvertent exposure to HBAg-positive blood through percutaneous (needlestick) or transmucosal (splashes in contact with mucous membrane) routes, sexual contact with people positive for HBAg, and perinatal exposure (infants born to HBV-infected mothers should receive HBIG within 12 hours after delivery). HBIG is prepared from plasma selected for high titers of anti-HBs. Prompt immunization with HBIG (within hours to a few days after exposure to hepatitis B) increases the likelihood of protection. Both active and passive immunization are recommended for people who have been exposed to HBV through sexual contact or through the percutaneous or transmucosal routes. If HBIG and hepatitis B vaccines are given at the same time, separate sites and separate syringes should be used. HBIG is considered very safe, and there has been no evidence that infectious diseases have been transmitted due its administration (Chi et al., 2018; Terrault et al., 2018).

Medical Management

Goals are to minimize infectivity and liver inflammation and decrease symptoms. Of all the agents that have been used to treat chronic type B viral hepatitis, alpha-interferon is the single modality of therapy that offers the most promise. A regimen of 5 million U daily or 10 million U three times weekly for 16 to 24 weeks results in remission of disease in approximately one third of patients (Chan et al., 2016; Terrault et al., 2018). A prolonged course of treatment may also have additional benefits and is under study. Interferon must be given by injection and has significant side effects, including fever, chills, anorexia, nausea, myalgias, and fatigue. Delayed side effects are more serious and may necessitate dosage reduction or discontinuation. These include bone marrow suppression, thyroid dysfunction, alopecia, and bacterial infections. Several recombinant forms of alpha-interferon are also available, including the pegylated form (peginterferon alfa-2a), with once-weekly dosing. Pegylated interferon, also referred to as peginterferon, has largely replaced standard interferon due to its dosing schedule (Terrault et al., 2018). The American Association for the Study of Liver Diseases (AASLD) recommends pegylated interferon, entecavir or tenofovir, as preferred initial therapy for adults with chronic hepatitis B (AASLD, 2020; Terrault et al., 2018). These two antiviral agents, entecavir and tenofovir, are oral nucleoside analogs approved for use in chronic hepatitis B in the United States. They are the currently recommended agents for patients with HBV-related decompensated cirrhosis (AASLD, 2020; Terrault et al., 2018). Studies have revealed improved seroconversion rates and loss of detectable virus, improved liver function, and reduced progression to cirrhosis with entecavir and tenofovir. These agents can also be used for patients with decompensated cirrhosis who are awaiting liver transplantation (Chan et al., 2016; Terrault et al., 2018). Patients with decompensated cirrhosis have such severely damaged liver parenchyma that normal liver function severely deteriorates, resulting in life-threatening ascites, encephalopathy, or variceal hemorrhage (Chan et al., 2016; Schiff et al., 2018).

Bed rest may be recommended until the symptoms of hepatitis have subsided. Activities are restricted until the hepatic enlargement and levels of serum bilirubin and liver enzymes have decreased. Gradually, increased activity is then allowed.

Adequate nutrition should be maintained. Proteins are not restricted. Protein intake should be 1.2 to 1.5 g/kg/day (Schiff et al., 2018; Yao et al., 2018). Measures to control the dyspeptic symptoms and general malaise include the use of antacids and antiemetic agents, but all medications should be avoided if vomiting occurs. If vomiting persists, the patient may require hospitalization and fluid therapy. Because of the mode of transmission, the patient is evaluated for other bloodborne diseases (e.g., human immune deficiency virus infection).

Nursing Management

Convalescence may be prolonged, with complete symptomatic recovery sometimes requiring 3 to 4 months or longer (Papadakis & McPhee, 2020). During this stage, gradual resumption of physical activity is encouraged after the jaundice has resolved.

The nurse identifies psychosocial issues and concerns, particularly the effects of separation from family and friends if the patient is hospitalized during the acute and infective stages. Even if not hospitalized, the patient will be unable to work and must avoid sexual contact. Planning is required to minimize social isolation. Planning that includes the family helps to reduce their fears and anxieties about the spread of the disease.

Promoting Home, Community-Based and Transitional Care



Educating Patients About Self-Care

Because of the prolonged period of convalescence, the patient and family must be prepared for care in the home. Provision for adequate rest and nutrition must be ensured. The nurse educates family members and friends who have had intimate contact with the patient about the risks of contracting HBV and makes arrangements for them to receive hepatitis B vaccine or HBIG as prescribed. Those at risk must be made aware of the early signs of HBV and of ways to reduce risk by avoiding all modes of transmission. Patients with all forms of hepatitis should avoid drinking alcohol (Chan et al., 2016; Friedman & Martin, 2018; Schiff et al., 2018; Wang, Cheng, & Kao, 2020).

Continuing and Transitional Care

Follow-up visits by a transitional or home health nurse may be needed to assess the patient's progress and answer family members' questions about disease transmission. During a home visit, the nurse assesses the patient's physical and psychological status and confirms that the patient and family understand the importance of adequate rest and nutrition. The nurse also reinforces previous education. Because of the risk of transmission through sexual activity, strategies to prevent exchange of body fluids are recommended, such as abstinence or the use of condoms. The nurse emphasizes the importance of keeping follow-up appointments and participating in other health promotion activities and recommended health screenings.

Hepatitis C Virus

Blood transfusions and sexual contact once accounted for most cases of HCV in the United States, but other parenteral means, such as sharing of contaminated needles by those who use IV or injection drugs and unintentional needlesticks and other injuries in health care workers now account for a significant number of cases. In 2017, a total of 3186 cases of acute hepatitis C were reported to the CDC. After adjusting for under-ascertainment and under-reporting, an estimated 44,300 acute hepatitis C cases occurred in 2017. Approximately 2.5 million people in the United States are living with HCV, making it the most common chronic bloodborne infection nationally, though many of those infected are unaware of the disease (CDC, 2017). The highest prevalence of HCV is in adults 40 to 59 years of age; in this age group, its prevalence is highest among African Americans. In 2017, 17,253 U.S. death certificates had HCV recorded as an underlying or contributing cause of death but it has been suggested that deaths from this cause are underestimated (CDC, 2017; Houghton, 2019). HCV is the underlying cause of about one third of cases of HCC, and it is one of the most common reason for liver transplantation (CDC, 2017; Houghton, 2019).

People who are at particular risk for HCV include those who use IV or injection drugs, people who are sexually active with multiple partners, patients receiving frequent transfusions, those who require large volumes of blood, and health care personnel (see [Chart 43-8](#)). The incubation period is variable and may range from 15 to 160 days. The clinical course of acute HCV is similar to that of HBV; symptoms are usually mild or absent. However, a chronic carrier state occurs frequently, and there is an increased risk of chronic liver disease, including cirrhosis or liver cancer, after HCV. It is for this reason that those in high- and moderate-risk groups as well as those known to have high prevalence rates (e.g., those born in certain countries or regions) be screened for HCV as well as for HBV (CDC, 2017; Houghton, 2019; Schiff et al., 2018). Small amounts of alcohol taken regularly appear to cause progression of the disease. Therefore, alcohol and medications that may affect the liver should be avoided.

Chart 43-8 RISK FACTORS

Hepatitis C

- Children born to women infected with hepatitis C virus
- Health care and public safety workers after needlestick injuries or mucosal exposure to blood
- Multiple contacts with a person who is infected with hepatitis C virus
- Multiple sex partners, history of sexually transmitted infection, unprotected sex
- Past/current illicit IV/injection drug use
- Recipient of blood products or organ transplant before 1992 or clotting factor concentrates before 1987

Adapted from Kumar, V., Abbas, A. K., Fausto, N., et al. (2014). *Robbins and Cotran pathologic basis of disease* (9th ed.). Philadelphia, PA: Saunders Elsevier.

There is no benefit from rest, diet, or vitamin supplements. The treatment for HCV infection has evolved since the introduction of highly effective HCV protease inhibitor therapies in 2011. Currently available therapies can achieve sustained virologic response (SVR) defined as the absence of detectable virus 12 weeks after completion of treatment; an SVR is indicative of a cure of HCV infection. Over 90% of persons infected with HCV can be cured of HCV infection regardless of HCV genotype (there are over 67 identified subtypes but type 1 is most common), with 8 to 12 weeks of oral therapy (CDC, 2017). HCV direct-acting antivirals (DAA) including simeprevir plus sofosbuvir, ledipasvir-sofosbuvir, and ombitasvir-paritaprevir-ritonavir packaged with dasabuvir have fewer side effects, shorter treatment durations, and higher cure rates than previously recommended antiviral agents. A newer dual combination that includes another derivative of daclatasivir with sofosbuvir is equally effective against all HCV genotypes (Chan et al., 2016; Houghton, 2019). Prescribers must take into account the degree of cirrhosis in individual patients in order to determine the most appropriate protease inhibitor. The American Association for the Study of Liver Diseases (AASLD, 2020) recommends regimens of glecaprevir/pibrentasvir or sofosbuvir/velpatasvir for those patients who meet the criteria for simplified HCV treatment that includes treatment-naïve adults without cirrhosis or those with compensated cirrhosis. The choice of medication is dependent on the genotype as well (AASLD, 2020).

There are HCV treatments in development that are expected to be more effective in curing the disease and better tolerated with fewer adverse effects and contraindications (Houghton, 2019). As a result of the rapidly changing landscape for treatment of viral infections, it is important to monitor related websites for the most up-to-date recommendations (CDC, 2017; Houghton,

2019). A key challenge remains how to deliver the currently expensive DAA drugs to all global carriers of HCV (Houghton, 2019).

Screening of blood has reduced the incidence of HCV associated with blood transfusion, and public health programs are helping to reduce the number of cases associated with shared needles in IV or injection drug use (see [Chart 43-5](#)).

Hepatitis D Virus

Hepatitis D virus (delta agent) infection occurs in some cases of hepatitis B. Because the virus requires HBsAg for its replication, only people with hepatitis B are at risk for hepatitis D. Anti-delta antibodies in the presence of HBAg on testing confirm the diagnosis. Hepatitis D is common among those who use IV or injection drugs, patients undergoing hemodialysis, and recipients of multiple blood transfusions. Sexual contact with those who have hepatitis B is considered to be an important mode of transmission of hepatitis B and D. The incubation period varies between 30 and 150 days (Schiff et al., 2018).

The symptoms of hepatitis D are similar to those of hepatitis B, except that patients are more likely to develop acute hepatic failure and to progress to chronic active hepatitis and cirrhosis. Treatment is similar to that of other forms of hepatitis. Currently, interferon alfa is the only licensed drug available in the treatment for HDV infection. The rate of recurrence is high, and the efficacy of interferon is related to the dose and duration of treatment. High-dose, long-duration therapy for at least a year is recommended (Friedman & Martin, 2018; Schiff et al., 2018; Sedhom, D'Souza, John, et al., 2018).

Hepatitis E Virus

It is believed that HEV is transmitted by the fecal–oral route, principally through contaminated water in areas with poor sanitation. The incubation period is variable, estimated to range between 15 and 65 days. In general, hepatitis E resembles hepatitis A. It has a self-limited course with an abrupt onset. Jaundice is almost always present. Chronic forms do not develop.

Avoiding contact with the virus through good hygiene, including handwashing, is the major method of prevention of hepatitis E. The effectiveness of immune globulin in protecting against HEV is uncertain.

Hepatitis G Virus and GB Virus-C

It has long been believed that there is another non-A–E agent causing hepatitis in humans. The incubation period for posttransfusion hepatitis is 14 to 145 days—too long for hepatitis B or C. In the United States, about 5% of chronic liver disease remains cryptogenic (i.e., does not appear to be autoimmune or viral in origin), and 50% of these patients have received blood transfusions before developing disease. Therefore, another form of hepatitis, referred to as hepatitis G virus (HGV) or GB virus-C (GBV-C), has been described; these are thought to be two different isolates of the same virus, which are percutaneously transmitted. Autoantibodies are absent.

The clinical significance of this virus remains uncertain. Risk factors are similar to those for hepatitis C. There is no clear relationship between HGV/GBV-C infection and progressive liver disease. Persistent infection does occur but does not affect the clinical course (Papadakis & McPhee, 2020; Sedhom et al., 2018).

NONVIRAL HEPATITIS

Certain chemicals have toxic effects on the liver and produce acute liver cell necrosis or toxic hepatitis when inhaled, injected parenterally, or taken by mouth. Some chemicals commonly implicated in this disease include carbon tetrachloride and phosphorus. These substances are true hepatotoxins. Many medications can induce hepatitis but are only sensitizing rather than toxic. Drug-induced hepatitis is similar to acute viral hepatitis, but parenchymal destruction tends to be more extensive. Medications that can lead to hepatitis include isoniazid, halothane, acetaminophen, methyldopa, and certain antibiotics, antimetabolites, and anesthetic agents.

Toxic Hepatitis

At the onset of disease, toxic hepatitis resembles viral hepatitis. Obtaining a history of exposure to hepatotoxic chemicals, medications, botanical agents, or other toxic agents assists in early treatment and removal of the causative agent. Anorexia, nausea, and vomiting are the usual symptoms; jaundice and hepatomegaly are noted on physical assessment. Symptoms are more intense for the more severely toxic patient.

Recovery from acute toxic hepatitis is rapid if the hepatotoxin is identified early and removed or if exposure to the agent has been limited. Recovery is unlikely if there is a prolonged period between exposure and onset of symptoms. There are no effective antidotes. The fever rises; the patient becomes toxic and prostrated. Vomiting may be persistent, with the emesis containing blood. Clotting abnormalities may be severe, and hemorrhages may appear under the skin. The severe GI symptoms may lead to vascular collapse. Delirium, coma, and seizures develop, and within a few days the patient may

die of acute hepatic failure (discussed later) unless they receive a liver transplant.

Short of liver transplantation, few treatment options are available. Therapy is directed toward restoring and maintaining fluid and electrolyte balance, blood replacement, and comfort and supportive measures. A few patients recover from acute toxic hepatitis only to develop chronic liver disease. If the liver heals, there may be scarring, followed by postnecrotic cirrhosis.

Drug-Induced Hepatitis

Drug-induced liver disease is the most common cause of acute liver failure, accounting for more than 50% of all cases in the United States (Schiff et al., 2018; Stravitz & Lee, 2019; Thomas & Lewis, 2018). Manifestations of sensitivity to a medication may occur on the first day of its use or not until several months later. Usually, the onset is abrupt, with chills, fever, rash, pruritus, arthralgia, anorexia, and nausea. Later, there may be jaundice, dark urine, and an enlarged and tender liver. After the offending medication is withdrawn, symptoms may gradually subside. However, reactions can be severe, or even fatal, even if the medication is stopped. If fever, rash, or pruritus occurs from any medication, its use should be stopped immediately.

Although any medication can affect liver function, the use of acetaminophen (found in many OTC medications used to treat fever and pain) has been identified as the leading cause of acute liver failure (Schiff et al., 2018; Stravitz & Lee, 2019). Other causes commonly associated with liver injury include many anesthetic agents, medications used to treat rheumatic and musculoskeletal disease, antidepressants, psychotropic medications, anticonvulsants, and antituberculosis agents (Schiff et al., 2018; Stravitz & Lee, 2019; Thomas & Lewis, 2018).

A short course of high-dose corticosteroids may be used in patients with severe hypersensitivity reactions, although its efficacy is uncertain. Liver transplantation is an option for drug-induced hepatitis, but outcomes may not be as successful as with other causes of liver failure.



Acute Liver Failure

Acute hepatic failure or **acute liver failure** (ALF) is the clinical syndrome of sudden and severely impaired liver function in a person who was previously healthy. The definition of ALF includes neurologic dysfunction, an elevated prothrombin time and international normalized ratio (PT/INR) ≥ 1.5 , no prior evidence of liver disease, and a disease course of ≤ 26 weeks (Friedman & Martin, 2018; Maher & Schreibman, 2018; Montrief, Koyfman, & Long, 2019; Schiff et al., 2018). The time from the onset of symptoms such as jaundice to

the development of hepatic encephalopathy categorizes the different forms of acute liver failure: a very rapid injury (within hours) is referred to as hyperacute liver failure; and a slower, immune-based injury (days to weeks) is considered acute or subacute (Maher & Schreibman, 2018; Stravitz & Lee, 2019). In hyperacute liver failure, the duration of jaundice before the onset of encephalopathy is 0 to 7 days; in acute liver failure, it is 8 to 28 days; and in subacute liver failure, it is 28 to 72 days. The prognosis for acute hepatic failure is much worse than for chronic liver failure. However, in acute failure, the hepatic lesion is potentially reversible, and survival rates are approximately 20% to 50%, depending greatly on the cause. Those who do not survive die of massive hepatocellular injury and necrosis (Maher & Schreibman, 2018; Montrief et al., 2019; Stravitz & Lee, 2019).

Viral hepatitis is a common cause of ALF; other causes include toxic medications (e.g., acetaminophen) and chemicals (e.g., carbon tetrachloride), metabolic disturbances (e.g., Wilson disease, a hereditary syndrome with deposition of copper in the liver), and structural changes (e.g., Budd–Chiari syndrome, an obstruction to outflow in major hepatic veins) (Maher & Schreibman, 2018; Stravitz & Lee, 2019).

Jaundice and profound anorexia may be the initial reasons the patient seeks health care. ALF is often accompanied by coagulation defects, kidney disease and electrolyte disturbances, cardiovascular abnormalities, infection, hypoglycemia, encephalopathy, and cerebral edema (Maher & Schreibman, 2018; Montrief et al., 2019; Stravitz & Lee, 2019).

The key to optimized treatment is rapid recognition of ALF and intensive intervention. Supporting the patient in the ICU and assessing the indications for and feasibility of liver transplantation are hallmarks of management. The use of antidotes for certain conditions may be indicated, such as *N*-acetylcysteine for acetaminophen toxicity and penicillin for mushroom poisoning. Treatment modalities may include plasmapheresis to correct coagulopathy, to reduce serum ammonia levels, and to stabilize the patient awaiting liver transplantation, and prostaglandin therapy to enhance hepatic blood flow. Although these treatment modalities may be implemented, no evidence exists indicating any clinical improvement with their use (Maher & Schreibman, 2018; Montrief et al., 2019; Stravitz & Lee, 2019; Wendon et al., 2017). Hepatocytes within synthetic fiber columns have been tested as liver support systems (liver assist devices) to provide a bridge to transplantation.

Research into interventions for ALF has begun to focus on techniques that combine the efficacy of a whole liver with the convenience and biocompatibility of hemodialysis. The acronyms ELAD (*extracorporeal liver assist devices*) and BAL (*bioartificial liver*) have been used to describe these hybrid devices. These short-term devices, which remain experimental, may help patients survive until transplantation is possible (Villarreal & Sussman, 2019; Wendon et al., 2017). The BAL device exposes separated plasma to a

cartridge containing porcine liver cells after the plasma has flowed through a charcoal column that removes substances toxic to hepatocytes. The ELAD exposes whole blood to cartridges containing human hepatoblastoma cells, resulting in removal of toxic substances. These approaches appear promising and have had success in animal studies. In human clinical application, the use of various BAL systems has resulted in improved neurologic and biochemical parameters. Adding albumin to extracorporeal dialysis in a process known as molecular adsorbent recirculating system (MARS), and therapeutic plasma exchanges (TPE) have been used to remove protein-bound toxins and is potentially useful in unstable patients with ALF or acute or chronic liver disease (Bañares et al., 2019; Larsen, 2019; Wendon et al., 2017).

In patients who have ALF with stage 4 encephalopathy (see [Table 43-3](#)), there is a high risk of cerebral edema, a life-threatening complication. The cause is not fully understood, although disruption of the blood–brain barrier and plasma leakage into the cerebrospinal fluid may be one cause. An increase in the intracellular osmolarity within cerebral astrocyte cells, possibly related to increased sodium and glutamine in these cells, may be another (Montrief et al., 2019; Stravitz & Lee, 2019). These patients require intracranial pressure monitoring. Measures to promote adequate cerebral perfusion include careful fluid balance and hemodynamic assessments, a quiet environment, and diuresis with mannitol, an osmotic diuretic.

The use of pharmacologic neuromuscular blockade (NMB) and sedation is indicated to prevent surges in intracranial pressure related to agitation. Other support measures include monitoring for and treating hypoglycemia, coagulopathies, and infection. Despite these treatment modalities, the mortality rate remains high. Consequently, liver transplantation (discussed later) is the treatment of choice for ALF.

HEPATIC CIRRHOSIS

Cirrhosis is a chronic disease characterized by replacement of normal liver tissue with diffuse fibrosis that disrupts the structure and function of the liver. There are three types of cirrhosis or scarring of the liver:

- Alcoholic cirrhosis, in which the scar tissue characteristically surrounds the portal areas. This is most frequently caused by chronic alcoholism and is the most common type of cirrhosis.
- Postnecrotic cirrhosis, in which there are broad bands of scar tissue. This is a late result of a previous bout of acute viral hepatitis.
- Biliary cirrhosis, in which scarring occurs in the liver around the bile ducts. This type of cirrhosis usually results from chronic biliary obstruction and cholangitis (bile duct infection); it is much less common.

The portion of the liver chiefly involved in cirrhosis consists of the portal and the periportal spaces, where the bile canaliculi of each lobule communicate to form the liver bile ducts. These areas become the sites of inflammation, and the bile ducts become occluded with inspissated (thickened) bile and pus. The liver attempts to form new bile channels; hence, there is an overgrowth of tissue made up largely of disconnected, newly formed bile ducts and surrounded by scar tissue.

Pathophysiology

The logo for Concepts in Action features the word "CONCEPTS" stacked above the word "ACTION". A stylized yellow sun-like icon with rays is positioned between the two words.

CONCEPTS
ACTION

Several factors have been implicated in the etiology of cirrhosis. Nutritional deficiency with reduced protein intake contributes to liver destruction in cirrhosis, but excessive alcohol intake is the major causative factor in fatty liver and its consequences. However, cirrhosis can occur in people who do not consume alcohol and in those who consume a normal diet and have a high alcohol intake.

Some people appear to be more susceptible than others to this disease, whether or not they have alcoholism or are malnourished. Other factors may play a role, including exposure to certain chemicals (carbon tetrachloride, chlorinated naphthalene, arsenic, or phosphorus) or infectious schistosomiasis. Twice as many men as women are affected, although, for unknown reasons, women are at greater risk for development of alcohol-induced liver disease. Most patients are between 40 and 60 years of age. Alcohol-associated cirrhosis contributes to up to 50% of the overall cirrhosis burden in the United States and worldwide (Lucey, 2019). From 1999 to 2016 in the United States, annual deaths from cirrhosis increased by 65% to approximately 35,000 (Baki, 2019; Tapper, 2018).

Alcoholic cirrhosis is characterized by episodes of necrosis involving the liver cells, which sometimes occur repeatedly throughout the course of the disease. The destroyed liver cells are gradually replaced by scar tissue. Eventually, the amount of scar tissue exceeds that of the functioning liver tissue. Islands of residual normal tissue and regenerating liver tissue may project from the constricted areas, giving the cirrhotic liver its characteristic hobnail appearance. The disease usually has an insidious onset and a protracted course, occasionally proceeding over a period of 30 or more years.

The prognoses for different forms of cirrhosis caused by various liver diseases have been investigated in several studies. Of the many prognostic indicators, the Child–Pugh classification seems most useful in predicting the outcome of patients with liver disease (see [Table 43-5](#)). It is also used in choosing management approaches.

Clinical Manifestations

Signs and symptoms of cirrhosis increase in severity as the disease progresses, and severity is used to categorize the disorder as compensated or decompensated cirrhosis (see [Chart 43-9](#)). Compensated cirrhosis, with its less severe, often vague symptoms, may be discovered secondarily at a routine physical examination. The hallmarks of decompensated cirrhosis result from failure of the liver to synthesize proteins, clotting factors, and other substances and manifestations of portal hypertension (see earlier sections of this chapter for clinical manifestations and management of portal hypertension, ascites, varices, and hepatic encephalopathy).

TABLE 43-5 Modified Child–Pugh Classification of the Severity of Liver Disease

Parameter	Points Assigned		
	1	2	3
Ascites	Absent	Slight	Moderate
Bilirubin (mg/dL)	≤2	2–3	>3
Albumin (g/dL)	>3.5	2.8–3.5	<2.8
Prothrombin time (seconds over control)	1–3	4–6	>6
Encephalopathy	None	Grade 1–2	Grade 3–4

Total score of 5–6, grade A; 7–9, grade B; 10–15, grade C.

Adapted from Feldman, M., Friedman, L. S., & Brandt, L. J. (2016). *Sleisinger & Fordtran's gastrointestinal & liver disease* (10th ed.). Philadelphia, PA: Saunders Elsevier.

Chart 43-9 ASSESSMENT



Assessing for Cirrhosis

Be alert to the following signs and symptoms:

Compensated

- Abdominal pain
- Ankle edema
- Firm, enlarged liver
- Flatulent dyspepsia
- Intermittent mild fever
- Palmar erythema (reddened palms)
- Splenomegaly
- Unexplained epistaxis
- Vague morning indigestion
- Vascular spiders

Decompensated

- Ascites
- Clubbing of fingers
- Continuous mild fever
- Epistaxis
- Gonadal atrophy
- Hypotension
- Jaundice
- Muscle wasting
- Purpura (due to decreased platelet count)
- Sparse body hair
- Spontaneous bruising
- Weakness
- Weight loss
- White nails

Adapted from Lee, S. S., & Moreau, R. (2015). *Cirrhosis: A practical guide to management* (1st ed.). Hoboken, NJ: John Wiley & Sons, Ltd.

Liver Enlargement

Early in the course of cirrhosis, the liver tends to be large, and the cells are loaded with fat. The liver is firm and has a sharp edge that is noticeable on palpation. Abdominal pain may be present because of recent, rapid enlargement of the liver, which produces tension on Glisson capsule (the fibrous covering of the liver). Later in the disease, the liver decreases in size as scar tissue contracts the liver tissue. The liver edge, if palpable, is nodular.

Portal Obstruction and Ascites

Portal obstruction and ascites—late manifestations of cirrhosis—are caused partly by chronic failure of liver function and partly by obstruction of the portal circulation. Almost all of the blood from the digestive organs is collected in the portal veins and carried to the liver. Because a cirrhotic liver does not allow free blood passage, blood backs up into the spleen and the GI tract, and these organs become the seat of chronic passive congestion—that is, they are stagnant with blood and therefore cannot function properly. Indigestion and altered bowel function result. Fluid rich in protein may accumulate in the peritoneal cavity, producing ascites. This can be detected through percussion for shifting dullness or a fluid wave (see Fig. 43-6).

Infection and Peritonitis

Bacterial peritonitis may develop in patients with cirrhosis and ascites in the absence of an intra-abdominal source of infection or an abscess. This condition is referred to as spontaneous bacterial peritonitis (SBP). Bacteremia due to translocation of intestinal flora is believed to be the most likely route of infection. Clinical signs may be absent, necessitating paracentesis for diagnosis. Antibiotic therapy is effective in the treatment and prevention of recurrent episodes of SBP. The development of SBP is a precipitating factor to the onset of hepatorenal syndrome, a form of acute kidney injury unresponsive to administration of fluid or diuretic agents (Adebayo, Neong, & Wong, 2019; Schiff et al., 2018). This type of kidney disease is characterized by a lack of pathologic changes in the kidney; there is no evidence of dehydration or obstruction of the urinary tract or any other renal disorder.

Gastrointestinal Varices

The obstruction to blood flow through the liver caused by fibrotic changes also results in the formation of collateral blood vessels in the GI system and shunting of blood from the portal vessels into blood vessels with lower pressures. As a result, the patient with cirrhosis often has prominent, distended abdominal blood vessels, called *caput medusae*, which are visible on abdominal inspection and distended blood vessels throughout the GI tract. The esophagus, stomach, and lower rectum are common sites of collateral blood vessels. These distended blood vessels form varices or hemorrhoids, depending on their location.

Because these vessels were not intended to carry the high pressure and volume of blood imposed by cirrhosis, they may rupture and bleed. Therefore, assessment must include observation for occult and frank bleeding from the GI tract.

Edema

Another late symptom of cirrhosis is edema, which is attributed to chronic liver failure. A reduced plasma albumin concentration predisposes the patient to the formation of edema. Although edema is generalized, it often affects the lower extremities, the upper extremities, and the presacral area. Facial edema is not typical. Overproduction of aldosterone occurs, causing sodium and water retention and potassium excretion.

Vitamin Deficiency and Anemia

Because of inadequate formation, use, and storage of certain vitamins (notably vitamins A, C, and K), signs of deficiency are common, particularly hemorrhagic phenomena associated with vitamin K deficiency. Chronic gastritis and impaired GI function, together with inadequate dietary intake and impaired liver function, account for the anemia that is often associated with cirrhosis. The patient's anemia, poor nutritional status, and poor state of health result in severe fatigue, which interferes with the ability to carry out routine activities of daily living.

Mental Deterioration

Additional clinical manifestations include deterioration of mental and cognitive function with impending hepatic encephalopathy and hepatic coma, as described previously. Serial neurologic assessment is indicated, including assessment of the patient's general behavior, cognitive abilities, orientation to time and place, and speech patterns.

Assessment and Diagnostic Findings

The extent of liver disease and the type of treatment are determined after review of the laboratory findings. The functions of the liver are complex, and many diagnostic tests provide information about liver function (see [Table 43-1](#)). The patient needs to know why these tests are being performed and how to cooperate.

In severe parenchymal liver dysfunction, the serum albumin level tends to decrease, and the serum globulin level rises. Enzyme tests indicate liver cell damage: serum alkaline phosphatase, AST, ALT, and GGT levels increase, and the serum cholinesterase level may decrease. Bilirubin tests are performed to measure bile excretion or retention; increased levels of bilirubin can occur with cirrhosis and other liver disorders. Prothrombin time is prolonged. Normal values for laboratory data are listed in Appendix A on [thePoint](#).

Ultrasound scanning is used to measure the difference in density of parenchymal cells and scar tissue. CT, MRI, radioisotope liver scans, and elastography studies give information about liver size, hepatic blood flow and obstruction and the presence of liver fibrosis. Diagnosis is confirmed by liver

biopsy. Arterial blood gas analysis may reveal a ventilation–perfusion imbalance and hypoxia.

Medical Management

Management of the patient with cirrhosis is usually based on the presenting symptoms. For example, antacids or H₂ antagonists are prescribed to decrease gastric distress and minimize the possibility of GI bleeding. Vitamins and nutritional supplements promote healing of damaged liver cells and improve the patient's general nutritional status. Potassium-sparing diuretic agents such as spironolactone or triamterene may be indicated to decrease ascites, if present; these diuretics are preferred because they minimize the fluid and electrolyte changes commonly seen with other agents. An adequate diet and avoidance of alcohol are essential. Although the fibrosis of the cirrhotic liver cannot be reversed, its progression may be halted or slowed by such measures.

Many medications possess antifibrotic activity for the treatment of cirrhosis. Some of these medications include colchicine, angiotensin system inhibitors, statins, diuretics including spironolactone, immunosuppressants, and glitazones such as pioglitazone or rosiglitazone. Angiotensin receptor blocker (ARB) medications also have antifibrogenic properties and may also be prescribed (Schiff et al., 2018).

Many advances have been made in the treatment of liver fibrosis. Understanding of the pathogenic mechanisms of liver disease and fibrogenesis has led to the recent increase in the number of clinical trials, particularly for patients with NASH. Fibrosis is a key predictor of liver mortality in NASH, and many studies have focused on evaluating the potential of drugs to reduce liver fibrogenesis. Drugs that target different pathways in NASH are, therefore, being evaluated in isolation or as combination therapy. Some of the antifibrotic medications currently under study can reduce injury and inflammation and include vitamin E and chemokine receptor (CCR2/CCR5) inhibitors. Peroxisome proliferator-activated receptor (PPAR) agonists have been shown to cause cell death of hepatic stellate cells that potentiate fibrosis. Farnesoid X receptor agonists, such as obeticholic acid, have been reported to prevent chronic inflammation and liver fibrosis (Manka Zeller, & Syn, 2019; Schiff et al., 2018).

Many patients who have ESLD with cirrhosis use the herb milk thistle (*Silybum marianum*) to treat jaundice and other symptoms. This herb has been used for centuries because of its healing and regenerative properties for liver disease. Silymarin from milk thistle has anti-inflammatory and antioxidant properties that may have beneficial effects, especially in hepatitis, alcohol-induced liver injury and HCC (Weiskirchen, Weiskirchen, & Tacke, 2018). The natural compound SAM-e (S-adenosylmethionine) may improve outcomes in liver disease by improving liver function, possibly through enhancing

antioxidant function. Primary biliary cirrhosis has been treated with ursodeoxycholic acid to improve liver function.

Nursing Management



Nursing management for the patient with cirrhosis of the liver is described in detail in [Chart 43-10](#). Nursing interventions are directed toward promoting patient's rest, improving nutritional status, providing skin care, reducing risk of injury, and monitoring and managing potential complications.

Promoting Rest

The patient with cirrhosis requires rest and other supportive measures to permit the liver to reestablish its functional ability. If the patient is hospitalized, weight and I&O are measured and recorded daily. The nurse adjusts the patient's position in bed for maximal respiratory efficiency, which is especially important if ascites is marked, because it interferes with adequate thoracic excursion. Oxygen therapy may be required in liver failure to oxygenate the damaged cells and prevent further cell destruction.

**Chart 43-10** **PLAN OF NURSING CARE**

The Patient with Impaired Liver Function

NURSING DIAGNOSIS: Activity intolerance associated with fatigue, lethargy, and malaise

GOAL: Patient reports decrease in fatigue and reports increased ability to participate in activities

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Assess level of activity tolerance and degree of fatigue, lethargy, and malaise when performing routine activities of daily living. 2. Assist with activities and hygiene when fatigued. 3. Encourage rest when fatigued or when abdominal pain or discomfort occurs. 4. Assist with selection and pacing of desired activities and exercise. 5. Provide diet high in carbohydrates with protein intake of 1.2 to 1.5 g/kg/day. 6. Administer supplemental vitamins (A, B complex, C, and K). 	<ol style="list-style-type: none"> 1. Provides baseline for further assessment and criteria for assessment of effectiveness of interventions. 2. Promotes exercise and hygiene within patient's level of tolerance. 3. Conserves energy and protects the liver. 4. Stimulates patient's interest in selected activities. 5. Provides calories for energy and protein for healing. 6. Provides additional nutrients. 	<ul style="list-style-type: none"> • Exhibits increased interest in activities and events • Participates in activities and gradually increases exercise within physical limits • Reports increased strength and well-being • Reports absence of abdominal pain and discomfort • Plans activities to allow ample periods of rest • Takes vitamins as prescribed

NURSING DIAGNOSIS: Impaired nutritional intake associated with abdominal distention, discomfort, and anorexia

GOAL: Positive nitrogen balance, no further loss of muscle mass; meets nutritional requirements

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Assess dietary intake and nutritional status through diet history and diary, daily weight measurements, and laboratory data. 2. Provide diet high in carbohydrates with protein intake of 1.2 to 1.5 g/kg/day. 3. Assist patient in identifying low-sodium foods. 4. Elevate the head of the bed during meals. 5. Provide oral hygiene before meals and pleasant environment for meals at mealtime. 6. Offer smaller, more frequent meals (6/day). 7. Encourage patient to eat meals and supplementary feedings. 8. Provide attractive meals and an aesthetically pleasing setting at mealtime. 	<ol style="list-style-type: none"> 1. Identifies deficits in nutritional intake and adequacy of nutritional state. 2. Provides calories for energy and protein for healing. 3. Reduces edema and ascites formation. 4. Reduces discomfort from abdominal distention and decreases sense of fullness produced by pressure of abdominal contents and ascites on the stomach. 5. Promotes positive environment and increased appetite; reduces unpleasant taste. 6. Decreases feeling of fullness, bloating. 7. Encouragement is essential for the patient with anorexia and gastrointestinal discomfort. 8. Promotes appetite and sense of well-being. 9. Eliminates "empty calories" and further damage from alcohol. 10. Reduces gastrointestinal symptoms and 	<ul style="list-style-type: none"> • Exhibits improved nutritional status by increased weight (without fluid retention) and improved laboratory data • States rationale for dietary modifications • Identifies foods high in carbohydrates and protein • Reports improved appetite • Participates in oral hygiene measures • Reports increased appetite; identifies rationale for smaller, frequent meals • Demonstrates intake of high-calorie diet; adheres to protein intake recommendations • Identifies foods and fluids that are nutritious and permitted on diet • Gains weight without increased edema or ascites formation

9. Eliminate alcohol.	discomforts that decrease the appetite and interest in food.	<ul style="list-style-type: none"> Reports increased appetite and well-being
10. Administer medications prescribed for nausea, vomiting, diarrhea, or constipation.	11. Promotes normal bowel pattern and reduces abdominal discomfort and distention.	<ul style="list-style-type: none"> Excludes alcohol from diet Takes medications for gastrointestinal disorders as prescribed
11. Encourage increased fluid intake and exercise if the patient reports constipation.		<ul style="list-style-type: none"> Reports normal gastrointestinal function with regular bowel function

NURSING DIAGNOSIS: Impaired skin integrity associated with pruritus from jaundice and edema

GOAL: Decrease potential for pressure injury development; breaks in skin integrity

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> Assess degree of discomfort related to pruritus and edema. Note and record degree of jaundice and extent of edema. 	<ol style="list-style-type: none"> Assists in determining appropriate interventions. Provides baseline for detecting changes and evaluating effectiveness of interventions. 	<ul style="list-style-type: none"> Exhibits intact skin without redness, excoriation, or breakdown Reports relief from pruritus Exhibits no skin excoriation from scratching
<ol style="list-style-type: none"> Keep patient's fingernails short and smooth. Provide frequent skin care; avoid the use of soaps 	<ol style="list-style-type: none"> Prevents infection and from scratching. Removes waste products from skin 	<ul style="list-style-type: none"> Uses nondrying soaps and lotions; states rationale for the use of nondrying soaps and lotions

<p>and alcohol-based lotions.</p> <p>5. Massage every 2 hours with emollients; turn every 2 hours.</p> <p>6. Initiate use of alternating-pressure mattress or low air loss bed.</p> <p>7. Recommend avoiding the use of harsh detergents.</p> <p>8. Assess skin integrity every 4–8 hours. Instruct patient and family in this activity.</p> <p>9. Restrict sodium as prescribed.</p> <p>10. Perform range-of-motion exercises every 4 hours; elevate edematous extremities whenever possible.</p>	<p>while preventing dryness of skin.</p> <p>5. Promotes mobilization of edema.</p> <p>6. Minimizes prolonged pressure on bony prominences susceptible to breakdown.</p> <p>7. May decrease skin irritation and need for scratching.</p> <p>8. Edematous skin and tissue have compromised nutrient supply and are vulnerable to pressure and trauma.</p> <p>9. Minimizes edema formation.</p> <p>10. Promotes mobilization of edema.</p>	<ul style="list-style-type: none"> • Turns self periodically; exhibits reduced edema of dependent parts of the body • Exhibits no areas of skin breakdown • Exhibits decreased edema; normal skin turgor
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NURSING DIAGNOSIS: Risk for injury associated with altered clotting mechanisms and altered level of consciousness

GOAL: Reduced risk of injury

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Assess level of consciousness and cognitive level. 2. Provide safe environment (pad side rails, remove obstacles in room, prevent falls). 3. Provide frequent surveillance to orient patient, and avoid the use of restraints. 4. Replace sharp objects (razors) with safer items. 5. Observe each stool for color, consistency, and amount. 6. Be alert to symptoms of anxiety, epigastric fullness, weakness, and restlessness. 7. Test each stool and emesis for occult blood. 8. Observe for hemorrhagic manifestations: ecchymosis, epistaxis, petechiae, and bleeding gums. 9. Record vital signs at frequent intervals, depending on patient 	<ol style="list-style-type: none"> 1. Assists in determining patient's ability to protect self and comply with required self-protective actions; may detect deterioration of hepatic function. 2. Minimizes falls and injury if falls occur. 3. Protects patient from harm while stimulating and orienting patient; the use of restraints may disturb patient further. 4. Avoids cuts and bleeding. 5. Permits detection of bleeding in gastrointestinal tract. 6. May indicate early signs of bleeding and shock. 7. Detects early evidence of bleeding. 8. Indicates altered clotting mechanisms. 	<ul style="list-style-type: none"> • Is oriented to time, place, and person • Exhibits no hallucinations and demonstrates no efforts to get up unassisted or to leave hospital • Exhibits no ecchymoses (bruises), cuts, or hematoma • Uses electric razor rather than sharp-edged razor • Exhibits absence of frank bleeding from gastrointestinal tract • Exhibits absence of restlessness, epigastric fullness, and other indicators of hemorrhage and shock • Exhibits negative results of test

acuity (every 1–4 hours).	9. Provides baseline and evidence of hypovolemia and hemorrhagic shock.	for occult gastrointestinal bleeding
10. Keep patient quiet, and limit activity.	10. Minimizes risk of bleeding and straining.	• Is free of ecchymotic areas or hematoma formation
11. Assist provider in passage of tube for esophageal balloon tamponade, if its insertion is indicated.	11. Promotes nontraumatic insertion of tube in a patient who is anxious and combative for immediate treatment of bleeding.	• Exhibits normal vital signs
12. Observe during blood transfusions.	12. Permits detection of transfusion reactions (risk increased with multiple blood transfusions needed for active bleeding from esophageal varices).	• Maintains rest and remains quiet if active bleeding occurs
13. Measure and record nature, time, and amount of vomitus.	13. Assists in evaluating extent of bleeding and blood loss.	• Identifies rationale for blood transfusions and measures to treat bleeding
14. Maintain patient in fasting state, if indicated.	14. Reduces risk of aspiration of gastric contents and minimizes risk of further trauma to esophagus and stomach by preventing vomiting.	• Uses measures to prevent trauma (e.g., uses soft toothbrush, blows nose gently, avoids bumps, falls, straining during defecation)
		• Experiences no side effects of medications
		• Takes all medications as prescribed
		• Identifies rationale for precautions

- with the use of all medications
- Adheres to prescribed treatment modalities

Nursing Interventions	Rationale	Expected Outcomes
<p>15. Administer vitamin K as prescribed.</p> <p>16. Remain with patient during episodes of bleeding.</p> <p>17. Offer cold liquids by mouth when bleeding stops (if prescribed).</p> <p>18. Institute measures to prevent trauma.</p> <ul style="list-style-type: none"> a. Maintain safe environment. b. Encourage gentle blowing of nose. c. Provide soft toothbrush, and avoid the use of toothpicks. d. Encourage intake of foods with high content of vitamin C. e. Apply cold compresses where indicated. f. Record location of bleeding sites. g. Use small-gauge needles for injections. 	<p>15. Promotes clotting by providing fat-soluble vitamin necessary for clotting.</p> <p>16. Reassures patient who is anxious and permits monitoring and detection of further needs of the patient.</p> <p>17. Minimizes risk of further bleeding by promoting vasoconstriction of esophageal and gastric blood vessels.</p> <p>18. Promotes safety of patient.</p> <ul style="list-style-type: none"> a. Minimizes risk of trauma and bleeding by avoiding falls and cuts, etc. b. Reduces risk of epistaxis (nosebleed) secondary to trauma and decreased clotting. c. Prevents trauma to oral mucosa 	<ul style="list-style-type: none"> with the use of all medications • Adheres to prescribed treatment modalities

19. Administer medications carefully; monitor for side effects.
- while promoting good oral hygiene.
- d. Promotes healing.
 - e. Minimizes bleeding into tissues by promoting local vasoconstriction.
 - f. Permits detection of new bleeding sites and monitoring of previous sites of bleeding.
 - g. Minimizes oozing and blood loss from repeated injections.
19. Reduces risk of side effects secondary to damaged liver's inability to detoxify (metabolize) medications normally.

Nursing Diagnosis: Disturbed body image associated with changes in appearance, sexual dysfunction, and role function

Goal: Patient verbalizes feelings consistent with improvement of body image and self-esteem

Nursing Interventions	Rationale	Expected Outcomes
<p>1. Assess changes in appearance and the meaning these changes have for patient and family.</p> <p>2. Encourage patient to verbalize reactions</p>	<p>1. Provides information for assessing impact of changes in appearance, sexual function, and role on the patient and family.</p>	<ul style="list-style-type: none"> • Verbalizes concerns related to changes in appearance, life, and lifestyle

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| <p>and feelings about these changes.</p> <p>3. Assess patient's and family's previous coping strategies.</p> <p>4. Assist and encourage patient to maximize appearance (such as strategies to limit the appearance of jaundice and ascites through careful selection of colors and type of clothing) and explore alternatives to previous sexual and role functions.</p> <p>5. Assist patient in identifying short-term goals.</p> <p>6. Encourage and assist patient in decision making about care.</p> <p>7. Identify with patient resources to provide additional support (counselor, spiritual advisor).</p> <p>8. Assist patient in identifying previous practices that may have been harmful to self (substance use disorder). Involve patient in goal setting, and provide positive feedback for accomplishments.</p> | <p>2. Enables patient to identify and express concerns; encourages patient and significant others to share these concerns.</p> <p>3. Permits encouragement of those coping strategies that are familiar to patient and have been effective in the past.</p> <p>4. Encourages patient to continue safe roles and functions while encouraging exploration of alternatives.</p> <p>5. Accomplishing these goals serves as positive reinforcement and increases self-esteem.</p> <p>6. Promotes patient's control of life and improves sense of well-being and self-esteem.</p> <p>7. Assists patient in identifying resources and accepting assistance from others when indicated.</p> <p>8. Recognition and acknowledgment of the harmful effects of these practices are necessary for identifying a healthier lifestyle.</p> | <ul style="list-style-type: none"> • Shares concerns with significant others • Identifies past coping strategies that have been effective • Uses past effective coping strategies to deal with changes in appearance, life, and lifestyle • Maintains good grooming and hygiene • Identifies short-term goals and strategies to achieve them • Takes an active role in decision making about self and care • Identifies resources that are not harmful • Verbalizes that some of previous lifestyle practices may have been harmful, if applicable • Uses healthy expressions of |
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frustration,
anger, anxiety

NURSING DIAGNOSIS: Comfortable with respect to enlarged tender liver and ascites

GOAL: Increased level of comfort

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none">1. Maintain bed rest when patient experiences abdominal discomfort.2. Administer antispasmodic and analgesic agents as prescribed.3. Observe, record, and report presence and character of pain and discomfort.4. Reduce sodium and fluid intake if prescribed.5. Prepare patient and assist with procedures for management of ascites such as paracentesis or TIPS procedure, if indicated.6. Encourage the use of distracting activities such as music, reading, or meditation.	<ol style="list-style-type: none">1. Reduces metabolic demands and protects the liver.2. Reduces irritability of the gastrointestinal tract and decreases abdominal pain and discomfort.3. Provides baseline to detect further deterioration of status and to evaluate interventions.4. Minimizes further formation of ascites.5. Removal of ascites fluid may decrease abdominal discomfort.6. Distraction may limit the perception of pain.	<ul style="list-style-type: none">• Reports pain and discomfort if present• Maintains bed rest and decreases activity in presence of pain• Takes antispasmodic and analgesic agents as indicated and as prescribed• Reports decreased pain and abdominal discomfort• Reduces sodium and fluid intake to prescribed levels if indicated to treat ascites• Exhibits decreased abdominal girth and appropriate weight changes

- Reports decreased discomfort after paracentesis or other procedure to manage ascites such as TIPS procedure

NURSING DIAGNOSIS: Hypervolemia associated with ascites and edema formation

GOAL: Restoration of normal fluid volume

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none">1. Restrict sodium and fluid intake if prescribed.2. Administer diuretic agents, potassium, and protein supplements as prescribed.3. Record intake and output every 1–8 hours depending on response to interventions and on patient acuity.4. Measure and record abdominal girth and weight daily.5. Explain rationale for sodium and fluid restriction.6. Prepare patient and assist with paracentesis or TIPS procedure, if indicated.	<ol style="list-style-type: none">1. Minimizes formation of ascites and edema.2. Promotes excretion of fluid through the kidneys and maintenance of normal fluid and electrolyte balance.3. Indicates effectiveness of treatment and adequacy of fluid intake.4. Monitors changes in ascites formation and fluid accumulation.5. Promotes patient's understanding of restriction and cooperation with it.6. Paracentesis will temporarily decrease amount of ascites present and a TIPS procedure will lower portal pressure and thus limit the accumulation of ascitic fluid.	<ul style="list-style-type: none">• Consumes diet low in sodium and within prescribed fluid restriction• Takes diuretic agents, potassium, and protein supplements as indicated without experiencing side effects• Exhibits increased urine output• Exhibits decreasing abdominal girth• Exhibits no rapid increase in weight• Identifies rationale for sodium and fluid restriction• Shows a decrease in ascites with decreased weight

NURSING DIAGNOSIS: Acute confusion associated with abnormal liver function and increased serum ammonia level

GOAL: Improved mental status; safety maintained; ability to cope with cognitive and behavioral changes

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Give frequent, small feedings of carbohydrates and protein. 2. Protect from infection. 3. Keep environment warm and draft free. 4. Pad the side rails of the bed. 5. Limit visitors. 	<ol style="list-style-type: none"> 1. Promotes consumption of adequate carbohydrates for energy requirements and protein for healing. 2. Minimizes risk of further increase in metabolic requirements. 3. Minimizes shivering, which would increase metabolic requirements. 4. Provides protection for the patient should hepatic coma and seizure activity occur. 5. Minimizes patient's activity and metabolic requirements. 	<ul style="list-style-type: none"> • Demonstrates an interest in events and activities in environment • Demonstrates normal attention span • Follows and participates in conversation appropriately • Is oriented to person, place, and time • Remains in bed when indicated • Experiences no seizures • No neurologic or respiratory depression • Patient develops no cognitive impairments, but if they develop, they are quickly identified and treated, enhancing the potential of recovery
<ol style="list-style-type: none"> 6. Provide careful nursing surveillance to 	<ol style="list-style-type: none"> 6. Provides close monitoring of new symptoms and minimizes trauma to 	<ul style="list-style-type: none"> • Patient and family describe adequate feelings of

	ensure patient's safety.	the patient who is confused.	coping and lowered anxiety. They demonstrate ability to listen and to make decisions as able
7.	Avoid opioids and barbiturates.	7. Prevents masking of symptoms of hepatic coma and prevents drug overdose secondary to reduced ability of the damaged liver to metabolize opioids and barbiturates; prevents respiratory depression.	• Patient and family communicate their feelings and their needs in a secure and caring environment
8.	Awaken at intervals (every 2–4 hours during daytime hours for the patient who is stable) to assess cognitive status.	8. Provides stimulation to the patient and opportunity for observing patient's level of consciousness.	
9.	Identify subtle changes in behavior or sleep-wake pattern (consistent staff caring for the patient enhances this assessment as they become familiar with patient's baseline).	9. These changes may herald worsening of encephalopathy, which requires rapid intervention, including medication.	
10.	Assess handwriting or drawing skill daily as indication of cognitive ability.	10. These changes may herald worsening of encephalopathy, which requires rapid intervention, including medication.	
11.	Encourage patient and family to participate in therapeutic strategies to enhance coping with episodes of mental deterioration.	11. Promoting activities such as listening to music, relaxation techniques, or preillness coping strategies can reduce anxiety.	
12.	Encourage patient and family to discuss feeling of fear, powerlessness, or emotional distress related to patient's	12. Actively listening demonstrates caring and concern.	

mental deterioration.

NURSING DIAGNOSIS: Risk for impaired thermoregulation: failure to maintain normal body temperature due to inflammatory process of cirrhosis or hepatitis

GOAL: Maintenance of normal body temperature, free from infection

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none">1. Record temperature regularly (every 4 hours).2. Encourage fluid intake.3. Apply cool sponges or ice bag for elevated temperature.4. Administer antibiotics as prescribed.5. Avoid exposure to infections by use of appropriate hand hygiene and limiting use of central lines and urinary catheters to the shortest period of time, only when they are necessary.6. Keep patient at rest while temperature is elevated.7. Assess for abdominal pain, tenderness.8. Use sterile technique for all invasive procedures.	<ol style="list-style-type: none">1. Provides baseline to detect fever and to evaluate interventions.2. Corrects fluid loss from perspiration and fever and increases patient's level of comfort.3. Promotes reduction of fever and increases patient's comfort.4. Ensures appropriate serum concentration of antibiotics to treat infection.5. Minimizes risk of further infection and further increases in body temperature and metabolic rate.6. Reduces metabolic rate.7. May occur with bacterial peritonitis.8. Many evidence-based practice guidelines (e.g., central venous catheter care) recommend the use of sterile technique to prevent health care-associated infections.	<ul style="list-style-type: none">• Exhibits normal temperature and reports absence of chills or sweating• Demonstrates adequate intake of fluids• Exhibits no evidence of local or systemic infection• Develops no health care-associated infections related to invasive procedures/lines

NURSING DIAGNOSIS: Impaired breathing associated with restriction of thoracic excursion secondary to ascites, abdominal distention, and fluid in the thoracic cavity

GOAL: Improved respiratory status

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Elevate head of bed to at least 30 degrees. 2. Conserve patient's strength by providing rest periods and assisting with activities. 3. Change position every 2 hours. 4. Assist with paracentesis, TIPS or thoracentesis, if indicated. <ul style="list-style-type: none"> a. Explain procedure and its purpose to patient. b. Have patient void before paracentesis. c. Support and maintain position during procedure. d. Record both the amount and the character of fluid aspirated. e. Observe for evidence of coughing, increasing dyspnea, or pulse rate. 5. Provide education to patients who may be discharged with an indwelling peritoneal drainage catheter for 	<ol style="list-style-type: none"> 1. Reduces abdominal pressure on the diaphragm and permits fuller thoracic excursion and lung expansion. 2. Reduces metabolic and oxygen requirements. 3. Promotes expansion and oxygenation of all areas of the lungs. 4. Paracentesis, TIPS, and thoracentesis (performed to remove fluid from the abdominal and thoracic cavities, respectively) may be frightening to the patient. <ul style="list-style-type: none"> a. Helps obtain patient's cooperation with procedures. b. Prevents inadvertent bladder injury. c. Prevents inadvertent organ or tissue injury. 	<ul style="list-style-type: none"> • Reports decreased shortness of breath • Reports increased strength and sense of well-being • Exhibits normal respiratory rate (12–18 breaths/min) with no adventitious sounds • Exhibits full thoracic excursion without shallow respirations • Exhibits normal arterial blood gases • Exhibits adequate oxygen saturation by pulse oximetry • Absence of confusion or cyanosis • Reports improved comfort level • Exhibits no complications

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| <p>palliation of refractory ascites.</p> <ol style="list-style-type: none"> Explain procedure and its purpose to patient. Explain care of the catheter and assessment of complications | <p>d. Provides record of fluid removed and indication of severity of limitation of lung expansion by fluid.</p> <p>e. Indicates irritation of the pleural space and evidence of pneumothorax or hemothorax.</p> <p>5. a. Explaining the purpose of the catheter demonstrates respect for the patient's self-determination</p> <p>b. Explanations promote patient adherence to the therapeutic regimen</p> | <p>related to the indwelling catheter, as appropriate</p> |
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COLLABORATIVE PROBLEM: Gastrointestinal bleeding and hemorrhage

GOAL: Absence of episodes of gastrointestinal bleeding and hemorrhage

Nursing Interventions	Rationale	Expected Outcomes
<p>1. Assess patient for evidence of gastrointestinal bleeding or hemorrhage. If bleeding does occur:</p> <ol style="list-style-type: none"> Monitor vital signs (blood pressure, pulse, respiratory rate) every 4 hours or more frequently, depending on acuity. 	<p>1. Allows early detection of signs and symptoms of bleeding and hemorrhage.</p>	<ul style="list-style-type: none"> Experiences no episodes of bleeding and hemorrhage Vital signs are within acceptable range for patient No evidence of bleeding from gastrointestinal tract

- b. Assess skin temperature, level of consciousness every 4 hours or more frequently, depending on acuity.
 - c. Monitor gastrointestinal secretions and output (emesis, stool for occult or obvious bleeding). Test emesis for blood once per shift and with any color change. Hematest each stool.
 - d. Monitor hematocrit and hemoglobin for trends and changes.

 - 2. Avoid activities that increase intra-abdominal pressure (straining, turning).
 - a. Avoid coughing/sneezing.
 - b. Assist patient to turn.
 - c. Keep all needed items within easy reach.
 - d. Use measures to prevent constipation such as adequate fluid intake, stool softeners.

 - 2. Minimizes increases in intra-abdominal pressure that could lead to rupture and bleeding of esophageal or gastric varices.

 - 3. Equipment, medications, and supplies will be readily available if patient experiences bleeding from ruptured
- Hematocrit and hemoglobin levels within acceptable limits
 - Turns and moves without straining and increasing intra-abdominal pressure
 - No straining with bowel movements
 - No further bleeding episodes if aggressive treatment of bleeding and hemorrhage was needed
 - Patient and family state rationale for treatments
 - Patient and family identify supports available to them
 - Patient and family describe signs and symptoms of a recurrent bleeding episode and identify needed action

- e. Ensure small meals.
- 3. Have equipment (balloon tamponade tube medications, IV fluids) available if indicated.
- 4. Assist with procedures and therapy needed to treat gastrointestinal bleeding and hemorrhage such as endoscopic variceal ligation (EVL) or sclerotherapy.
- 5. Monitor respiratory status every hour, and minimize risk of respiratory complications if balloon tamponade is needed.
- 6. Prepare patient physically and psychologically for other treatment modalities if needed.
- 7. Monitor patient for recurrence of bleeding and hemorrhage.
- 8. Keep family informed of patient's status.
- 9. Once recovered from bleeding episode, provide patient and family with information regarding signs and symptoms of gastrointestinal bleeding.
- esophageal or gastric varices.
- 4. Gastrointestinal bleeding and hemorrhage require emergency measures (e.g., insertion of Sengstaken–Blakemore tube™, administration of fluids, medications).
- 5. The patient is at high risk for respiratory complications, including asphyxiation if gastric balloon of tamponade tube ruptures or migrates upward.
- 6. The patient who experiences hemorrhage is very anxious and fearful; minimizing anxiety assists in control of hemorrhage.
- 7. Risk of rebleeding is high with all treatment modalities used to halt gastrointestinal bleeding.
- 8. Family members are likely to be anxious about the patient's status; providing information will reduce their anxiety level and

promote more effective coping.

9. Risk of rebleeding is high. Subtle signs may be more quickly identified.

COLLABORATIVE PROBLEM: Hepatic encephalopathy

GOAL: Absence of changes in cognitive status and of injury

Nursing Interventions	Rationale	Expected Outcomes
<p>1. Assess cognitive status every 4–8 hours.</p> <p>a. Assess patient's orientation to person, place, and time.</p> <p>b. Monitor patient's level of activity, restlessness, and agitation. Assess for presence of asterixis (flapping hand tremors) (see Fig. 43-12).</p> <p>c. Obtain and record daily sample of patient's handwriting or ability to construct a simple figure (e.g., star) (see Fig. 43-13).</p> <p>d. Assess neurologic signs (deep tendon reflexes, ability to follow instructions).</p> <p>2. Monitor medications to prevent administration of those that may precipitate hepatic encephalopathy</p>	<p>1. Data will provide baseline of patient's cognitive status and enable detection of changes.</p> <p>2. Medications are a common precipitating factor in development of hepatic encephalopathy in patients at risk.</p> <p>3. Increases in serum ammonia level are associated with hepatic encephalopathy and coma.</p>	<ul style="list-style-type: none"> • Remains awake, alert, and aware of surroundings • Is oriented to time, place, and person • Deep tendon reflexes remain within normal limits • Absence of asterixis • Exhibits no restlessness or agitation • Record of handwriting demonstrates no deterioration in cognitive function • States rationale for treatment used to prevent or treat hepatic encephalopathy • Demonstrates stable serum ammonia level within

	(sedative, hypnotic, analgesic agents).	acceptable limits
3.	Monitor laboratory data, especially serum ammonia level.	<ul style="list-style-type: none"> • Takes medications as prescribed • Breath sounds normal without adventitious sounds • Skin and tissue intact without evidence of pressure or breaks in integrity • Verbalizes understanding of need for treatments and procedures to promote recovery
Nursing Interventions	Rationale	Expected Outcomes
4. Notify primary provider of even subtle changes in patient's neurologic assessment, cognitive function, sleep pattern, or mood.	4. Allows early initiation of treatment of hepatic encephalopathy and prevention of hepatic coma.	
5. Administer medications prescribed to reduce serum ammonia level (e.g., lactulose, antibiotics, glucose, benzodiazepine antagonist [flumazenil] if indicated).	5. Reduces breakdown and conversion of protein to ammonia. Reduces serum ammonia level.	
6. Assess respiratory status, and initiate	6. The patient who develops hepatic coma is at risk for respiratory complications (i.e.,	

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| <p>measures to prevent complications.</p> <p>7. Protect patient's skin and tissue from pressure and breakdown.</p> <p>8. Provide support and active listening for patient and family as patient's mental status deteriorates.</p> | <p>pneumonia, atelectasis, infection).</p> <p>7. The patient in coma is at risk for skin breakdown and pressure injury formation.</p> <p>8. The patient with hepatic encephalopathy can experience episodes of mental deterioration due to liver failure. This can produce feelings of fear and anxiety.</p> |
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Rest reduces the demands on the liver and increases the liver's blood supply. Because the patient is susceptible to the hazards of immobility, efforts to prevent respiratory, circulatory, and vascular disturbances are initiated. These measures may help prevent such problems as atelectasis, pneumonia, venous thromboemboli formation, and pressure injuries. After nutritional status improves and strength increases, the nurse encourages the patient to increase activity gradually. Activity and mild exercise, as well as rest, are planned.

Improving Nutritional Status

The patient with cirrhosis without ascites, edema, or signs of impending hepatic coma should receive a nutritious, high-protein diet, if tolerated, supplemented by vitamins of the B complex, as well as A, C, and K. The nurse encourages the patient to eat. If ascites is present, small, frequent meals may be better tolerated than three large meals because of the abdominal pressure exerted by ascites.

The use of probiotics for the management of hepatic encephalopathy is currently the topic of ongoing research. Imbalance of the intestinal flora is not uncommon. Some research suggests that the oral ingestion of 1 cup of probiotic yogurt three times a day reduces intestinal flora imbalance by decreasing *Escherichia coli* counts and promoting the growth of non–urease-producing bacteria. This strategy is thought to then reduce ammonia levels and improve mental status (Acharya & Bajaj, 2018).

Patients with steatorrhea (fatty stools) should receive water-soluble forms of fat-soluble vitamins A, D, and E. Folic acid and iron are prescribed to prevent anemia. Sodium restriction is also indicated to prevent ascites. Patients with prolonged or severe anorexia and those who are vomiting or eating poorly for any reason may receive nutrients by the enteral or parenteral route (see [Chapter 39](#) for further details on enteral nutrition and [Chapter 41](#) for further details on parenteral nutrition).

Providing Skin Care

Providing careful skin care is important because of subcutaneous edema, the patient's immobility, jaundice, and increased susceptibility to skin breakdown and infection. Frequent changes in position are necessary to prevent pressure injuries. Irritating soaps and the use of adhesive tape are avoided to prevent trauma to the skin. Lotion may be soothing to irritated skin; the nurse takes measures to minimize scratching by the patient.

Reducing Risk of Injury

The nurse protects the patient with cirrhosis from falls and other injuries. The side rails should be in place and pads used in case the patient becomes agitated or restless. To minimize agitation, the nurse orients the patient to time and place and explains all procedures. The nurse instructs the patient to ask for assistance to get out of bed. The nurse carefully evaluates any injury because of the possibility of internal bleeding.

Because of the risk for bleeding from abnormal clotting, the patient should use an electric razor rather than a safety razor. A soft-bristled toothbrush helps minimize bleeding gums, and pressure applied to all venipuncture sites helps minimize bleeding.

Monitoring and Managing Potential Complications

A major role of the nurse is monitoring of the patient with cirrhosis for complications.

Bleeding and Hemorrhage

The patient is at increased risk for bleeding and hemorrhage because of decreased production of prothrombin and decreased ability of the diseased liver to synthesize the necessary substances for blood coagulation (see the Esophageal Varices section).



Hepatic Encephalopathy

Hepatic encephalopathy and coma, which are complications of cirrhosis, may manifest as deteriorating mental status (delirium) or as physical signs such as

abnormal voluntary and involuntary movements. Hepatic encephalopathy was discussed earlier in the chapter in detail.

Monitoring is essential to identify early deterioration in mental status. The nurse monitors the patient's mental status closely and reports changes so that treatment for encephalopathy can be initiated promptly. An extensive baseline and ongoing neurologic evaluation is key to identify progression through the four stages of encephalopathy (see [Table 43-3](#)).

Each advancing stage demands more intensive nursing interventions aimed at providing for patient safety and prevention and early identification of life-threatening complications such as respiratory failure and cerebral edema, which would necessitate interventions in an ICU. Because electrolyte disturbances can contribute to encephalopathy, serum electrolyte levels are carefully monitored and corrected if abnormal. Oxygen is given if oxygen desaturation occurs. The nurse monitors for fever or abdominal pain, which may signal the onset of bacterial peritonitis or other infection (see earlier discussion of hepatic encephalopathy).

Fluid Volume Excess

Patients with advanced chronic liver disease develop cardiovascular abnormalities. These occur due to an increased cardiac output and decreased peripheral vascular resistance, possibly resulting from the release of vasodilators. A hyperdynamic circulatory state develops in patients with cirrhosis, and plasma volume increases. This increase in circulating plasma volume is probably multifactorial, but some studies have implicated excess production of nitrous oxide, such as that seen in sepsis, as one causative factor (Friedman & Martin, 2018). The greater the degree of hepatic decompensation, the more severe the hyperdynamic state. Close assessment of cardiovascular and respiratory status is of key importance for the care of patients with this disorder. Pulmonary compromise is always a potential complication of ESLD because of plasma volume excess; consequently, the nurse has an important role in preventing pulmonary complications. Administering diuretic agents, implementing fluid restrictions, and enhancing patient positioning can optimize pulmonary function. Fluid retention may be noted in the development of ascites, lower extremity swelling, and dyspnea. Monitoring of I&O, daily weight changes, changes in abdominal girth, and edema formation is part of nursing assessment in the hospital or in the home setting. Patients are also monitored for nocturia and, later, for oliguria, because these states indicate increasing severity of liver dysfunction (Mansour & McPherson, 2018; Schiff et al., 2018).

Promoting Home, Community-Based and Transitional Care



Educating Patients About Self-Care

During the hospital stay, the nurse and other health care providers prepare the patient with cirrhosis for discharge, focusing on dietary education. Of greatest importance is the exclusion of alcohol from the diet. The patient may benefit from referral to Alcoholics Anonymous, psychiatric care, or counseling or support from a spiritual advisor. The patient should avoid the consumption of raw shellfish.

Sodium restriction will continue for a considerable time, if not permanently. The patient will require written education, reinforcement, and support from the staff as well as family members.

Successful treatment depends on convincing the patient of the need to adhere completely to the therapeutic plan. This includes rest, lifestyle changes, adequate dietary intake, and the elimination of alcohol. The nurse also educates the patient and family about symptoms of impending encephalopathy, possible bleeding tendencies, and susceptibility to infection. Nurses should consider implementing the teach-back method when educating patients and families to insure that they are able to describe what they have been taught in their own words or perform a task as instructed (see [Chapter 3](#) for further discussion of the teach-back method).

Recovery is neither rapid nor easy; there are frequent setbacks and apparent lack of improvement. Many patients find it difficult to refrain from using alcohol for comfort or escape. The nurse has a significant role in offering support and encouragement to the patient and in providing positive feedback when the patient experiences success.

Continuing and Transitional Care

Referral for transitional or home care may assist the patient in dealing with the transition from hospital to home. The use of alcohol may have been an important part of normal home and social life in the past. The nurse assesses the patient's progress at home and the manner in which the patient and family are coping with the elimination of alcohol and the dietary restrictions. The nurse also reinforces previous education and answers questions that may not have occurred to the patient or family until the patient is back home and trying to establish new patterns of eating, drinking, and lifestyle.

CANCER OF THE LIVER

Hepatic tumors may be malignant or benign. Benign liver tumors were uncommon until oral contraceptives were in widespread use in western countries. Now, benign liver tumors such as hepatic adenomas occur most frequently in women in their reproductive years who are taking oral contraceptives, though the incidence has decreased with the development of

modern contraceptives which contain less estrogen. The risk of these tumors is increased in those who are overweight or have obesity. These lesions may be complicated by hemorrhage and conversion to a malignant state (Tsilimigras, Rahnemai-Azar, Ntanasis-Stathopoulos, et al., 2019).

Primary Liver Tumors

Few cancers originate in the liver. Primary liver tumors usually are associated with chronic liver disease, hepatitis B and C infections, and cirrhosis. HCC is the most common type of primary liver cancer, responsible for 75% of all liver cancers, with more than half a million cases diagnosed each year on a worldwide basis. HCC is the second leading cause of cancer-related mortality worldwide. It is rare in the United States and northern Europe, accounting for fewer than 6 cases per 100,000 inhabitants (Akinyemiju, Abera, Ahmed, et al., 2017; Schiff et al., 2018). Other types of primary liver cancer include fibrolamellar carcinoma, angiosarcoma, hepatoblastoma, cholangiocellular carcinoma, and combined hepatocellular and cholangiocellular carcinoma. HCC is usually nonresectable because of rapid growth and metastasis. If found early, resection of primary liver cancer may be possible; however, early detection is not common.

Cirrhosis, chronic infection with HBV and HCV, and exposure to certain chemical toxins (e.g., vinyl chloride, arsenic) have been implicated as causes of HCC. Cigarette smoking has also been identified as a risk factor, especially when combined with alcohol (Petrick, Campbell, Koshiol, et al., 2018). Some evidence suggests that aflatoxin, a metabolite of the fungus *Aspergillus flavus*, may be a risk factor for HCC. This is especially true in areas where HCC is endemic (i.e., Asia, Africa). Aflatoxin and other similar toxic molds can contaminate food such as ground nuts and grains and may act as co-carcinogens with hepatitis B (Friedman & Martin, 2018). The risk of contamination is greatest when these foods are stored unrefrigerated in tropical or subtropical climates.

Liver Metastases

Metastases from other primary sites, particularly the digestive system, breast, and lung, are found in the liver 2.5 times more frequently than tumors due to primary liver cancers (Friedman & Martin, 2018; Goldman & Schafer, 2019). Malignant tumors are likely to reach the liver eventually, by way of the portal system or lymphatic channels, or by direct extension from an abdominal tumor. Moreover, the liver apparently is an ideal place for these malignant cells to thrive. Often, the first evidence of cancer in an abdominal organ is the

appearance of liver metastases; unless exploratory surgery or an autopsy is performed, the primary tumor may never be identified.

Clinical Manifestations

The early manifestations of malignancy of the liver include pain—a continuous dull ache in the right upper quadrant, epigastrium, or back. Weight loss, loss of strength, anorexia, and anemia may also occur. The liver may be enlarged and irregular on palpation. Jaundice is present only if the larger bile ducts are occluded by the pressure of malignant nodules in the hilum of the liver. Ascites develops if such nodules obstruct the portal veins or if tumor tissue is seeded in the peritoneal cavity.

Assessment and Diagnostic Findings

The diagnosis of liver cancer is based on clinical signs and symptoms, the history and physical examination, and the results of laboratory and x-ray studies. Increased serum levels of bilirubin, alkaline phosphatase, AST, GGT, and lactic dehydrogenase may occur. Leukocytosis (increased white blood cells), erythrocytosis (increased red blood cells), hypercalcemia, hypoglycemia, and hypocholesterolemia may also be seen on laboratory assessment.

The serum level of alpha-fetoprotein, which serves as a tumor marker, is elevated in 80% to 90% of patients with HCC, commonly to levels greater than 200 ng/mL (Friedman & Martin, 2018). Patients with small tumors (<5 cm in diameter) have normal or minimally elevated levels of alpha-fetoprotein. The level of carcinoembryonic antigen, a marker of advanced cancer of the digestive tract, may be elevated. These two markers together are useful to distinguish between metastatic liver disease and primary liver cancer.

Many patients have metastases from the primary liver tumor to other sites by the time the diagnosis is made; metastases occur primarily to the lung but may also occur to regional lymph nodes, adrenals, bone, kidneys, heart, pancreas, or stomach.

X-rays, liver scans, CT scans, ultrasound studies, MRI, arteriography, and laparoscopy may be part of the diagnostic workup and may be performed to determine the extent of the cancer. Positron emission tomography (PET) scans are used to evaluate a wide range of metastatic tumors of the liver.

Confirmation of a tumor's histology can be made by biopsy under imaging guidance (CT scan or ultrasound) or laparoscopically. Local or systemic dissemination of the tumor by needle biopsy or fine-needle biopsy can occur. Because of the small but real risks of tumor seeding (0.5% to 2%), hemorrhage, and false-negative results from biopsy, many transplant centers avoid biopsy, particularly in patients who may be candidates for liver resection.

or liver transplantation. Assessment of the imaging characteristics for the diagnosis of HCC is preferred in these instances, and a confirmed diagnosis of HCC is made by frozen section at the time of surgery (Goldman & Schafer, 2019).

Medical Management

Although surgical resection of the liver tumor is possible in some patients, the underlying cirrhosis is so prevalent in cancer of the liver that it increases the risks associated with surgery. Radiation therapy and chemotherapy have been used to treat cancer of the liver with varying degrees of success. Although these therapies may prolong survival and improve quality of life by reducing pain and discomfort, their major effect is palliative.

Radiation Therapy

The use of external-beam radiation for the treatment of liver tumors has been limited by the radiosensitivity of normal hepatocytes and the risk of destruction of normal liver parenchyma. More effective methods of delivering radiation to tumors of the liver include IV or transarterial injection of a lipiodol-ethanol (radiopaque agent) mixture (also known as transarterial chemoembolization [TACE]) that specifically attacks tumor-associated antigens, use of drug-eluting beads (DEB-TACE) and percutaneous placement of a high-intensity source for interstitial radiation therapy (delivery of radiation directly to the tumor cells). Internal radiotherapy can result in reduction in tumor size, but its effect on survival is yet to be determined (Friedman & Martin, 2018; Schiff et al., 2018).

Chemotherapy

Typically, studies of patients with advanced cases of liver cancer have shown that the use of systemic chemotherapeutic agents leads to poor outcomes. For patients with stable hepatic function (Child class A), a targeted molecular therapy, sorafenib, has been developed and approved for use and is the standard systemic treatment for patients with advanced HCC. Regorafenib may be used in patients who cease responding to sorafenib (Friedman & Martin, 2018). Systemic chemotherapy may be used to treat metastatic liver lesions. Embolization of tumor vessels with chemotherapy produces anoxic necrosis with high concentrations of trapped chemotherapeutic agents. This therapy has begun to show some promising results. An implantable pump has been used to deliver a high concentration of chemotherapy by constant infusion to the liver through the hepatic artery in cases of metastatic disease. This method has shown a moderate response rate (Friedman & Martin, 2018).

Percutaneous Biliary Drainage

Percutaneous biliary or transhepatic drainage is used to bypass biliary ducts obstructed by liver, pancreatic, or bile duct tumors in patients who have inoperable tumors or are considered poor surgical risks. Under fluoroscopy, a catheter is inserted through the abdominal wall and past the obstruction into the duodenum. Such procedures are used to reestablish biliary drainage, relieve pressure and pain from the buildup of bile behind the obstruction, and decrease pruritus and jaundice. As a result, the patient is made more comfortable, and quality of life and survival are improved.

For several days after its insertion, the catheter is opened to external drainage. The bile is observed closely for amount, color, and presence of blood and debris. Complications of percutaneous biliary drainage include sepsis, leakage of bile, hemorrhage, and reobstruction of the biliary system by debris in the catheter or by encroaching tumor. Therefore, the patient is observed for fever and chills, bile drainage around the catheter, changes in vital signs, and evidence of biliary obstruction, including increased pain or pressure, pruritus, and recurrence of jaundice.

Other Nonsurgical Treatments

Laser hyperthermia has been used to treat hepatic metastases. Heat has been directed to tumors through several methods to cause necrosis of the tumor cells while sparing normal tissue. In radiofrequency thermal ablation, a needle electrode is inserted into the liver tumor under imaging guidance. Radiofrequency energy passes through to the noninsulated needle tip, causing heat and tumor cell death from coagulation necrosis.

Immunotherapy is another treatment modality under investigation. In this therapy, lymphocytes with antitumor reactivity are given to the patient with hepatic cancer. Tumor regression has been demonstrated in patients with metastatic cancer for whom standard treatment has failed.

Transcatheter arterial embolization interrupts the arterial blood flow to small tumors by injecting small particulate embolic or chemotherapeutic agents (as described previously) into the artery supplying the tumor. As a result, ischemia and necrosis of the tumor occur.

For multiple small lesions, ultrasound-guided injection of alcohol promotes dehydration of tumor cells and tumor necrosis (Srinivasan & Friedman, 2018).

Surgical Management

Surgical resection is the treatment of choice when HCC is confined to one lobe of the liver and the function of the remaining liver is considered adequate for postoperative recovery. In the case of metastasis, hepatic resection can be performed if the primary site can be completely excised and the metastasis is limited. However, metastases to the liver are rarely limited or solitary. Capitalizing on the regenerative capacity of the liver cells, some surgeons have

successfully removed 90% of the liver. However, the presence of cirrhosis limits the ability of the liver to regenerate. Laparoscopic liver resection for malignant tumors has also been described. Staging of liver tumors aids in predicting the likelihood of surgical cure (Cameron & Cameron, 2020; Friedman & Martin, 2018; Schiff et al., 2018).

In preparation for surgery, the patient's nutritional, fluid, and general physical status are assessed, and efforts are undertaken to ensure the best physical condition possible. Extensive diagnostic studies may be performed. Specific studies may include liver scan, liver biopsy, cholangiography, selective hepatic angiography, percutaneous needle biopsy, peritoneoscopy, laparoscopy, ultrasound, CT scan, PET scan, MRI, and blood tests, particularly determinations of serum alkaline phosphatase, AST, and GGT and its isoenzymes.

Lobectomy

Removal of a lobe of the liver is the most common surgical procedure for excising a liver tumor. If it is necessary to restrict blood flow from the hepatic artery and portal vein for longer than 15 minutes, it is likely that hypothermia will be used. For a right-liver lobectomy or an extended right lobectomy (including the medial left lobe), a thoracoabdominal incision is used. An extensive abdominal incision is made for a left lobectomy.

Local Ablation

In patients who are not candidates for resection or transplantation, ablation of HCC may be accomplished by chemicals such as ethanol or by physical means such as radiofrequency ablation (most frequently used local ablative therapy) or microwave coagulation. These techniques may be performed under ultrasound or CT guidance laparoscopically or percutaneously. Radiofrequency ablation is becoming a standard mode of treatment; a tumor up to 5 cm in size can be destroyed in one session. The most common complications following ablation are local pain or bleeding. Serious complications are rare (Friedman & Martin, 2018).

Immunotherapy with interferon may be used after surgical resection for HCC to prevent recurrence of the lesion in those patients who have developed the lesion related to hepatitis B or C.

Liver Transplantation

Liver transplantation offers good patient outcomes. Candidates with liver cancer meet stringent selection criteria, including having small, early-stage lesions (Friedman & Martin, 2018; Gerber, Baliga, & Karp, 2018; Yang, Larson, Watt, et al., 2017). The Milan criteria have been developed to limit transplantation to patients who are most likely to have better outcomes. The

Milan criteria include that the patient must have a single tumor measuring less than 5 cm, or have three or fewer lesions with none over 3 cm in size (Friedman & Martin, 2018; Schiff et al., 2018). This treatment involves removing the liver and replacing it with a healthy donor organ. Studies report decreased recurrence rates of the primary liver malignancy after transplantation, with improvement in 4-year survival rates to approximately 85%, which is similar to survival rates seen in patients transplanted for nonmalignant disorders (Friedman & Martin, 2018; Schiff et al., 2018). Metastasis and recurrence may be enhanced by the immunosuppressive therapy that is needed to prevent rejection of the transplanted liver. In patients with small (less than 5 cm), single lesions, liver transplantation has been shown to be beneficial, but its use is limited by organ shortages. The increasing use of living donor transplantation may improve this situation and decrease the waiting time and tumor proliferation that is characteristic of patients with liver cancer (see later discussion).

Nursing Management

For patients with liver cancer anticipating surgery, support, education, and encouragement are provided to help them prepare psychologically for the surgery. After surgery, potential problems related to cardiopulmonary involvement may include vascular complications and respiratory and liver dysfunction. Metabolic abnormalities require careful attention. Because extensive blood loss may occur as well, the patient receives infusions of blood and IV fluids. The patient requires constant, close monitoring and care for the first 2 or 3 days, similar to postsurgical abdominal and thoracic nursing care.

If the patient is to receive chemotherapy or radiation therapy in an effort to relieve symptoms, they may be discharged home while still receiving one or both of these therapies. The patient may also go home with a biliary drainage system or hepatic artery catheter in place. In most cases, the hepatic artery catheter has been inserted surgically and has a prefilled infusion pump implanted subcutaneously that delivers a continuous chemotherapeutic dose until completed (Friedman & Martin, 2018; Schiff et al., 2018). A hepatic artery port may also be inserted to provide access for intermittent chemotherapy infusion. This port dwells under the skin, but because it provides direct arterial access, it is not used for continuous infusion therapy in the home environment; the access line is discontinued once the chemotherapeutic agent has infused. The patient and family require education about care of the biliary catheter and the effects and side effects of hepatic artery chemotherapy. This education is necessary because of participation of the patient and family in patient care in the home setting.

Promoting Home, Community-Based and Transitional Care



Educating Patients About Self-Care

The nurse educates the patient to recognize and report the potential complications and side effects of the chemotherapy and the desirable and undesirable effects of the specific chemotherapy regimen. The nurse also emphasizes the importance of follow-up visits to assess the response to chemotherapy and radiation therapy. In addition, if the patient is receiving chemotherapy on an outpatient basis, the nurse explains the patient's and family's role in managing the chemotherapy infusion and in assessing the infusion or insertion site. The nurse encourages the patient to resume routine activities as soon as possible while cautioning about falls and activities that may damage the infusion pump or site.

Patients at home with a biliary drainage system in place typically fear that the catheter will become dislodged; this fear is often shared by the patient's family. Reassurance and education can help reduce their fear that the catheter will fall out easily. The patient and family also require education on catheter care, including instruction on how to keep the catheter site clean and dry and how to assess the catheter and its insertion site. Irrigation of the catheter with sterile normal saline solution or water may be prescribed to keep the catheter patent and free of debris. The patient and caregivers are taught proper technique to avoid introducing bacteria into the biliary system or catheter during irrigation. They are instructed not to aspirate or draw back on the syringe during irrigation in order to prevent entry of irritating duodenal contents into the biliary tree or catheter. The patient and caregivers are also educated about the signs of complications and are encouraged to notify the nurse or primary provider if problems or questions arise. The nurse should consider using the teach-back method when educating patients and family about these interventions (see [Chapter 3](#) for further discussion of the teach-back method).

Patients with implantable ports are educated about the chemotherapy regimen, types of medications, effects and side effects that may occur, and appropriate management strategies if problems occur. If a hepatic artery port is inserted for intermittent chemotherapy, patients and their families are provided the same educational content. Such a port has an internal one-way valve; therefore, it is not aspirated for a blood return before the infusion is initiated. The patient is instructed to assess the port site between infusions and to note and report any sign of infection or inflammation.

Continuing and Transitional Care

In many cases, referral for transitional or home care enables the patient with liver cancer to be at home in a familiar environment with family and friends. Because of the poor prognosis associated with liver cancer, the nurse serves a

vital role in assisting the patient and family to cope with the symptoms that may occur and the prognosis. The patient's physical and psychological statuses are assessed as well as adequacy of pain relief, nutritional status, and presence of symptoms indicating complications of treatment or progression of disease. During home visits, the nurse assesses the function of the chemotherapy pump, the infusion site, and the biliary drainage system, if indicated. The nurse collaborates with the other members of the health care team, the patient, and the family to ensure effective pain management and to manage potential problems, which include weakness, pruritus, inadequate dietary intake, jaundice, and symptoms associated with metastasis to other sites. The nurse also assists the patient and family in making decisions about hospice care and assists with initiation of referrals. The patient is encouraged to discuss preferences for end-of-life care with family members and health care providers (see [Chapter 13](#)).

LIVER TRANSPLANTATION

Liver transplantation is used to treat life-threatening ESLD for which no other form of treatment is available. The transplantation procedure involves total removal of the diseased liver and replacement with a healthy liver from a deceased donor or with the right lobe from a live donor in the same anatomic location, referred to as **orthotopic liver transplantation [OLT]**. Removal of the liver creates a space for the new liver and permits anatomic reconstruction of the hepatic vasculature and biliary tract as close to normal as possible.

The success of liver transplantation depends on successful immunosuppression. Immunosuppressant agents currently used include the calcineurin inhibitors cyclosporine and tacrolimus. Corticosteroids are commonly used for induction immunosuppression. Mycophenolate mofetil blocks lymphocyte proliferation and results in lower dose requirements of calcineurin inhibitors. Target of rapamycin inhibitors sirolimus (formerly known as rapamycin) and everolimus inhibit T- and B-cell proliferation. Antibody therapies such as antithymocyte globulin, basiliximab, and daclizumab deplete lymphocytes and inhibit T-cell proliferation (Friedman & Martin, 2018). There is no one, accepted, optimal immunosuppressive regimen. Most transplant centers have developed their own therapeutic practices, largely based on experience. Multiple immunosuppressive strategies can be used to prevent transplanted organ rejection. Most strategies involve the use of more than one agent, but current recommendations advise the minimization of immunosuppression in order to avoid toxicity (Friedman & Martin, 2018). Using multiple immunosuppressive agents has the effect of blocking multiple targets in the immune response cascade. This allows the use of lower doses of each drug, thus avoiding toxicity associated with high doses of these powerful drugs. Some patients are treated with "triple therapy" using corticosteroids, a calcineurin inhibitor, and either an antiproliferative agent

(mycophenolate mofetil) or a target of rapamycin inhibitor (Friedman & Martin, 2018). Some transplant centers also prescribe steroid-free immunosuppressant regimens after liver transplantation. This regimen has been found to be a safe alternative. Still other transplant centers advocate monotherapy with a calcineurin inhibitor alone to provide long-term immunosuppression (Cameron & Cameron, 2020; Friedman & Martin, 2018). In order to prevent acute rejection in the early, high-risk months after liver transplantation, some centers employ induction therapy, which is the use of prophylactic, perioperative course of immunosuppressant drugs that suppress the body's response to the foreign, transplanted tissue and may improve graft survival by reducing the risk of acute cellular rejection (Friedman & Martin, 2018). Over time, withdrawal of all immunosuppressants is possible but rare; randomized, prospective studies are needed before this clinical practice is accepted.

Despite the success of immunosuppression in reducing the incidence of rejection of transplanted organs, liver transplantation is not routine and may be accompanied by complications related to the lengthy surgical procedure, immunosuppressive therapy, infection, and technical difficulties encountered in reconstructing the blood vessels and biliary tract. Long-standing systemic problems resulting from the primary liver disease may complicate the pre- and postoperative course. Previous surgery of the abdomen, including procedures to treat complications of advanced liver disease (i.e., shunt procedures used to treat portal hypertension, esophageal varices) increase the complexity of the transplantation procedure.

General indications for liver transplantation include irreversible advanced chronic liver disease, ALF, metabolic liver diseases, and some hepatic malignancies. Examples of disorders that are indications for liver transplantation include hepatocellular liver diseases (e.g., viral hepatitis, drug- or alcohol-induced liver disease, Wilson disease) and cholestatic diseases (primary biliary cirrhosis, sclerosing cholangitis, NASH, and biliary atresia).

The patient being considered for liver transplantation frequently has many systemic problems that influence pre- and postoperative care. Because transplantation is more difficult if the patient has developed severe GI bleeding and hepatic coma, efforts are made to perform the procedure before the disease progresses to this stage. The patient must undergo a thorough evaluation of hepatic reserve and general health. Part of this evaluation includes classification of the degree of medical need, an objective determination known as the MELD classification, which stratifies the level of illness of those awaiting a liver transplant. The MELD score is derived from a complex formula incorporating bilirubin levels, PT/INR, creatinine, and the cause of the liver disease (i.e., cholestatic, alcoholic, or other). The MELD score is an indicator of short-term mortality for those with ESLD. Organs are allocated

using the MELD score in an effort to provide transplants to the most severely ill patients (Friedman & Martin, 2018; Schiff et al., 2018).

Liver transplantation is an established therapeutic modality, and the number of liver transplant centers is increasing. Patients requiring transplantation are often referred from distant hospitals to these centers. To prepare the patient and family for liver transplantation, nurses in all settings must understand the processes and procedures of liver transplantation.

Many ethical issues arise concerning liver transplantation, particularly concerning the allocation of organs. The way in which some persons contracted liver disease (e.g., alcohol use, hepatitis) leads others to question allocation of organs to them, and some believe that preference should be given to people who need liver transplants but do not have a history of socially unacceptable behavior. More issues arise when a patient requires a second transplant operation because of a return to alcohol or drug use or failure to follow immunosuppressive regimens. Transplant recipients must go through a rigorous selection and preparation process that includes counseling and education to aid them in making critical choices for their improved health. Nurses and other health care providers need to be aware of and confront their own biases and work toward improved understanding and acceptance.

Surgical Procedure

During the procedure, the donor liver is freed from other structures, the bile is flushed from the gallbladder to prevent damage to the walls of the biliary tract, and the liver is perfused with a preservative and cooled. Before the donor liver is placed in the recipient, it is flushed with cold lactated Ringer solution to remove potassium and air bubbles. The presence of portal hypertension increases the difficulty of the procedure.

Anastomoses (connections) of the blood vessels and bile duct are performed between the donor liver and the recipient liver. There are two types of biliary anastomoses. Biliary reconstruction is performed with an end-to-end anastomosis of the donor and recipient common bile ducts; a stented T-tube may be inserted for external drainage of bile. In patients with biliary disease such as primary sclerosing cholangitis or if the recipient's bile duct is not suitable for anastomosis for other reasons, a biliary-enteric end-to-side anastomosis with a 40- to 50-cm Roux-en-Y loop of jejunum is created for biliary drainage (known as a Roux-en-Y procedure) (see Fig. 43-14A); in this case, bile drainage is internal, and a T-tube is not inserted (Cameron & Cameron, 2020). Figure 43-14B,C illustrates the final appearance of the grafted liver and final closure and one method of drain placement. Some transplant centers may use fewer drains.

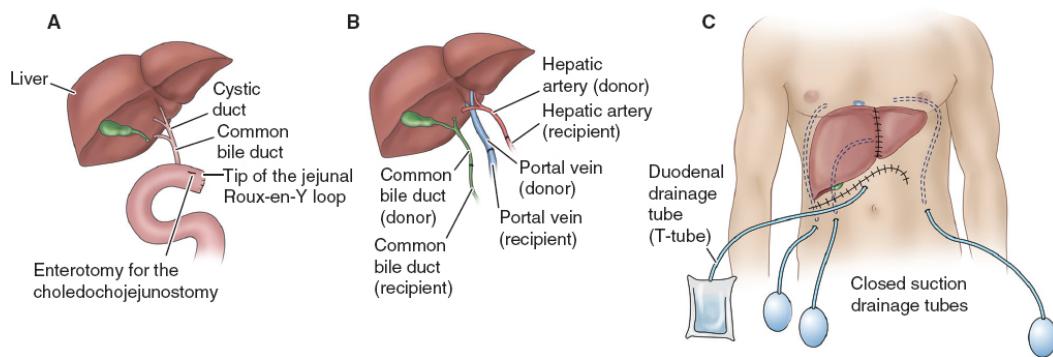


Figure 43-14 • A. Some transplant recipients have diseases or conditions that cause their bile ducts to be unusable for anastomosis to the donor liver bile duct. In this case, a loop of jejunum is used as a bridge from the donor liver bile duct to the recipient's small bowel for biliary continuity and drainage. This procedure is termed *Roux-en-Y hepaticojjunostomy*. **B.** Final appearance of implanted liver graft with an end-to-end biliary anastomosis. **C.** Final closure and drain placement after liver transplantation with an end-to-end biliary anastomosis and T-tube placement.

Several additional techniques have been developed to expand the donor pool for liver transplantation. In a split-liver transplant, a single organ is used to provide grafts for two individuals with ESLD, with the smaller patient receiving the smaller left lobe. This procedure has resulted in a higher complication rate and lower survival rate than traditional liver transplantation. Living donor transplantation is being increasingly performed from adult to adult using full right lobes, although it is controversial because it is a major surgical procedure for the donor, and some donor deaths have occurred.

Living donor liver transplantation (LDLT) is considered for patients who have a high potential for mortality while awaiting a deceased liver donor, such as those patients with HCC or those with severe complications of cirrhosis including GI bleeding or hepatic encephalopathy (Cameron & Cameron, 2020). The results thus far have indicated that this procedure is most successful when donor and recipient are appropriately selected using careful screening criteria (Cameron & Cameron, 2020). The option of LDLT decreases the waiting list mortality and produces positive recipient outcomes with a low risk of morbidity and mortality for the donor. The LDLT procedure involves transplantation of the right hepatic lobe from an adult donor to the recipient. Potential donors are evaluated by a donor advocate team. Donors must be completely healthy and have hepatic size and anatomy compatible with right lobe transplantation (Cameron & Cameron, 2020). There is an extensive informed consent process for live liver donors. The donor advocate ensures that the concern for donor safety is paramount, especially in the intra- and

postoperative period when complications can occur. The clear separation of donor and recipient teams ensures that the donor is treated without ulterior motives, which might occur if the same team cared for both the donor and recipient.

In the LDLT procedure, the surgeon performs a formal right hepatic lobectomy. The right lobe is then flushed with preservative solution and vascular reconstruction is completed to prepare for implantation. The recipient operation involves an inferior vena cava–sparing hepatectomy with anastomosis of the donor right-sided vascular and biliary structures to the corresponding recipient structures (Cameron & Cameron, 2020).

Liver transplantation is a long surgical procedure, partly because the patient with liver failure often has portal hypertension, requiring ligation of many venous collateral vessels. Blood loss during the surgical procedure may be extensive. If the patient has adhesions from previous abdominal surgery, lysis of adhesions is often necessary. If a shunt procedure was performed previously, it must be surgically reversed to permit adequate portal venous blood supply to the new liver. During the lengthy surgery, it is important to provide regular updates to the family about the progress of the operation and the patient's status.

Complications

The postoperative complication rate is high, primarily because of technical complications or infection. Immediate postoperative complications may include bleeding, infection, and rejection. Disruption, infection, obstruction of the biliary anastomosis, and impaired biliary drainage may occur. Vascular thrombosis and stenosis are other potential complications. These complications occur in patients receiving either a deceased donor or live donor organ. Despite the development of some complications, the 1-year patient survival rate approaches 90%, and the 5-year patient survival rate is approximately 80% (Cameron & Cameron, 2020; Friedman & Martin, 2018).

Bleeding

Bleeding is common in the postoperative period and may result from coagulopathy, portal hypertension, and fibrinolysis caused by ischemic injury to the donor liver. Hemodynamic instability and transient hypotension may occur in this phase, secondary to blood loss, loss of vasomotor tone, and vasodilatation due to rewarming the hypothermic patient or due to preexisting cardiac conditions such as cardiomyopathy (Goldman & Schafer, 2019). Administration of platelets, fresh-frozen plasma, or other blood products may be necessary. Hypertension may co-occur postoperatively but is more common later in the postoperative phase, although its cause is uncertain. Currently available hypertension guidelines do not have specific recommendations for

management in the liver transplant population. However, calcium channel blockers such as nifedipine or amlodipine are frequently used for their vasodilatory effects and are the agents of choice. These drugs also are preferable due to their low interaction level with the cytochrome P450 enzyme system with resultant minimal risk of disruption of immunosuppressant levels. Angiotensin-converting enzyme (ACE) inhibitors and ARB medications are not first-line drugs for the treatment of hypertension during the first year after transplant due to low levels of renin during this time period. Thiazide diuretics are reserved for patients requiring more than one medication for blood pressure control (Friedman & Martin, 2018). Blood pressure elevation that is significant or sustained is also managed with lifestyle modifications, a low-sodium diet, and an exercise regimen.



Infection

Infection is the leading cause of death after liver transplantation. Pulmonary and fungal infections are common; susceptibility to infection is increased by the immunosuppressive therapy that is needed to prevent rejection (Friedman & Martin, 2018). Therefore, precautions must be taken to prevent health care-associated infections. The nurse uses strict asepsis when manipulating central venous catheters, arterial lines, and urine, bile, and other drainage systems; obtaining specimens; and changing dressings. Meticulous hand hygiene is crucial. In the ICU, the nurse vigilantly monitors for early clinical manifestations of sepsis (see [Chapter 11, Chart 11-6](#)) and uses evidence-based practice guidelines (or bundles) developed by the Institute for Healthcare Improvement (IHI) in the care of the postoperative liver transplant patient. Some of these care guidelines include prevention of sepsis through prevention of central line-associated bloodstream infections and its rapid treatment (see [Chapter 11, Chart 11-2](#)) and prevention of ventilator-associated pneumonia (VAP) (see [Chapter 19, Chart 19-6](#)).

Rejection

Rejection is a major concern. A transplanted liver is perceived by the immune system as a foreign antigen. This triggers an immune response, leading to the activation of T lymphocytes that attack and destroy the transplanted liver. Immunosuppressive agents are used as long-term therapy to prevent this response and rejection of the transplanted liver. These agents inhibit the activation of immunocompetent T lymphocytes to prevent the production of effector T-cells.

Although the 1- and 5-year survival rates have increased dramatically with the use of new immunosuppressive therapies, these advances are not without major side effects. A major side effect of cyclosporine, which has been widely used in transplantation, is nephrotoxicity; this problem seems to be dose

related. Cyclosporine-related side effects have caused many transplant centers to use tacrolimus instead as first-line therapy because of its efficacy and lower side-effect profile.

Corticosteroids, azathioprine, mycophenolate mofetil, sirolimus, antithymocyte globulin, basiliximab, and daclizumab are also used in various regimens of immunosuppression. These agents may be used as the initial therapy to prevent rejection or used later to treat rejection. Liver biopsy and ultrasound may be required to evaluate suspected episodes of rejection.

Retransplantation is usually attempted if the transplanted liver fails, but the success rate of retransplantation does not approach that of initial transplantation. The greater rates of organ dysfunction and loss after a second or third liver transplant are related to technical intraoperative difficulties and higher bleeding risk (Cameron & Cameron, 2020; Friedman & Martin, 2018).

Complications of the LDLT Donor

Improved surgical techniques have made the LDLT procedure an increasingly safe one; however, complications do occur for donors as well. The most frequently occurring complications include pulmonary emboli, portal vein thrombosis, bile duct injury, and liver insufficiency secondary to a resection that is too extensive (Cameron & Cameron, 2020; Friedman & Martin, 2018).

Nursing Management

The patient considering transplantation, together with the family, must make difficult choices about treatment, the use of financial resources, and relocation to another area to be closer to the medical center. They must also be aware of the risks and benefits of the procedure and its consequences. In addition, they must also cope with the patient's long-standing health problems and any social and family problems associated with behaviors that may have caused the patient's liver failure. As a result, considerable emotional stress occurs while the patient and family consider liver transplantation and wait for an available liver. The nurse must be aware of these issues and attuned to the emotional and psychological status of the patient and family. Referral to a psychiatric liaison nurse, psychologist, psychiatrist, or spiritual advisor may help them cope with the stressors associated with ESLD and liver transplantation.

If the patient and family are considering undergoing an LDLT, they are subject to additional stressors. Both the patient and the potential donor must undergo a thorough and exhaustive physical and psychological workup to ensure that all involved parties are physically and emotionally prepared. Often, but not always, the donor is a close family member. Coercion must be excluded as influencing the decision to donate a portion of one's liver to another. The potential donor must be aware of the risks associated with the procedure.

If the patient and family believe that liver transplantation may be appropriate, the nurse, surgeon, hepatologist, and other health care team members provide the patient and family with full explanations about the procedure, the chances of success, and the risks (for the donor—bleeding and venous thromboembolism), including the side effects of long-term immunosuppression and postoperative complications in the recipient as well as bleeding and biliary abnormalities (Cameron & Cameron, 2020; Friedman & Martin, 2018). The need for close follow-up and lifelong adherence to the therapeutic regimen, including immunosuppression, is emphasized to the patient and family.

Preoperative Nursing Interventions

Once the patient has been accepted as a candidate, they are placed on a waiting list at the transplant center, and patient information is entered into the United Network for Organ Sharing (UNOS) computer system. The UNOS system uses the MELD score to determine organ allocation priorities so that the patient with the highest MELD score will receive the first available organ. Candidates may be matched with appropriate organs as they become available. MELD scores provide the necessary information regarding medical need.

Except in the case of LDLT, a liver becomes available for transplantation only with the death of another person, usually someone who had been healthy except for severe brain injury and brain death. Therefore, the patient and family undergo a stressful waiting period, and the nurse is often their major source of support. The patient must be accessible at all times in case an appropriate liver becomes available. During this time, liver function may deteriorate further, and the patient may experience complications from the progressing disease. Because of the shortage of donor organs, many patients die awaiting transplantation.

Malnutrition, massive ascites, and fluid and electrolyte disturbances are treated before surgery to increase the likelihood of a successful outcome. If the patient's liver dysfunction has a very rapid onset, as in ALF, there is little time or opportunity for the patient to consider and weigh options and their consequences; the patient may be in a coma and the decision to proceed with transplantation made by the family.

The nurse coordinator is an integral member of the transplant team and plays an important role in preparing the patient for liver transplantation. The nurse serves as an advocate for the patient and family and assumes the important role of liaison between the patient and the other members of the transplant team. The nurse also serves as a resource to other nurses and health care team members involved in evaluating and caring for the patient.



Postoperative Nursing Interventions

The organ recipient is maintained in an environment as free from bacteria, viruses, and fungi as possible, because immunosuppressive medications reduce the body's natural defenses. In the immediate postoperative period, cardiovascular, pulmonary, renal, neurologic, and metabolic functions are monitored continuously. Mean arterial and pulmonary artery pressures are also monitored continuously. Cardiac output, central venous pressure, pulmonary artery wedge pressure, arterial and mixed venous blood gases, oxygen saturation, oxygen demand and delivery, urine output, heart rate, and blood pressure are used to evaluate the patient's hemodynamic status and intravascular fluid volume. Liver function tests, electrolyte levels, the coagulation profile, chest x-ray, electrocardiogram, and fluid output (including urine, bile from the T-tube, and drainage from Jackson–Pratt tubes) are monitored closely. Because the liver is responsible for the storage of glycogen and the synthesis of protein and clotting factors, these substances need to be monitored and replaced in the immediate postoperative period.

There is a high risk of atelectasis and an altered ventilation–perfusion ratio caused by insult to the diaphragm during the surgical procedure, prolonged anesthesia, immobility, and postoperative pain. The patient may have an endotracheal tube in place and require mechanical ventilation during the initial postoperative period. Suctioning is performed as required, and sterile humidification is provided. Evidence-based practice guidelines are implemented to prevent the development of pneumonia in the postoperative liver transplant recipient (see [Chapter 19, Chart 19-6](#)).

As the patient's condition stabilizes, efforts are made to promote recovery from the trauma of this complex surgery. After removal of the endotracheal tube, the nurse encourages the patient to use an incentive spirometer to decrease the risk of atelectasis (see [Chapter 19, Chart 19-1](#) for further details on incentive spirometry). Following extubation, the patient is assisted to get out of bed, to ambulate as tolerated, and to participate in self-care to prevent the complications associated with immobility (Pearson, Mangold, Kosiorek, et al., 2018). Close monitoring for signs and symptoms of liver dysfunction and rejection continue throughout the hospital stay. Plans are made for close follow-up after discharge as well. Education is initiated during the preoperative period and continues after surgery.

The live donor is frequently admitted to an ICU setting along with the recipient. The donor also requires close monitoring for cardiovascular, hemodynamic, and pulmonary stability. The nurse closely assesses the donor for signs of hemorrhage, biliary complication, respiratory decompensation, and infection. The donor is mobilized early in the postoperative phase to prevent the development of complications such as pulmonary embolism. Studies suggest that the donor may experience more pain than the recipient, possibly requiring more analgesia for pain control (Friedman & Martin, 2018). Patient

education focuses on prevention and recognition of complications as well as activity progression and pain management.

Promoting Home, Community-Based and Transitional Care



Educating Patients About Self-Care

Educating the patient, family, and caregivers about long-term measures to promote health is crucial for the success of transplantation and is an important role of the nurse. The patient and family must understand why they need to adhere closely to the therapeutic regimen, with special emphasis on the methods of administration, rationale, and side effects of the prescribed immunosuppressive agents. The nurse provides written and verbal education about how and when to take the medications. To avoid running out of medication or skipping a dose, the patient must make sure that an adequate supply of medication is available. Education is provided about the signs and symptoms that indicate problems necessitating consultation with the transplant team. The patient with a T-tube in place must be educated about how to manage the tube, drainage, and skin care.

Chart 43-11



NURSING RESEARCH PROFILE

Psychosocial Adjustments in Patients Undergoing Liver Transplantation

Yıldız, E., & Kılıç, G. (2018). The relationship between anxietydepression status and psychosocial adjustments in the patients undergoing liver transplantation. *Perspectives in Psychiatric Care*, 54(2), 221–229.

Purpose

The purpose of this study was to determine the relationship between the anxiety-depression status and psychosocial adjustments in patients undergoing liver transplantation.

Design

This was a descriptive correlational study conducted with 90 participants who received a liver transplant in one transplant facility in Turkey. The participants included in the study were administered two questionnaires, the Hospital Anxiety and Depression Scale (HADS) and the Psychosocial Adjustment to Illness Self Report (PAIS).

Findings

The study found that as the anxiety risk of participants increased, their adjustments to their domestic and psychological environments were negatively affected.

In younger participants, the risk of experiencing anxiety increased and psychological adjustment to undergoing liver transplantation decreased. Women had a greater risk of anxiety. Most participants showed low psychosocial adjustment after liver transplantation, but this was most pronounced in those who were single, had a moderate socioeconomic level, and who were employed.

Nursing Implications

A psychiatric assessment is one element of the intensive preoperative evaluation of patients prior to undergoing liver transplantation. Psychiatric nurse practitioners may take part in such evaluations, and this information is important to have not only in the preoperative realm but after surgery should patients experience anxiety. It is important that patients are able to adjust socially and psychologically to their life changes after liver transplantation. Nurses are key participants in identifying methods to alleviate anxiety and improve coping strategies. Nurses provide encouragement for patients to participate in self-care activities that can promote effective psychosocial adjustment. Some of the patient education and support that is carried out preoperatively may facilitate this adjustment postoperatively.

Coordinating an effective plan with the interprofessional team, including psychiatric professionals, is part of the nursing role in caring for recipients of liver transplants and in helping these patients to achieve seamless transitions, less anxiety, and optimum levels of adjustment.

Following liver transplantation, the patient experiences a period of psychosocial adjustment (Ko, Muehrer, & Bratzke, 2018; Yıldız, & Kılınç, 2018). The nurse educates patients and caregivers to be alert for signs and symptoms of anxiety and depression and to report them to the transplant team so that appropriate referrals can be made for treatment as needed. See the Nursing Research Profile in [Chart 43-11](#).

Continuing and Transitional Care

The nurse emphasizes the importance of follow-up laboratory tests and appointments with the transplant team. Trough blood levels of immunosuppressive agents are obtained, along with other blood tests that assess the function of the liver and kidneys. During the first months, the patient is likely to require blood tests two or three times a week. As the patient's condition stabilizes, laboratory studies and visits to the transplant team are less frequent. The importance of routine ophthalmologic examinations is emphasized because of the increased incidence of cataracts and glaucoma associated with the long-term corticosteroid therapy used with transplantation. Regular oral hygiene and follow-up dental care, with administration of prophylactic antibiotics before dental examinations and treatments, are recommended because of the immunosuppression.

The nurse reminds the patient that preventing rejection and infection is essential and increases the chances for survival and a more normal life than before transplantation. Many patients live successful and productive lives after receiving a liver transplant. Pregnancy may be considered after transplantation but it is not without risks. Despite advances in immunosuppressive therapy, increasing experience in the management of pregnancy after liver transplantation and some successful outcomes, these pregnancies are considered high risk for both mother and infant and referral should be made to a high-risk pregnancy center well before conception (Baskiran, Karakas, Ince, et al., 2017). Transplant recipients should be advised about birth control. The waiting period allows time to establish good health, stable liver function, and lower maintenance levels of immunosuppressive therapy (Baskiran et al., 2017).

LIVER ABSCESES

Two categories of liver abscess have been identified: amebic and pyogenic. Amebic liver abscesses are most commonly caused by *Entamoeba histolytica*. Most amebic liver abscesses occur in the developing countries of the tropics and subtropics because of poor sanitation and hygiene. Pyogenic liver abscesses are much less common, but they are more common in developed countries than the amebic type (Friedman & Martin, 2018).

Pathophysiology

Whenever an infection develops anywhere along the biliary or GI tract, infecting organisms may reach the liver through the biliary system, portal venous system, or hepatic arterial or lymphatic system. Most bacteria are destroyed promptly, but occasionally some gain a foothold. The bacterial toxins destroy the neighboring liver cells, and the resulting necrotic tissue serves as a protective wall for the organisms.

Meanwhile, leukocytes migrate into the infected area. The result is an abscess cavity full of a liquid containing living and dead leukocytes, liquefied liver cells, and bacteria. Pyogenic abscesses of this type may be either single or multiple and small. Examples of causes of pyogenic liver abscess include cholangitis (usually related to benign or malignant obstruction of the biliary tree) and abdominal trauma.

Clinical Manifestations

The clinical picture is one of sepsis with few or no localizing signs. Fever with chills and diaphoresis, malaise, anorexia, nausea, vomiting, and weight loss may occur. The patient may complain of dull abdominal pain and tenderness in the right upper quadrant of the abdomen. Hepatomegaly, jaundice, anemia, and pleural effusion may develop. Sepsis and shock may be severe and life-threatening.

Appropriate diagnosis may be delayed and requires a high degree of clinical suspicion, especially in light of the lack of localizing signs in some patients. The diagnosis is made with the wide availability of various radiologic modalities including ultrasound, CT, and MRI (Goldman & Schaefer, 2019).

Assessment and Diagnostic Findings

Although blood cultures are obtained, the organism may not be identified with this test. Aspiration of the liver abscess, guided by ultrasound, CT, or MRI, may be performed to assist in diagnosis, obtain cultures and identify the organism. Percutaneous drainage of pyogenic abscesses is carried out to evacuate the abscess material and promote healing. A catheter may be left in place for continuous drainage; the patient must be educated about its management. Percutaneous drainage alone without catheter placement has received recent attention with patients followed with close clinical monitoring and serial ultrasounds. Results of this method are promising but controlled clinical trials are necessary to clarify effects and outcomes (Goldman & Schaefer, 2019).

Medical Management

Treatment includes IV antibiotic therapy; the specific antibiotic used in treatment depends on the organism identified. Continuous supportive care is indicated because of the serious condition of the patient. Open surgical drainage may be required if antibiotic therapy and percutaneous drainage are ineffective (Friedman & Martin, 2018; Schiff et al., 2018).

Nursing Management

Although the manifestations of liver abscess vary with the type of abscess, most patients appear acutely ill. Others appear to be chronically ill and debilitated. The nursing management depends on the patient's physical status and the medical management that is indicated. For patients who undergo evacuation and drainage of an abscess, monitoring the drainage and providing skin care are imperative. Strategies must be implemented to contain the drainage and to protect the patient from other sources of infection. Vital signs are monitored to detect changes in the patient's physical status. Deterioration in vital signs or the onset of new symptoms such as increasing pain, which may indicate rupture or extension of the abscess, is reported promptly. The nurse administers IV antibiotic therapy as prescribed. The white blood cell count and other laboratory test results are monitored closely for changes consistent with worsening infection. The nurse prepares the patient for discharge by providing education about symptom management, signs and symptoms that should be reported to the primary provider, management of drainage, and the importance of taking antibiotics as prescribed.

CRITICAL THINKING EXERCISES

1  You are the nurse working with a 46-year-old woman who is at high risk for hepatitis B and C due to a history of IV drug abuse. What laboratory testing is likely to be done first for this patient? If laboratory testing is positive for either or both diseases, what invasive and noninvasive testing is warranted next? Describe the education you should provide about these tests. What are the priorities for the treatment plan if testing is positive for the diseases?

2  A 58-year-old man underwent an orthotopic liver transplant for decompensated alcoholic cirrhosis. He is placed on an immunosuppressant regimen including corticosteroids, mycophenolate mofetil, and tacrolimus. On day 9 postoperatively, he develops a fever with an acute change in mental status and confusion. What nursing and interprofessional assessments are indicated for this patient? What interprofessional services should be engaged?

3  A 44-year-old woman with obesity is diagnosed with NAFLD and is symptomatic with jaundice and fatigue. What are the best risk reduction tactics for this patient? How should you prepare and educate the patient to achieve specific evidence-based management goals and potentially control the disease? Should this patient be evaluated for malnutrition and sarcopenia even though she has obesity? If so, why would an evaluation for sarcopenia be important?

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*Asterisk indicates nursing research.

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Resources

- Al-Anon Family Groups Headquarters, www.al-anon.alateen.org
Alcoholics Anonymous World Services (AAWS), www.aa.org
American Association for the Study of Liver Diseases (AASLD), www.aasld.org
American College of Gastroenterology (ACG), gi.org/
American Liver Foundation (ALF), www.liverfoundation.org
Hepatitis Foundation International (HFI), hepatitisfoundation.org
National Council on Alcoholism and Drug Dependence (NCADD),
www.ncadd.org
National Digestive Diseases Information Clearinghouse (NDDIC),
www.digestive.niddk.nih.gov
National Institute on Alcohol Abuse and Alcoholism (NIAAA),
www.niaaa.nih.gov
United Network for Organ Sharing (UNOS), www.unos.org

44 Management of Patients with Biliary Disorders

LEARNING OUTCOMES

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

- 1.** Identify the structure and function of the biliary tract and pancreas.
- 2.** Describe the pathophysiology, clinical manifestations, and medical management of cholelithiasis.
- 3.** Differentiate between acute and chronic pancreatitis.
- 4.** Apply the nursing process as a framework for care of the patient with cholelithiasis, undergoing laparoscopic or open cholecystectomy, or with acute pancreatitis.
- 5.** Explain the nutritional and metabolic effects of surgical treatment of tumors of the pancreas.

NURSING CONCEPTS

- Comfort
- Family
- Infection
- Inflammation
- Metabolism
- Nutrition

GLOSSARY

- amylase:** pancreatic enzyme; aids in the digestion of carbohydrates
- cholecystectomy:** removal of the gallbladder
- cholecystitis:** inflammation of the gallbladder which can be acute or chronic
- cholecystojejunostomy:** anastomosis of the jejunum to the gallbladder to divert bile flow
- cholecystokinin (CCK):** hormone; major stimulus for digestive enzyme secretion; stimulates contraction of the gallbladder
- cholecystostomy:** surgical opening and drainage of the gallbladder
- choledocholithiasis:** stones in the common bile duct
- choledochostomy:** opening into the common bile duct
- cholelithiasis:** calculi in the gallbladder
- dissolution therapy:** the use of medications to break up/dissolve gallstones
- endocrine:** secreting internally; hormonal secretion of a ductless gland
- endoscopic retrograde cholangiopancreatography (ERCP):** procedure using fiberoptic technology to visualize the biliary system
- endoscopic ultrasound (EUS):** invasive procedure using an ultrasound probe at the end of an endoscope to detect cholelithiasis and to decompress the gallbladder in the setting of acute cholecystitis
- exocrine:** secreting externally; hormonal secretion from excretory ducts
- lipase:** pancreatic enzyme; aids in the digestion of fats
- lithotripsy:** disintegration of gallstones by shock waves
- pancreatitis:** inflammation of the pancreas; may be acute or chronic
- secretin:** hormone responsible for stimulating bicarbonate secretion from the pancreas; also used as an aid in diagnosing pancreatic exocrine disease
- steatorrhea:** frothy, foul-smelling stools with a high fat content; results from impaired digestion of proteins and fats due to a lack of pancreatic juice in the intestine
- trypsin:** pancreatic enzyme; aids in the digestion of proteins
- Zollinger-Ellison syndrome:** hypersecretion of gastric acid that produces peptic ulcers as a result of a non-beta-cell tumor of the pancreatic islets

Disorders of the biliary tract and pancreas are common and include gallbladder stones and pancreatic dysfunction. An understanding of the structure and function of the biliary tract and pancreas is essential, along with an

understanding of how biliary tract disorders are closely linked with liver disease. Patients with acute or chronic biliary tract or pancreatic disease require care from nurses who are knowledgeable about the diagnostic procedures and interventions that are used in the management of gallbladder and pancreatic disorders.

ANATOMIC AND PHYSIOLOGIC OVERVIEW

The Gallbladder

The gallbladder, a pear-shaped, hollow, saclike organ that is 7.5 to 10 cm (3 to 4 inches) long, lies in a shallow depression on the inferior surface of the liver, to which it is attached by loose connective tissue. The capacity of the gallbladder is 30 to 50 mL of bile. Its wall is composed largely of smooth muscle. The gallbladder is connected to the common bile duct (CBD) by the cystic duct (see Fig. 44-1).

The gallbladder functions as a storage depot for bile. Between meals, when the sphincter of Oddi is closed, bile produced by the hepatocytes enters the gallbladder. During storage, a large portion of the water in bile is absorbed through the walls of the gallbladder; thus, bile in the gallbladder is five to 10 times more concentrated than that originally secreted by the liver. When food enters the duodenum, the gallbladder contracts and the sphincter of Oddi (located at the junction of the CBD with the duodenum) relaxes. Relaxation of this sphincter allows the bile to enter the intestine. This response is mediated by secretion of the hormone **cholecystokinin (CCK)** from the intestinal wall (Norris, 2019). CCK is the major stimulus for digestive enzyme secretion and acts by stimulating the gallbladder to contract.

Bile is composed of water and electrolytes (sodium, potassium, calcium, chloride, and bicarbonate) along with significant amounts of lecithin, fatty acids, cholesterol, bilirubin, and bile salts. The bile salts, together with cholesterol, assist in emulsification of fats in the distal ileum. They are then reabsorbed into the portal blood for return to the liver, after which they are once again excreted into the bile. This pathway from hepatocytes to bile to intestine and back to the hepatocytes is called the *enterohepatic circulation*. Because of this circulation, only a small fraction of the bile salts that enter the intestine are excreted in the feces. This decreases the need for active synthesis of bile salts by the liver cells.

Approximately half of the bilirubin (a pigment derived from the breakdown of red blood cells) is a component of bile. It is converted by the intestinal flora into urobilinogen, which is a highly soluble substance. Urobilinogen is either excreted in the feces or returned to the portal circulation, where it is re-excreted into the bile. About 5% is normally absorbed into the general circulation and then excreted by the kidneys (Goldman & Schafer, 2019; Norris, 2019).

If the flow of bile is impeded (e.g., by gallstones in the bile ducts), bilirubin does not enter the intestine. As a result, blood levels of bilirubin increase. This causes increased renal excretion of urobilinogen, which results from conversion of bilirubin in the small intestine, and decreased excretion in the stool. These changes produce many of the signs and symptoms seen in gallbladder disorders.

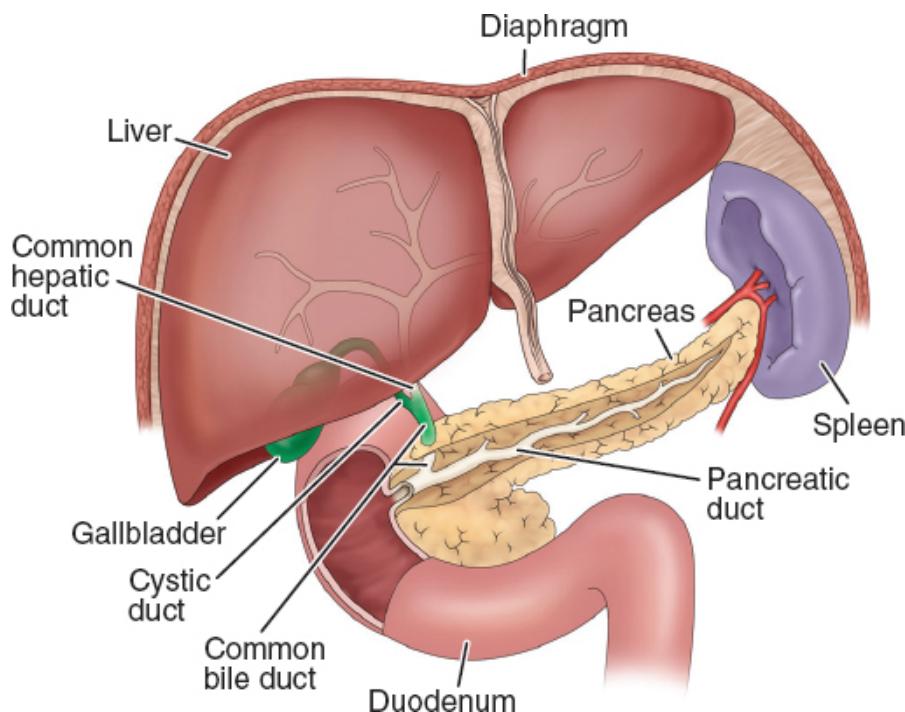


Figure 44-1 • The liver, biliary system, and pancreas.

The Pancreas

The pancreas is located in the upper abdomen (see Fig. 44-1). It has both **exocrine** (secreting externally; hormonal secretion from excretory ducts) and **endocrine** (secreting internally; hormonal secretion of a ductless gland) functions. The exocrine functions include secretion of pancreatic enzymes into the gastrointestinal (GI) tract through the pancreatic duct. The endocrine functions include secretion of insulin, glucagon, and somatostatin directly into the bloodstream.

The Exocrine Pancreas

The secretions of the exocrine portion of the pancreas are collected in the pancreatic duct, which joins the CBD and enters the duodenum at the ampulla of Vater. Surrounding the ampulla is the sphincter of Oddi, which partially

controls the rate at which secretions from the pancreas and the gallbladder enter the duodenum.

The secretions of the exocrine pancreas are digestive enzymes high in protein content and an electrolyte-rich fluid. The secretions, which are very alkaline because of their high concentration of sodium bicarbonate, are capable of neutralizing the highly acidic gastric juice that enters the duodenum. Pancreatic enzymes include **amylase**, which aids in the digestion of carbohydrates; **trypsin**, which aids in the digestion of proteins; and **lipase**, which aids in the digestion of fats. Other enzymes that promote the breakdown of more complex foodstuffs are also secreted.

Hormones originating in the GI tract stimulate the secretion of these exocrine pancreatic juices. The hormone **secretin** is the major stimulus for increased bicarbonate secretion from the pancreas, and the major stimulus for digestive enzyme secretion is the hormone CCK. The vagus nerve also influences exocrine pancreatic secretion.

The Endocrine Pancreas

The islets of Langerhans, the endocrine part of the pancreas, are collections of cells embedded in the pancreatic tissue. They are composed of alpha, beta, and delta cells. The hormone produced by the beta cells is called *insulin*, the alpha cells secrete glucagon, and the delta cells secrete somatostatin.

Insulin

A major action of insulin is to lower blood glucose by permitting entry of glucose into the cells of the liver, muscle, and other tissues, where it is either stored as glycogen or used for energy. Insulin also promotes the storage of fat in adipose tissue and the synthesis of proteins in various body tissues. In the absence of insulin, glucose cannot enter the cells and is excreted in the urine. This condition, called *diabetes*, can be diagnosed by high levels of glucose in the blood. In diabetes, stored fats and protein are used for energy instead of glucose, causing loss of body mass. Diabetes is discussed in detail in [Chapter 46](#). The level of glucose in the blood normally regulates the rate of insulin secretion from the pancreas (Goldman & Schafer, 2019; Norris, 2019).

Glucagon

The effect of glucagon (opposite to that of insulin) is chiefly to raise the blood glucose by converting glycogen to glucose in the liver. Glucagon is secreted by the pancreas in response to a decrease in the level of blood glucose.

Somatostatin

Somatostatin exerts a hypoglycemic effect by interfering with release of growth hormone from the pituitary and glucagon from the pancreas, both of which tend to raise blood glucose levels.

Endocrine Control of Carbohydrate Metabolism

Glucose required for energy is derived by metabolism of ingested carbohydrates and also from proteins by the process of gluconeogenesis. Glucose can be stored temporarily in the form of glycogen in the liver, muscles, and other tissues. The endocrine system controls the level of blood glucose by regulating the rate at which glucose is synthesized, stored, and moved to and from the bloodstream. Through the action of hormones, blood glucose is normally maintained at less than 100 mg/dL (5.6 mmol/L) (Norris, 2019; Papadakis & McPhee, 2020). Insulin is the primary hormone that lowers the blood glucose level. Hormones that raise the blood glucose level are glucagon, epinephrine, adrenocorticosteroids, growth hormone, and thyroid hormone.

The endocrine and exocrine functions of the pancreas are interrelated. The major exocrine function is to facilitate digestion through secretion of enzymes into the proximal duodenum. Secretin and CCK are hormones from the GI tract that aid in the digestion of food substances by controlling the secretions of the pancreas. Neural factors also influence pancreatic enzyme secretion. Considerable dysfunction of the pancreas must occur before enzyme secretion decreases and protein and fat digestion becomes impaired. Pancreatic enzyme secretion is normally 1500 to 3000 mL/day (Norris, 2019; Papadakis & McPhee, 2020).



Gerontologic Considerations

There is little change in the size of the pancreas with age. However, there is an increase in fibrous material and some fatty deposition in the normal pancreas in people older than 70 years. Some localized arteriosclerotic changes occur with age. There is also a decreased rate of pancreatic enzyme secretion (i.e., amylase, lipase, and trypsin) and decreased bicarbonate output in older adults. Some impairment of normal fat absorption occurs with increasing age, possibly because of delayed gastric emptying and pancreatic insufficiency (Eliopoulos, 2018; Norris, 2019; Papadakis & McPhee, 2020). Decreased calcium absorption may also occur. These changes require care in interpreting diagnostic test results in the normal older patient and in providing dietary counseling.

DISORDERS OF THE GALLBLADDER

Several disorders affect the biliary system and interfere with normal drainage of bile into the duodenum. These disorders include inflammation of the biliary system and carcinoma that obstructs the biliary tree. Gallbladder disease with stones is the most common disorder of the biliary system. Not all occurrences of cholecystitis are related to stones (calculi) in the gallbladder (**cholelithiasis**) or stones in the CBD (**choledocholithiasis**). However, most of the 15 million Americans with gallstones have no pain and are unaware of the presence of stones (Cameron & Cameron, 2020; Kellerman & Rakel, 2018).

Cholecystitis

Cholecystitis (inflammation of the gallbladder which can be acute or chronic) causes pain, tenderness, and rigidity of the upper right abdomen that may radiate to the midsternal area or right shoulder and is associated with nausea, vomiting, and the usual signs of an acute inflammation. An empyema of the gallbladder develops if the gallbladder becomes filled with purulent fluid (pus).

Calculus cholecystitis is the cause of more than 90% of cases of acute cholecystitis (Brunicardi, 2019; Cameron & Cameron, 2020). In calculus cholecystitis, a gallbladder stone obstructs bile outflow. Bile remaining in the gallbladder initiates a chemical reaction; autolysis and edema occur; and the blood vessels in the gallbladder are compressed, compromising its vascular supply. Gangrene of the gallbladder with perforation may result. Bacteria play a minor role in acute cholecystitis; however, secondary infection of bile occurs in approximately 50% of cases. The organisms involved are generally enteric (normally live in the GI tract) and include *Escherichia coli*, *Klebsiella* species, and *Streptococcus*. Bacterial contamination is not believed to stimulate the actual onset of acute cholecystitis (Feldman, Friedman, & Brandt, 2016; Goldman & Schafer, 2019).

Acalculous cholecystitis describes acute gallbladder inflammation in the absence of obstruction by gallstones. Acalculous cholecystitis occurs after major surgical procedures, orthopedic procedures, severe trauma, or burns. Other factors associated with this type of cholecystitis include torsion, cystic duct obstruction, primary bacterial infections of the gallbladder, and multiple blood transfusions. It is speculated that acalculous cholecystitis is caused by alterations in fluids and electrolytes and alterations in regional blood flow in the visceral circulation. Bile stasis (lack of gallbladder contraction) and increased viscosity of the bile are also thought to play a role. The occurrence of acalculous cholecystitis with major surgical procedures or trauma makes its diagnosis difficult (Brunicardi, 2019; Cameron & Cameron, 2020; Hammer & McPhee, 2019).

Cholelithiasis

Calculi, or gallstones, usually form in the gallbladder from the solid constituents of bile; they vary greatly in size, shape, and composition (see Fig. 44-2). They are uncommon in children and young adults but become more prevalent with increasing age. It is estimated that the prevalence of gallstones ranges from 5% to 20% in women between the ages of 20 and 55 years and from 25% to 30% in women older than 50 years. Cholelithiasis affects approximately 50% of women by the age of 70 years (Littlefield & Lenahan, 2019).

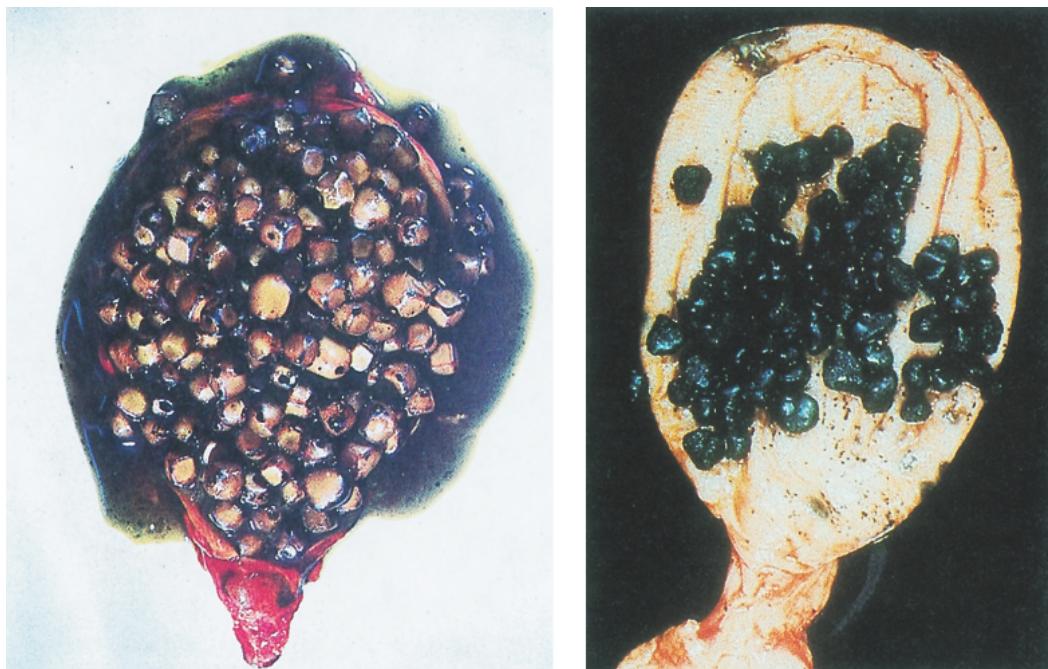


Figure 44-2 • Examples of cholesterol gallstones (**left**) made up of a coalescence of multiple small stones and pigment gallstones (**right**) composed of calcium bilirubinate. Reprinted with permission from Strayer, D. S., & Rubin, E. (2015). *Rubin's pathology: Clinicopathologic foundations of medicine* (7th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

Pathophysiology

The pathophysiology of gallstone development is multifactorial. There are two major types of gallstones: those composed predominantly of pigment and those composed primarily of cholesterol. Pigment stones probably form when unconjugated pigments in the bile precipitate to form stones; these stones account for about 10% to 25% of cases in the United States (Hammer & McPhee, 2019). The risk of developing such stones is increased in patients

with cirrhosis, hemolysis, and infections of the biliary tract. Pigment stones cannot be dissolved and must be removed surgically.

Cholesterol stones account for most of the remaining 75% of cases of gallbladder disease in the United States. Cholesterol, which is a normal constituent of bile, is insoluble in water. Its solubility depends on bile acids and lecithin (phospholipids) in bile (Hammer & McPhee, 2019). In gallstone-prone patients, there is decreased bile acid synthesis and increased cholesterol synthesis in the liver, resulting in bile supersaturated with cholesterol, which precipitates out of the bile to form stones (Hammer & McPhee, 2019). The cholesterol-saturated bile predisposes to the formation of gallstones and acts as an irritant that produces inflammatory changes in the mucosa of the gallbladder (Hammer & McPhee, 2019).

Two to three times more women than men develop cholesterol stones and gallbladder disease; affected women are usually older than 40 years, multiparous, and have obesity (Feldman et al., 2016; Goldman & Schafer, 2019; Hammer & McPhee, 2019). Stone formation is more frequent in people who use oral contraceptives, estrogens, or clofibrate; these medications are known to increase biliary cholesterol saturation (Hammer & McPhee, 2019). The incidence of stone formation increases with age as a result of increased hepatic secretion of cholesterol and decreased bile acid synthesis (Hammer & McPhee, 2019). In addition, there is an increased risk because of malabsorption of bile salts in patients with GI disease or T-tube fistula and in those who have undergone ileal resection or bypass. The incidence is also greater in people with diabetes (see [Chart 44-1](#)). The role of diet in the causation of cholesterol stones has not been confirmed but is under study. Those at high risk may be encouraged to maintain an optimal body weight and consider reducing modifiable risk factors by avoiding consumption of sugar and sweet foods, low-fiber foods, and fast foods (Di Ciaula, Garruti, Frühbeck, et al., 2019).

Chart 44-1 RISK FACTORS

Cholelithiasis

- Cystic fibrosis
- Diabetes
- Frequent changes in weight
- Ileal resection or disease
- Low-dose estrogen therapy—carries a small increase in the risk of gallstones
- Obesity
- Rapid weight loss (leads to rapid development of gallstones and high risk of symptomatic disease)
- Treatment with high-dose estrogen
- Women, especially those who have had multiple pregnancies or who are of Native American or U.S. southwestern Hispanic ethnicity

Adapted from Cox, M. R., Eslick, G. D., & Padbury, R. (2018). *The management of gallstone disease: A practical and evidence-based approach*. Cham, Switzerland: Springer Publishing.

Clinical Manifestations

Gallstones may be silent, producing no pain and only mild GI symptoms. Such stones may be detected incidentally during surgery or evaluation for unrelated problems (Hammer & McPhee, 2019; Srinivasan & Friedman, 2018).

The patient with gallbladder disease resulting from gallstones may develop two types of symptoms: those due to disease of the gallbladder itself and those due to obstruction of the bile passages by a gallstone. The symptoms may be acute or chronic. Epigastric distress, such as fullness, abdominal distention, and vague pain in the right upper quadrant of the abdomen, may occur. This distress may follow a meal rich in fried or fatty foods (Brunicardi, 2019; Cameron & Cameron, 2020; Hammer & McPhee, 2019).

Pain and Biliary Colic

If a gallstone obstructs the cystic duct, the gallbladder becomes distended, inflamed, and eventually infected (acute cholecystitis). The patient develops a fever and may have a palpable abdominal mass. The patient may have biliary colic with excruciating upper right abdominal pain that radiates to the back or right shoulder. Biliary colic is usually associated with nausea and vomiting, and it is noticeable several hours after a heavy meal. The patient moves about restlessly, unable to find a comfortable position. In some patients, the pain is constant rather than colicky (Brunicardi, 2019; Cameron & Cameron, 2020).

Such a bout of biliary colic is caused by contraction of the gallbladder, which cannot release bile because of obstruction by the stone. When distended, the fundus of the gallbladder comes in contact with the abdominal wall in the

region of the right ninth and 10th costal cartilages. This produces marked tenderness in the right upper quadrant on deep inspiration and prevents full inspiratory excursion.

The pain of acute cholecystitis may be so severe that analgesic medications are required. The use of morphine has traditionally been avoided because of concern that it could cause spasm of the sphincter of Oddi. This is controversial, because morphine is the preferred analgesic agent for management of acute pain. Furthermore, all opioids stimulate the sphincter of Oddi to some degree (Littlefield & Lenahan, 2019; Papadakis & McPhee, 2020).

If the gallstone is dislodged and no longer obstructs the cystic duct, the gallbladder drains and the inflammatory process subsides after a relatively short time. If the gallstone continues to obstruct the duct, abscess, necrosis, and perforation with generalized peritonitis may result.

Jaundice

Jaundice occurs in a few patients with gallbladder disease, usually with obstruction of the common bile duct. The bile, which is no longer carried to the duodenum, is absorbed by the blood and gives the skin and mucous membranes a yellow color. This is frequently accompanied by marked pruritus (itching) of the skin.

Changes in Urine and Stool Color

The excretion of the bile pigments by the kidneys gives the urine a very dark color. The feces, no longer colored with bile pigments, are grayish (like putty) or clay colored.

Vitamin Deficiency

Obstruction of bile flow interferes with absorption of the fat-soluble vitamins A, D, E, and K. Patients may exhibit deficiencies of these vitamins if biliary obstruction has been prolonged. For example, a patient may have bleeding caused by vitamin K deficiency (vitamin K is necessary for normal blood clotting).

Assessment and Diagnostic Findings

A wide range of diagnostic studies may be performed in patients with biliary disorders. [Table 44-1](#) identifies various procedures and their diagnostic uses. The nurse should educate the patient about the purpose, what to expect, and any possible side effects related to these examinations prior to testing. The nurse should note trends in results because they provide information about disease progression as well as the patient's response to therapy.

TABLE 44-1 Studies Used in the Diagnosis of Biliary Tract and Pancreatic Disease

Studies	Diagnostic Uses
Magnetic resonance cholangiopancreatography (MRCP)	Visualizes the biliary tree and capable of detecting biliary tract obstruction
Cholecystogram, cholangiogram	Visualize gallbladder and bile duct
Celiac axis arteriography	Visualizes liver and pancreas
Laparoscopy	Visualizes anterior surface of liver, gallbladder, and mesentery through a trocar
Ultrasonography	Shows size of abdominal organs and presence of masses
Helical computed tomography and magnetic resonance imaging	Detect neoplasms; diagnose cysts, pseudocysts, abscess, and hematomas; determine severity of pancreatitis based on the presence of necrosis or peripancreatic fluid collections
Endoscopic retrograde cholangiopancreatography	Visualizes biliary structures and pancreas via endoscopy
Endoscopic ultrasound (EUS)	Identifies small tumors and other abnormalities and facilitate fine-needle aspiration biopsy of tumors or lymph nodes for diagnosis
Serum alkaline phosphatase	In the absence of bone disease, to measure biliary tract obstruction
Gamma-glutamyl, gamma-glutamyl transpeptidase, lactate dehydrogenase	Markers for biliary stasis; also elevated in alcohol abuse
Cholesterol levels	Elevated in biliary obstruction; decreased in parenchymal liver disease

Adapted from Fischbach, F. T., & Fischbach, M. A. (2018). *Fischbach's manual of laboratory and diagnostic tests* (10th ed.). Philadelphia, PA: Wolters Kluwer.

Abdominal X-Ray

If the patient presents with symptoms of gallbladder disease, an abdominal x-ray may be obtained to exclude other causes of symptoms. However, only 10% to 15% of gallstones are calcified sufficiently to be visible on such x-ray studies (Brunicardi, 2019; Goldman & Shafer, 2019).

Ultrasonography

Ultrasonography is the diagnostic procedure of choice because it is rapid and accurate and can be used in patients with liver dysfunction and jaundice. It does not expose patients to ionizing radiation. The procedure is most accurate if the patient fasts overnight so that the gallbladder is distended.

Ultrasonography can detect calculi in the gallbladder or a dilated CBD with 90% accuracy (Brunicardi, 2019; Goldman & Shafer, 2019; Littlefield & Lenahan, 2019; Mou, Tesfasilassie, Hirji, et al., 2019).

Radionuclide Imaging or Cholescintigraphy

Cholescintigraphy is used successfully in the diagnosis of acute cholecystitis or blockage of a bile duct (Brunicardi, 2019; Goldman & Schafer, 2019; Littlefield & Lenahan, 2019; Mou et al., 2019). During this procedure, a radioactive agent is administered intravenously (IV), which is taken up by the hepatocytes and excreted rapidly through the biliary tract. The biliary tract is then scanned, and images of the gallbladder and biliary tract are obtained. This test is more expensive than ultrasonography, takes longer to perform, and exposes the patient to radiation. It is often used when ultrasonography is not conclusive, such as in acalculous cholecystitis (Brunicardi, 2019; Goldman & Schafer, 2019; Littlefield & Lenahan, 2019; Mou et al., 2019).

Oral Cholecystography

Oral cholecystography is used if ultrasound equipment is not available or if the ultrasound results are inconclusive. This study may be performed to detect gallstones and to assess the ability of the gallbladder to fill, concentrate its contents, contract, and empty. If the patient is not allergic to iodine or seafood, an iodide-containing contrast agent that is excreted by the liver and concentrated in the gallbladder is given 10 to 12 hours before the x-ray study (Brunicardi, 2019; Goldman & Schafer, 2019; Littlefield & Lenahan, 2019; Mou et al., 2019). The normal gallbladder fills with this radiopaque substance. If gallstones are present, they appear as shadows on the x-ray image.

Oral cholecystography may be used as part of the evaluation of patients who have been treated with gallstone **dissolution therapy** (the use of medications to break up/dissolve gallstones) or **lithotripsy** (disintegration of gallstones by shock waves).

Endoscopic Retrograde Cholangiopancreatography

Endoscopic retrograde cholangiopancreatography (ERCP) permits direct visualization of structures that previously could be seen only during laparotomy. This procedure examines the hepatobiliary system via a side-viewing flexible fiberoptic endoscope inserted through the esophagus to the descending duodenum (see Fig. 44-3). Multiple position changes are required to pass the endoscope during the procedure, beginning in the left semiprone position.

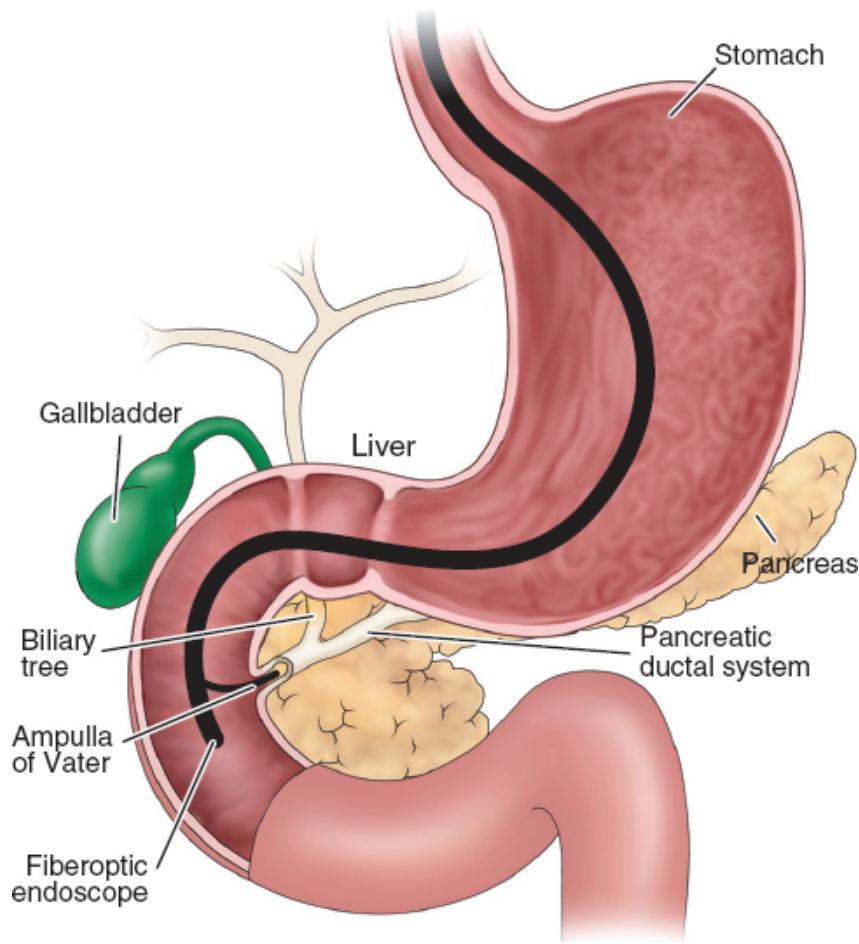


Figure 44-3 • Endoscopic retrograde cholangiopancreatography. A fiberoptic duodenoscope, with side-viewing apparatus, is inserted into the duodenum. The ampulla of Vater is catheterized, and the biliary tree is injected with contrast agent. The pancreatic ductal system is also assessed, if indicated. This procedure is of special value in visualizing neoplasms of the ampulla area and extracting a biopsy specimen.

Fluoroscopy and multiple x-rays are used during ERCP to evaluate the presence and location of ductal stones. Careful insertion of a catheter through the endoscope into the CBD is the most important step in sphincterotomy (division of the muscles of the biliary sphincter) for gallstone extraction via this technique (see later discussion). ERCP is not recommended for the evaluation of suspected CBD stones but can be used to treat confirmed choledocholithiasis before or during laparoscopic cholecystectomy (Brunicardi, 2019; Cameron & Cameron, 2020).

Nursing Implications

Before ERCP, the patient is educated about the procedure and their role in it. This preparation can allay anxiety and facilitate the insertion of the endoscope without damage to the GI tract structures, including the biliary tree. The patient takes nothing by mouth for several hours before the procedure. The procedure requires intravenous (IV) sedation and monitored anesthesia care. In some cases general anesthesia is required, and the sedated patient is monitored closely during and after the procedure (Brunicardi, 2019). It may be necessary to administer medications, such as glucagon or anticholinergic agents, to make cannulation easier by decreasing duodenal peristalsis. The nurse observes closely for signs of respiratory and CNS depression, hypotension, oversedation, and vomiting (if glucagon is given). During ERCP, the nurse monitors IV fluids, administers medications, and positions the patient. After the procedure, the nurse monitors the patient's condition, observing vital signs and assessing for signs of perforation or infection. The nurse also monitors the patient for side effects of any medications received during the procedure.

Percutaneous Transhepatic Cholangiography

Percutaneous transhepatic cholangiography (PTC) is rarely used for diagnostic purposes alone due to the multitude of other less invasive and reliable imaging studies. PTC is reserved for those patients in whom an ERCP may be unsafe due to previous surgery involving the biliary tract (Brunicardi, 2019; Cameron & Cameron, 2020; Feldman et al., 2016). The use of PTC has mainly been replaced by ERCP and magnetic resonance cholangiopancreatography (MRCP). PTC involves the injection of dye directly into the biliary tract. Because of the relatively large concentration of dye that is introduced into the biliary system, including the hepatic ducts within the liver, the entire length of the common bile duct, the cystic duct, and the gallbladder is outlined clearly.

This procedure can be carried out in the presence of liver dysfunction and jaundice. It is useful for distinguishing jaundice caused by liver disease (hepatocellular jaundice) from that caused by biliary obstruction, investigating the GI symptoms of a patient whose gallbladder has been removed, locating stones within the bile ducts, and diagnosing cancer involving the biliary system (Brunicardi, 2019; Cameron & Cameron, 2020; Feldman et al., 2016).

This sterile procedure is performed under moderate sedation on a patient who has been fasting; the patient also receives local anesthesia. Coagulation parameters and platelet count should be normal to minimize the risk of bleeding. Broad-spectrum antibiotics are given during the procedure because of the high prevalence of bacterial colonization from obstructed biliary systems (Feldman et al., 2016; Brunicardi, 2019; Cameron & Cameron, 2020). After infiltration with a local anesthetic agent has occurred, a flexible needle is inserted into the liver from the right side in the midclavicular line immediately beneath the right costal margin. Successful entry of a duct is noted when bile is aspirated or on injection of a contrast agent. Ultrasound can be used to guide

puncture of the duct. Bile is aspirated, and samples are sent for bacteriology and cytology (Brunicardi, 2019; Feldman et al., 2016; Kellerman & Rakel, 2018). A water-soluble contrast agent is injected to fill the biliary system. The fluoroscopy table is tilted and the patient is repositioned to allow x-rays to be taken in multiple projections. Delayed x-ray views can identify abnormalities of more distant ducts and determine the length of a stricture or multiple strictures. Before the needle is removed, as much dye and bile as possible are aspirated to forestall subsequent leakage into the needle tract and eventually into the peritoneal cavity, thus minimizing the risk of bile peritonitis.

Nursing Implications

Although the complication rate after this procedure is low, the nurse must closely observe the patient for symptoms of bleeding, peritonitis, and sepsis. The nurse assesses the patient for pain and indications of these complications and reports them promptly to the primary provider, takes measures to reassure the patient, and ensures patient comfort. Antibiotic agents are often prescribed to minimize the risk of sepsis and septic shock.

Medical Management

The major objectives of medical therapy are to reduce the incidence of acute episodes of gallbladder pain and cholecystitis by supportive and dietary management and, if possible, to remove the cause of cholecystitis by pharmacologic therapy, endoscopic procedures, or surgical intervention. Although nonsurgical procedures eliminate risks associated with surgery, these approaches are associated with persistent symptoms or recurrent stone formation. Most of the nonsurgical approaches, including lithotripsy and dissolution of gallstones, provide only temporary solutions to gallstone problems and are infrequently used in the United States. In some instances, other treatment approaches may be indicated; these are described later.

Cholecystectomy (removal of the gallbladder) through traditional surgical approaches has largely been replaced by laparoscopic cholecystectomy (removal of the gallbladder through a small incision through the umbilicus) (see later discussion). As a result, surgical risks have decreased, along with the length of hospital stay and the long recovery period required after standard surgical cholecystectomy. In relatively rare instances, a standard surgical procedure may be necessary.

Nutritional and Supportive Therapy

Approximately 80% of the patients with acute gallbladder inflammation achieve remission with rest, IV fluids, nasogastric suction, analgesia, and antibiotic agents. Unless the patient's condition deteriorates, surgical intervention is delayed just until the acute symptoms subside (usually within a

few days). At this time, the patient undergoes a laparoscopic cholecystectomy (Brunicardi, 2019; Cameron & Cameron, 2020; Goldman & Shafer, 2019).

The diet immediately after an episode is usually low-fat liquids. These can include powdered supplements high in protein and carbohydrate stirred into skim milk. Cooked fruits, rice or tapioca, lean meats, mashed potatoes, non-gas-forming vegetables, bread, coffee, or tea may be added as tolerated. The patient should avoid eggs, cream, pork, fried foods, cheese, rich dressings, gas-forming vegetables, and alcohol. It is important to remind the patient that fatty foods may induce an episode of cholecystitis. Dietary management may be the major mode of therapy in patients who have had only dietary intolerance to fatty foods and vague GI symptoms (Kellerman & Rakel, 2018).

Pharmacologic Therapy

Ursodeoxycholic acid (UDCA) and chenodeoxycholic acid (chenodiol or CDCA) have been used to dissolve small, radiolucent gallstones composed primarily of cholesterol (Goldman & Shafer, 2019). UDCA has fewer side effects than chenodiol and can be given in smaller doses to achieve the same effect. It acts by inhibiting the synthesis and secretion of cholesterol, thereby desaturating bile. Treatment with UDCA can reduce the size of existing stones, dissolve small stones, and prevent new stones from forming. Six to 12 months of therapy is required in many patients to dissolve stones, and monitoring of the patient for recurrence of symptoms or the occurrence of side effects (e.g., GI symptoms, pruritus, headache) is required during this time. The effective dose of medication depends on body weight. This method of treatment is generally indicated for patients who refuse surgery or for whom surgery is contraindicated. The success rate of this therapy is low as the recurrence following it is high (Goldman & Shafer, 2019).

Patients with frequent symptoms, cystic duct occlusion, or pigment stones are not candidates for pharmacologic therapy. Laparoscopic or open cholecystectomy is more appropriate for symptomatic patients with acceptable operative risk (Goldman & Shafer, 2019).

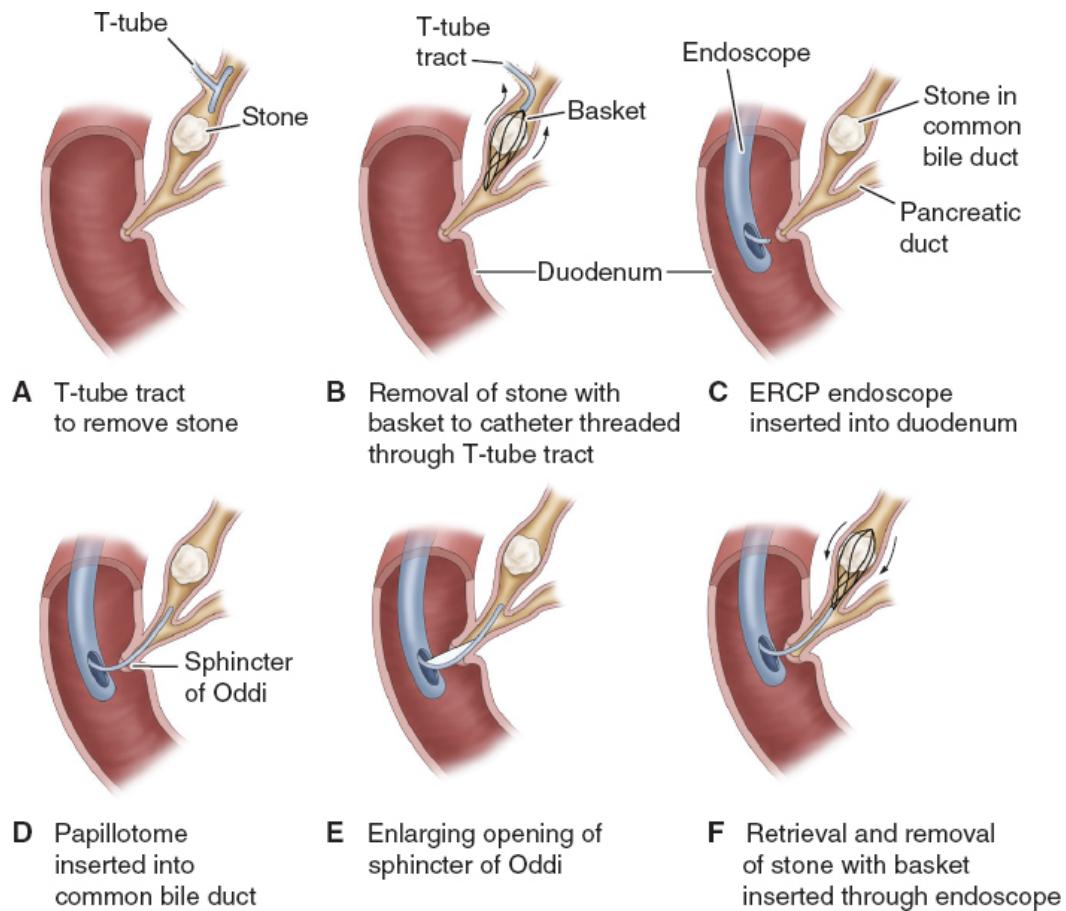


Figure 44-4 • A–F. Procedures for removing gallstones.

Nonsurgical Removal of Gallstones

Dissolving Gallstones

Several methods have been used to dissolve gallstones by infusion of a solvent (mono-octanoin or methyl tertiary butyl ether [MTBE]) into the gallbladder. The solvent can be infused through the following routes: through a tube or catheter inserted percutaneously directly into the gallbladder; through a tube or drain inserted through a T-tube tract to dissolve stones not removed at the time of surgery, endoscopically with ERCP; or via a transnasal biliary catheter, a rarely used procedure due to its lack of success, potential side effects, and recurrence rates of up to 50% (Goldman & Shafer, 2019; Townsend, Beauchamp, Evers, et al., 2016).

Laparoscopic cholecystectomy is the standard for management. Dissolution therapies are used for those patients who may not be candidates for the procedure due to safety concerns regarding general anesthesia (Goldman & Shafer, 2019; Townsend et al., 2016).

Stone Removal by Instrumentation

Several methods are used to remove stones that were not removed at the time of cholecystectomy or have become lodged in the CBD (see Fig. 44-4A,B). A catheter and instrument with a basket attached are threaded through the T-tube tract or fistula formed at the time of T-tube insertion; the basket is used to retrieve and remove the stones lodged in the common bile duct.

A second procedure involves the use of the ERCP endoscope (see Fig. 44-4C). After the endoscope is inserted, a cutting instrument is passed through the endoscope into the ampulla of Vater of the common bile duct. It may be used to cut the submucosal fibers, or papilla, of the sphincter of Oddi, enlarging the opening, which may allow the lodged stones to pass spontaneously into the duodenum. Another instrument with a small basket or balloon at its tip may be inserted through the endoscope to retrieve the stones (see Fig. 44-4D–F). The patient is observed closely for bleeding, perforation, and the development of pancreatitis (see later discussion) or sepsis.

The ERCP procedure is particularly useful in diagnosis and treatment of patients who have symptoms after biliary tract surgery, patients with intact gallbladders, and patients for whom surgery is particularly hazardous.

Intracorporeal Lithotripsy

Stones in the gallbladder or CBD may be fragmented by means of laser pulse technology. A laser pulse is directed under fluoroscopic guidance with the use of devices that can distinguish between stones and tissue. The laser pulse produces rapid expansion and disintegration of plasma on the stone surface, resulting in a mechanical shock wave. Electrohydraulic lithotripsy uses a probe with two electrodes that deliver electric sparks in rapid pulses, creating expansion of the liquid environment surrounding the gallstones. This results in pressure waves that cause stones to fragment. This technique can be used percutaneously with a basket or balloon catheter system or by direct visualization through an endoscope. Repeated procedures may be necessary because of stone size, local anatomy, bleeding, or technical difficulty. A nasobiliary tube can be inserted to allow for biliary decompression and to prevent stone impaction in the common bile duct. This approach allows time for improvement in the patient's clinical condition until gallstones are cleared endoscopically, percutaneously, or surgically.

Extracorporeal Shock Wave Lithotripsy

Extracorporeal shock wave lithotripsy (ESWL) has been used for nonsurgical fragmentation of gallstones. ESWL is a noninvasive procedure that uses repeated shock waves directed at the gallstones in the gallbladder or CBD to fragment the stones. The waves are transmitted to the body through a fluid-filled bag or by immersing the patient in a water bath. After the stones are gradually broken up, the stone fragments can be spontaneously passed from the gallbladder or common bile duct, removed by endoscopy, or dissolved with

oral bile acid or solvents. Because the procedure requires no incision and no hospitalization, patients are usually treated as outpatients, but usually several sessions are necessary. This procedure has largely been replaced by laparoscopic cholecystectomy. ESWL is used in some centers for a small percentage of suitable patients (those with CBD stones who may not be surgical candidates), sometimes in combination with dissolution therapy (Feldman et al., 2016; Kellerman & Rakel, 2018).

Surgical Management

Surgical treatment of gallbladder disease and gallstones is carried out to relieve persistent symptoms, to remove the cause of biliary colic, and to treat acute cholecystitis. Surgery may be delayed until the patient's symptoms have subsided, or it may be performed as an emergency procedure, if necessitated by the patient's condition.

Preoperative Measures

Chest x-ray, electrocardiogram, and liver function tests may be performed in addition to imaging studies of the gallbladder. Vitamin K may be given if the prothrombin level is low. Nutritional requirements are considered, and, if the nutritional status is suboptimal, it may be necessary to provide IV glucose with protein supplements to aid wound healing and help prevent liver damage.

Patient education for gallbladder surgery is similar to that for any upper abdominal laparotomy or laparoscopy. Instructions and explanations are given before surgery about turning and deep breathing. Postoperative pneumonia and atelectasis can be avoided by deep-breathing exercises, frequent turning, and early ambulation. The patient should be informed that drainage tubes and a nasogastric tube and suction might be required during the immediate postoperative period if an open cholecystectomy is performed.

Laparoscopic Cholecystectomy

Laparoscopic cholecystectomy (see Fig. 44-5) is the standard of therapy for symptomatic gallstones. Approximately 700,000 patients in the United States require surgery each year for removal of the gallbladder, and 80% to 90% of them are candidates for laparoscopic cholecystectomy (Brunicardi, 2019; Cameron & Cameron, 2020; Feldman et al., 2016; Goldman & Shafer, 2019). If the CBD is thought to be obstructed by a gallstone, an ERCP with sphincterotomy may be performed to explore the duct before laparoscopy (Brunicardi, 2019; Cameron & Cameron, 2020; Goldman & Shafer, 2019; Littlefield & Lenahan, 2019; Mou et al., 2019).

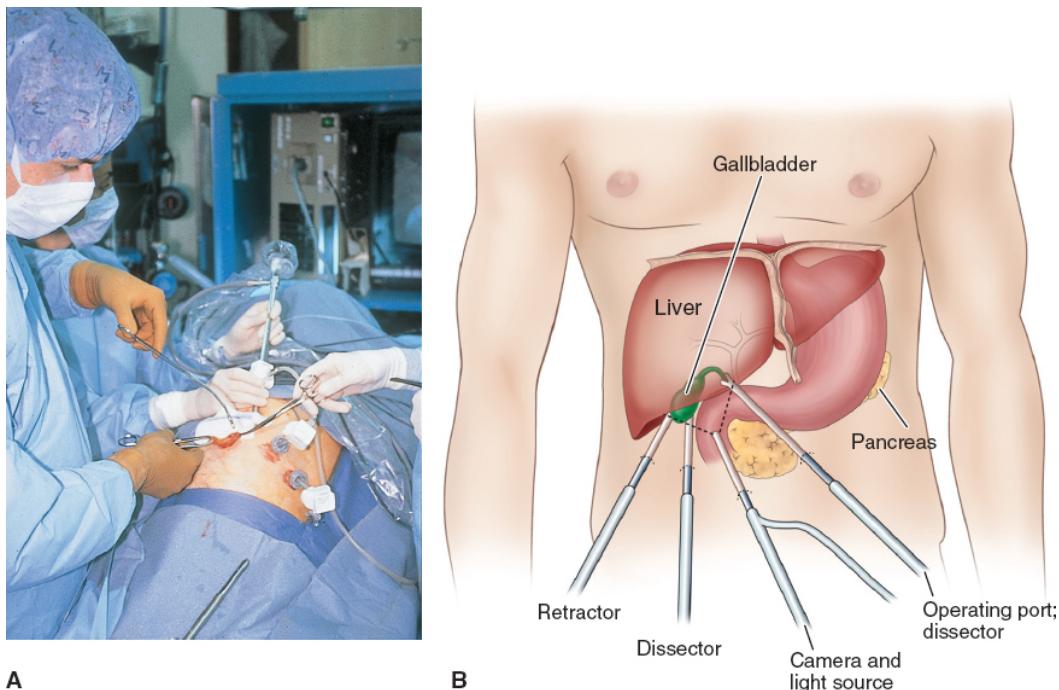


Figure 44-5 • In laparoscopic cholecystectomy (**A**), the surgeon makes four small incisions (less than one half inch each) in the abdomen and inserts a laparoscope with a miniature camera through the umbilical incision (**B**). The camera apparatus displays the gallbladder and adjacent tissues on a screen, allowing the surgeon to visualize the sections of the organ for removal.

Before the procedure, the patient is educated that an open abdominal procedure may be necessary, and general anesthesia is given. Laparoscopic cholecystectomy is performed through a small incision or puncture made through the abdominal wall at the umbilicus. The abdominal cavity is insufflated with carbon dioxide (pneumoperitoneum) to assist in inserting the laparoscope and to aid in visualizing the abdominal structures. The fiberoptic scope is inserted through the small umbilical incision. Several additional punctures or small incisions are made in the abdominal wall to introduce other surgical instruments into the operative field. A camera attached to the laparoscope permits the surgeon to view the intra-abdominal field and biliary system on a television monitor. After the cystic duct is dissected, the CBD can be visualized by ultrasound or cholangiography to evaluate the anatomy and identify stones. The cystic artery is dissected free and clipped. The gallbladder is separated from the hepatic bed and removed from the abdominal cavity after bile and small stones are aspirated. Stone forceps also can be used to remove or crush larger stones.

With the laparoscopic procedure, the patient does not experience the paralytic ileus that occurs with open abdominal surgery and has less postoperative abdominal pain. The patient is often discharged from the hospital

on the same day of surgery or within 1 or 2 days and resumes full activity and employment within 1 week after the procedure.

Conversion to a traditional abdominal surgical procedure occurs in 2.2% of cases in the United States and 3.6% to 8.2% of cases internationally. Conversion to an open procedure occurs if there is inflammation in and around the gallbladder, making safe dissection of the porta hepatis difficult (Brunicardi, 2019; Feldman et al., 2016; Goldman & Shafer, 2019). The porta hepatis is the fissure of the liver where the portal vein and the hepatic artery enter and the hepatic ducts exit the liver.

The most serious complication after laparoscopic cholecystectomy is a bile duct injury, which may be identified and corrected at the time of the procedure. Patients with a postoperative bile leak may not develop symptoms until several days after the procedure, and some have an even more prolonged period before injury to the bile duct becomes apparent (Brunicardi, 2019; Cameron & Cameron, 2020). A bile leak may result in fluid collections, which can usually be managed by endoscopic stent placement. Bile peritonitis, a rare complication, may result in serious illness or death.

Because of the short length of hospital stay with uncomplicated laparoscopic cholecystectomies, it is important to provide patient education about managing postoperative pain and reporting signs and symptoms of intra-abdominal complications, including loss of appetite, vomiting, pain, distention of the abdomen, and temperature elevation. Although recovery from laparoscopic cholecystectomy is rapid, patients are drowsy afterward. The patient must have assistance at home during the first 24 to 48 hours. If pain occurs in the right shoulder or scapular area (from migration of the carbon dioxide used to insufflate the abdominal cavity during the procedure), the nurse may recommend a heating pad for 15 to 20 minutes hourly.

Cholecystectomy

In cholecystectomy, the gallbladder is removed through an abdominal incision (usually right subcostal) after the cystic duct and artery are ligated. The procedure is performed for acute and chronic cholecystitis. In some patients, a drain is placed close to the gallbladder bed and brought out through a puncture wound if there is a bile leak. The drain type is chosen based on the surgeon's preference. A small leak should close spontaneously in a few days, with the drain preventing accumulation of bile. Usually, only a small amount of serosanguineous fluid drains in the initial 24 hours after surgery; afterward, the drain is removed. The drain is typically maintained if there is excess oozing or bile leakage. Insertion of a T-tube (named for its shape) into the CBD during the open procedure is now uncommon; it is used only in the setting of a complication (i.e., retained CBD stone). A T-tube is inserted into the CBD at the time of surgical exploration. It allows external drainage of bile into a collection bag, allowing the surgical site to heal.

Bile duct injury is a serious complication of cholecystectomy, but it occurs less frequently than with the laparoscopic approach, which has largely replaced traditional surgical cholecystectomy.

Small-Incision Cholecystectomy

Small-incision cholecystectomy is a surgical procedure in which the gallbladder is removed through a small abdominal incision, as the name implies. If needed, the surgical incision is extended to remove larger gallbladder stones. Drains may or may not be used. The short length hospital stay has been identified as a major advantage of this type of procedure (Brunicardi, 2019; Cameron & Cameron, 2020; Goldman & Schafer, 2019). The procedure is controversial because it limits exposure to all involved biliary structures.

Choledochostomy

Choledochostomy is reserved for the patient with acute cholecystitis who may be too ill to undergo a surgical procedure. This procedure involves making an incision in the common duct, usually for removal of stones. After the stones have been evacuated, a tube is usually inserted into the duct for drainage of bile until edema subsides. This tube is connected to gravity drainage tubing; the patient is monitored closely, and a laparoscopic cholecystectomy is planned for a future date after acute inflammation has resolved.

Surgical Cholecystostomy

Cholecystostomy is performed when the patient's condition precludes more extensive surgery or when an acute inflammatory reaction is severe. The gallbladder is surgically opened, stones and the bile or the purulent drainage are removed, and a drainage tube is secured with a purse-string suture. The drainage tube is connected to a drainage system to prevent bile from leaking around the tube or escaping into the peritoneal cavity. After recovery from the acute episode, the patient may return for subsequent laparoscopic cholecystectomy. Despite its lower risk, surgical cholecystostomy has a high mortality rate (reported to be as high as 10% to 30%) because of the underlying infectious disease process (Brunicardi, 2019; Cameron & Cameron, 2020; Feldman et al., 2016; Goldman & Schafer, 2019).

Percutaneous Cholecystostomy

Percutaneous cholecystostomy has been used in the treatment and diagnosis of acute cholecystitis in patients who are poor risks for any surgical procedure or for general anesthesia. This at-risk population may include patients with sepsis or severe cardiac, renal, pulmonary, or liver failure (Goldman & Shafer, 2019; Roberts, Plotnik, Chick, et al., 2019). Under local anesthesia, a fine needle is inserted through the abdominal wall and liver edge into the gallbladder under

the guidance of ultrasound or computed tomography (CT). Bile is aspirated to ensure adequate placement of the needle, and a catheter is inserted into the gallbladder to decompress the biliary tract. Almost immediate relief of pain and resolution of signs and symptoms of sepsis and cholecystitis have been reported with this procedure. Antibiotic agents are given before, during, and after the procedure.

Endoscopic Ultrasound

Endoscopic ultrasound (EUS) is a guided gallbladder drainage procedure that is an effective treatment option with success rates comparable to percutaneous drainage (Goldman & Shafer, 2019). EUS is described in more detail later in the chapter.



Gerontologic Considerations

Surgical intervention for disease of the biliary tract is the most common operative procedure performed in the older adult. Cholesterol saturation of bile increases with age because of increased hepatic secretion of cholesterol and decreased bile acid synthesis.

Although the incidence of gallstones increases with age, the older patient may not exhibit the typical symptoms of fever, pain, chills, and jaundice. Symptoms of biliary tract disease in the older adult may be accompanied or preceded by those of septic shock, which include oliguria, hypotension, changes in mental status, tachycardia, and tachypnea.

Although surgery in the older adult presents a risk because of preexisting associated diseases, the mortality rate from serious complications of biliary tract disease itself is also high. The risk of death and complications is increased in the older patient who undergoes emergency surgery for life-threatening disease of the biliary tract. Despite the presence of chronic illness in many older patients, elective cholecystectomy is usually well tolerated and can be carried out with low risk if expert assessment and care are provided before, during, and after the surgical procedure (Rothrock, 2019).

The higher risk of complications and shorter length of hospital stay make it essential that older patients and their family members receive specific information about signs and symptoms of complications and measures to prevent them.

NURSING PROCESS

The Patient Undergoing Surgery for Gallbladder Disease

Assessment

The patient undergoing surgical treatment of gallbladder disease is often admitted to the hospital or same-day surgery unit on the morning of surgery. Preadmission testing is often completed a week or longer before admission. At that time, the nurse educates the patient about the need to avoid smoking, to enhance pulmonary recovery postoperatively, and to avoid respiratory complications. The need to avoid aspirin, nonsteroidal antiinflammatory drugs, and other agents (over-the-counter medications and herbal remedies) that can alter coagulation and other biochemical processes is also emphasized.

Assessment should focus on the patient's respiratory status. If a traditional surgical approach is planned, the high abdominal incision required during surgery may interfere with full respiratory excursion. The nurse notes a history of smoking, previous respiratory problems, shallow respirations, a persistent or ineffective cough, and the presence of adventitious breath sounds. Nutritional status is evaluated through a dietary history and a general examination performed at the time of preadmission testing. The nurse also reviews previously obtained laboratory results to obtain information about the patient's nutritional status.

Diagnosis

NURSING DIAGNOSES

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

- Acute pain and discomfort associated with surgical incision
- Impaired gas exchange associated with the high abdominal surgical incision (if traditional surgical cholecystectomy was performed)
- Impaired skin integrity associated with altered biliary drainage after surgical intervention (if a T-tube was inserted because of retained stones in the common bile duct or another drainage device was employed)
- Impaired nutritional status associated with inadequate bile secretion
- Lack of knowledge about self-care activities associated with incision care, dietary modifications (if needed), medications, and reportable signs or symptoms (e.g., fever, bleeding, vomiting)

COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS

Potential complications may include the following:

- Bleeding
- GI symptoms (may be related to biliary leak or injury to the bowel)

Planning and Goals

The goals for the patient include relief of pain, adequate ventilation, intact skin and improved biliary drainage, optimal nutritional intake, absence of complications, and understanding of self-care routines.

Nursing Interventions

After recovery from anesthesia, the patient is placed in the low Fowler position. Fluids may be administered IV, and nasogastric suction (a nasogastric tube was probably inserted immediately before surgery for a nonlaparoscopic procedure) may be instituted to relieve abdominal distention. Water and other fluids are given within hours after laparoscopic procedures. A soft diet is started after bowel sounds return, which is usually the next day if the laparoscopic approach is used.

RELIEVING PAIN

The location of the subcostal incision in nonlaparoscopic gallbladder surgery often causes the patient to avoid turning and moving, to splint the affected site, and to take shallow breaths to prevent pain. Because full expansion of the lungs and gradually increased activity are necessary to prevent postoperative complications, the nurse administers analgesic agents as prescribed to relieve the pain and to help the patient turn, cough, breathe deeply, and ambulate as indicated. The use of a pillow or abdominal binder over the incision may reduce pain during these maneuvers.

IMPROVING RESPIRATORY STATUS

Patients undergoing biliary tract surgery are especially prone to pulmonary complications, as are all patients with upper abdominal incisions. Therefore, the nurse reminds the patient to take deep breaths and cough every hour to expand the lungs fully and prevent atelectasis. The early and consistent use of incentive spirometry also helps improve respiratory function. Early ambulation prevents pulmonary complications as well as other complications, such as venous thromboembolism (VTE) formation. Pulmonary complications are more likely to occur in patients who are older, those with obesity, and those with preexisting pulmonary disease.

MAINTAINING SKIN INTEGRITY AND PROMOTING BILIARY DRAINAGE

In patients who have undergone a cholecystostomy or choledochostomy, the drainage tube must be connected immediately to a drainage receptacle. The nurse should fasten the tubing to the dressings or to the patient's gown, with enough leeway for the patient to move without dislodging or kinking the tube. Because a drainage system remains attached when the patient is ambulating, the drainage bag may be placed in a bathrobe pocket or fastened so that it is below the waist or common duct level. If drains are used, the nurse changes the dressings as required.

After these surgical procedures, the patient is observed for indications of infection, leakage of bile into the peritoneal cavity, and obstruction of bile drainage. If bile is not draining properly, an obstruction is probably causing bile to be forced back into the liver and bloodstream. Because jaundice may result, the nurse should assess the color of the sclerae. The nurse should note and report right upper quadrant abdominal pain, nausea and vomiting, bile drainage around any drainage tube, clay-colored stools, and a change in vital signs.

Bile may continue to drain from the drainage tract in considerable quantities for some time, necessitating frequent changes of the outer dressings and protection of the skin from irritation (bile is corrosive to the skin).

To prevent total loss of bile, the surgeon may want the drainage tube (T-tube) or collection receptacle elevated above the level of the abdomen so that the bile drains externally only if pressure develops in the duct system. Every 24 hours, the nurse measures the bile collected and records the amount, color, and character of the drainage. After several days of drainage, the T-tube may be clamped for 1 hour before and after each meal to deliver bile to the duodenum to aid in digestion (Brunicardi, 2019; Cameron & Cameron, 2020; Townsend et al., 2016). Within 7 days to 3 weeks, the drainage tube is removed (Brunicardi, 2019; Cameron & Cameron, 2020; Townsend et al., 2016). The patient who goes home with a drainage tube in place requires instruction and reassurance about the function and care of the T-tube (Rothrock, 2019).

In all patients with biliary drainage, the nurse (or the patient, if at home) observes the color of stools daily. Urine and stool specimens may be sent to the laboratory for examination for bile pigments. In this way, it is possible to determine whether the bile pigment is disappearing from the blood and is draining again into the duodenum. Maintaining a careful record of fluid intake and output is important.

IMPROVING NUTRITIONAL STATUS

The nurse encourages the patient to eat a diet that is low in fats and high in carbohydrates and proteins immediately after surgery. At the time of hospital discharge, there are usually no special dietary instructions other than to maintain a healthy diet and avoid excessive fats. Fat restriction usually is lifted in 4 to 6 weeks, when the biliary ducts dilate to accommodate the volume of bile once held by the gallbladder and when the ampulla of Vater again functions effectively. After this time, when the patient eats fat, adequate bile will be released into the GI tract to emulsify the fats and allow their digestion. This is in contrast to the condition before surgery, when fats may not have been digested completely or adequately and flatulence may have occurred. One purpose of gallbladder surgery is to allow a normal diet.

MONITORING AND MANAGING POTENTIAL COMPLICATIONS

Bleeding may occur as a result of inadvertent puncture or injury to a major blood vessel. Postoperatively, the nurse closely monitors vital signs and inspects the surgical incisions and any drains for bleeding. The nurse also assesses the patient for increased tenderness and rigidity of the abdomen. If these signs and symptoms occur, they are reported to the surgeon. The patient and family are instructed to report any change in the color of stools, because this may indicate complications. GI symptoms, although not common, may occur with manipulation of the intestines during surgery.

After laparoscopic cholecystectomy, the nurse assesses the patient for anorexia, vomiting, pain, abdominal distension, and temperature elevation. These may indicate infection or disruption of the GI tract and should be reported to the surgeon promptly. Because the patient is discharged soon after laparoscopic surgery, the patient and family are instructed verbally and in writing about the importance of reporting these symptoms promptly. Nurses should consider implementing the teach-back method when educating patients and families to ensure that they are able to describe what they have been taught in their own words or perform a task as instructed (see [Chapter 3](#) for discussion of the teach-back method).

PROMOTING HOME, COMMUNITY-BASED CARE, AND TRANSITIONAL CARE



Educating Patients About Self-Care. The nurse educates the patient about the medications that are prescribed (vitamins, anticholinergic and antispasmodic agents) and their actions. The nurse also informs the patient and family about symptoms that should be reported to the primary provider, including jaundice, dark urine, pale-colored stools, pruritus, and signs of inflammation and infection such as pain or fever.

Some patients report one to three bowel movements a day, which is a result of a continual trickle of bile through the choledochoduodenal junction after cholecystectomy. Usually, such frequency diminishes over a period of a few weeks to several months.

Chart 44-2



PATIENT EDUCATION

Managing Self-Care After Laparoscopic Cholecystectomy

The nurse instructs the patient about pain management, activity and exercise, wound care, nutrition, and follow-up care as described below.

Managing Pain

- You may experience pain or discomfort from the gas used to inflate your abdominal area during surgery. Sitting upright in bed or a chair, walking, or using a heating pad may ease the discomfort.
- Take analgesic medications as needed and as prescribed. Report to your surgeon if pain is unrelieved even with analgesic use.

Resuming Activity

- Begin light exercise (walking) immediately.
- Take a shower or bath after 1 or 2 days.
- Drive a car after 3 or 4 days.
- Avoid lifting objects exceeding 5 lb after surgery, usually for 1 week.
- Resume sexual activity when desired.

Caring for the Wound

- Check puncture site daily for signs of infection.
- Wash puncture site with mild soap and water.
- Allow special adhesive strips on the puncture site to fall off. Do not pull them off.

Resuming Eating

- Resume your normal diet.
- If you had fat intolerance before surgery, gradually add fat back into your diet in small increments.

Managing Follow-Up Care

- Make an appointment with your surgeon for 7 to 10 days after discharge.
- Call your surgeon if you experience any signs or symptoms of infection at or around the puncture site: redness, tenderness, swelling, heat, or drainage.
- Call your surgeon if you experience a fever of 37.7°C (100°F) or more for 2 consecutive days.
- Call your surgeon if you develop nausea, vomiting, or abdominal pain.

If a patient is discharged from the hospital with a drainage tube still in place, the patient and family need education about its management. The nurse educates them in proper care of the drainage tube and the importance of reporting promptly any changes in the amount or characteristics of drainage. Assistance in securing the appropriate dressings reduces the patient's anxiety about going home with the drain or tube still in place. [Chart 44-2](#) provides additional details about patient education for managing self-care after laparoscopic cholecystectomy.

Continuing and Transitional Care. With sufficient support at home, most patients recover quickly from a cholecystectomy. However, some patients may require a referral for transitional or home care. The hospital nurse can help ease the unpredictability of the postoperative and postdischarge experience for patients by providing relevant patient education, prompt pain relief, and an attentive approach to the nursing care (Rothrock, 2019). During home visits, the nurse assesses the patient's physical status, especially wound healing, and progress toward recovery. Assessing the patient for adequacy of pain relief and pulmonary exercises is also important. If the patient has a drainage system in place, the nurse assesses it for patency and appropriate management by the patient and family. Assessing for signs of infection and educating the patient about the signs and symptoms of infection are also important nursing interventions. The patient's understanding of the therapeutic regimen (medications, gradual return to normal activities) is assessed, and previous education is reinforced. The nurse emphasizes the importance of keeping follow-up appointments and reminds the patient and family of the importance of participating in health promotion activities and recommended health screening.

Evaluation

Expected patient outcomes may include:

1. Reports decrease in pain
 - a. Splints abdominal incision to decrease pain
 - b. Avoids foods that cause pain
 - c. Uses postoperative analgesia as prescribed
2. Demonstrates appropriate respiratory function
 - a. Achieves full respiratory excursion, with deep inspiration and expiration
 - b. Coughs effectively, using pillow to splint abdominal incision
 - c. Uses postoperative analgesia as prescribed
 - d. Exercises as prescribed (e.g., turns, ambulates)
3. Exhibits normal skin integrity around biliary drainage site (if applicable)

- a. Is free of fever; abdominal pain; change in vital signs; and presence of bile, foul-smelling drainage, or pus around drainage tube
 - b. Demonstrates correct management of drainage tube (if applicable)
 - c. Identifies signs and symptoms of biliary obstruction to be noted and reported
 - d. Has serum bilirubin level within normal range
4. Obtains relief from dietary intolerance
- a. Maintains adequate dietary intake and avoids foods that cause GI symptoms
 - b. Reports decreased or absent nausea, vomiting, diarrhea, flatulence, and abdominal discomfort
5. Absence of complications
- a. Has normal vital signs (blood pressure, pulse, respiratory rate and pattern, and temperature)
 - b. Reports absence of bleeding from GI tract and from biliary drainage tube or catheter (if present) and no evidence of bleeding in stool
 - c. Reports return of appetite and no evidence of vomiting, abdominal distention, or pain
 - d. Lists symptoms that should be reported to surgeon promptly and demonstrates an understanding of self-care, including wound care

DISORDERS OF THE PANCREAS

Pancreatitis (inflammation of the pancreas) is a serious disorder. The most basic classification system used to describe or categorize the various stages and forms of pancreatitis divides the disorder into acute and chronic forms. Acute pancreatitis can be a medical emergency associated with a high risk of life-threatening complications and mortality, whereas chronic pancreatitis often goes undetected because classic clinical and diagnostic findings are not always present in the early stages of the disease (Feldman et al., 2016; Papadakis & McPhee, 2020; Srinivasan & Friedman, 2018). By the time symptoms occur in chronic pancreatitis, approximately 90% of normal acinar cell function (exocrine function) has been lost (Feldman et al., 2016; Goldman & Shaffer, 2019; Papadakis & McPhee, 2020; Srinivasan & Friedman, 2018). Acute pancreatitis does not usually lead to chronic pancreatitis unless complications develop. However, chronic pancreatitis can be characterized by acute episodes.

Although the mechanisms causing pancreatic inflammation are unknown, pancreatitis is commonly described as autodigestion of the pancreas. It is believed that the pancreatic duct becomes temporarily obstructed, accompanied by hypersecretion of the exocrine enzymes of the pancreas.

These enzymes enter the bile duct, where they are activated and, together with bile, back up (reflux) into the pancreatic duct, causing pancreatitis.

Acute Pancreatitis



Approximately 200,000 cases of acute pancreatitis occur in the United States each year, of which 80% are the result of cholelithiasis or sustained alcohol abuse (Faghah, Fan, & Singh, 2019; Olson, Perelman, & Birk, 2019). Acute pancreatitis ranges from a mild, self-limited disorder to a severe, rapidly fatal disease that does not respond to any treatment. These two main types of acute pancreatitis (mild and severe) are classified as interstitial edematous pancreatitis and necrotizing pancreatitis, respectively. Interstitial pancreatitis affects the majority of patients. It is characterized by a lack of pancreatic or peripancreatic parenchymal necrosis with diffuse enlargement of the gland due to inflammatory edema (Faghah et al., 2019; Olson et al., 2019). The edema and inflammation in interstitial pancreatitis is confined to the pancreas itself. Minimal organ dysfunction is present, and return to normal function usually occurs within 6 months. Although this is considered the milder form of pancreatitis, the patient is acutely ill and at risk for hypovolemic shock, fluid and electrolyte disturbances, and sepsis.

In the more severe form, necrotizing pancreatitis, there is tissue necrosis in either the pancreatic parenchyma or in the tissue surrounding the gland. This type can be sterile or infected; if the parenchyma is involved, this is a marker for more severe disease (Brunicardi, 2019; Faghah, et al., 2019; Olson et al., 2019). A more widespread and complete enzymatic digestion of the gland characterizes necrotizing pancreatitis. Enzymes damage the local blood vessels, and bleeding and thrombosis can occur. The tissue may become necrotic, with damage extending into the retroperitoneal tissues. Local complications include pancreatic cysts or abscesses and acute fluid collections in or near the pancreas. Patients who develop systemic complications with organ failure, such as pulmonary insufficiency with hypoxia, shock, kidney disease, and GI bleeding, are also characterized as having severe acute pancreatitis.



Gerontologic Considerations

Acute pancreatitis affects people of all ages, but the mortality rate associated with acute pancreatitis increases with age (Brunicardi, 2019; Faghah et al., 2019; Olson et al., 2019). In addition, the pattern of complications changes with age. Younger patients tend to develop local complications; the incidence

of multiple organ dysfunction syndrome (MODS) increases with age, possibly as a result of progressive decreases in physiologic function of major organs with increasing age. Close monitoring of major organ function (i.e., lungs, kidneys) is essential, and aggressive treatment is necessary to reduce mortality from acute pancreatitis in the older adult patient.

Pathophysiology

Self-digestion of the pancreas by its own proteolytic enzymes, principally trypsin, causes acute pancreatitis. These patients usually have had undiagnosed chronic pancreatitis before their first episode of acute pancreatitis. Gallstones enter the CBD and lodge at the ampulla of Vater, obstructing the flow of pancreatic juice or causing a reflux of bile from the CBD into the pancreatic duct, thus activating the powerful enzymes within the pancreas. Normally, these remain in an inactive form until the pancreatic secretions reach the lumen of the duodenum (Brunicardi, 2019; Faghah et al., 2019; Norris, 2019; Olson et al., 2019). Activation of the enzymes can lead to vasodilation, increased vascular permeability, necrosis, erosion, and hemorrhage (Brunicardi, 2019; Faghah et al., 2019; Norris, 2019; Olson et al., 2019; Townsend et al., 2016).

Other less common causes of pancreatitis include bacterial or viral infection, with pancreatitis occasionally developing as a complication of mumps viral infection. Spasm and edema of the ampulla of Vater, caused by duodenitis, can probably produce pancreatitis. Blunt abdominal trauma, peptic ulcer disease, ischemic vascular disease, hyperlipidemia, hypercalcemia, and the use of corticosteroids, thiazide diuretics, oral contraceptives, and other medications have also been associated with an increased incidence of pancreatitis. Acute pancreatitis may develop after surgery on or near the pancreas or after instrumentation of the pancreatic duct. In addition to alcohol consumption, use of tobacco products is a risk factor for the development of acute and chronic pancreatitis (Aune, Yahya, Norat, et al., 2019). Acute idiopathic pancreatitis accounts for up to 10% of the cases of acute pancreatitis. Some postulate that these cases may be related to occult microlithiasis (small stones in the bile) (Goodchild, Chouhan, & Johnson, 2019; Olson et al., 2019; Townsend et al., 2016). In addition, there is a small incidence of hereditary pancreatitis.

The mortality rate of patients with acute pancreatitis is 2% to 10% because of shock, anoxia, hypotension, or fluid and electrolyte imbalances. This mortality rate may also be related to the 10% to 30% of patients with severe acute disease characterized by pancreatic and peripancreatic necrosis (Goodchild et al., 2019; Olson et al., 2019; Townsend et al., 2016). Pancreatitis may result in complete recovery, may recur without permanent damage, or may progress to chronic pancreatitis. The patient who is admitted to the

hospital with a diagnosis of pancreatitis is acutely ill and needs expert nursing and medical care.

Clinical Manifestations

Severe abdominal pain is the major symptom of pancreatitis that causes the patient to seek medical care. Abdominal pain and tenderness and back pain result from irritation and edema of the inflamed pancreas. Increased tension on the pancreatic capsule and obstruction of the pancreatic ducts also contribute to the pain. Typically, the pain occurs in the midepigastrium. Pain is frequently acute in onset, occurring 24 to 48 hours after a very heavy meal or alcohol ingestion, and it may be diffuse and difficult to localize. It is generally more severe after meals and is unrelieved by antacids. Pain may be accompanied by abdominal distention; a poorly defined, palpable abdominal mass; decreased peristalsis; and vomiting that fails to relieve the pain or nausea.

The patient appears acutely ill. Abdominal guarding is present. A rigid or boardlike abdomen may develop, usually indicating peritonitis (Goodchild et al., 2019; Olson et al., 2019). Ecchymosis (bruising) in the flank or around the umbilicus may indicate severe pancreatitis. Nausea and vomiting are common in acute pancreatitis. The emesis is usually gastric in origin but may also be bile stained. Fever, jaundice, mental confusion, and agitation may also occur.

Hypotension is typical and reflects hypovolemia and shock caused by the loss of large amounts of protein-rich fluid into the tissues and peritoneal cavity. In addition to hypotension, the patient may develop tachycardia; cyanosis; and cold, clammy skin. Acute kidney injury is common.

Respiratory distress and hypoxia are common, and the patient may develop diffuse pulmonary infiltrates, dyspnea, tachypnea, and abnormal blood gas values. Myocardial depression, hypocalcemia, hyperglycemia, and disseminated intravascular coagulation may also occur with acute pancreatitis.

Assessment and Diagnostic Findings

The diagnosis of acute pancreatitis is based on the fulfillment of two out of the three following criteria: a history of upper abdominal pain, biochemical changes with serum amylase or lipase levels greater than three times the upper limit of normal, or typical findings on imaging (CT, magnetic resonance imaging [MRI] or ultrasonography). The presence of known risk factors is also helpful for diagnostic purposes (Feldman et al., 2016; Goodchild et al., 2019; Olson et al., 2019; Zorniak, Beyer, & Mayerle, 2019). In most cases, serum amylase and lipase levels are elevated within 24 hours of the onset of the symptoms. Serum amylase usually returns to normal within 48 to 72 hours, but serum lipase levels may remain elevated for a longer period, often days longer than amylase. Urinary amylase levels also become elevated and remain

elevated longer than serum amylase levels. The white blood cell count is usually elevated; hypocalcemia is present in many patients and correlates well with the severity of pancreatitis. Transient hyperglycemia and glucosuria and elevated serum bilirubin levels occur in some patients with acute pancreatitis.

X-ray studies of the abdomen and chest may be obtained to differentiate pancreatitis from other disorders that can cause similar symptoms and to detect pleural effusions. Ultrasound studies, contrast-enhanced CT scans, and MRI scans are used to identify an increase in the diameter of the pancreas and to detect pancreatic cysts, abscesses, or pseudocysts.

Hematocrit and hemoglobin levels are used to monitor the patient for bleeding. Peritoneal fluid, obtained through paracentesis or peritoneal lavage, may contain increased levels of pancreatic enzymes. ERCP is rarely used in the diagnostic evaluation of acute pancreatitis, because the patient is acutely ill; however, it may be valuable in the treatment of gallstone pancreatitis.

The severity of acute pancreatitis is difficult to predict early in the course of the disease, but mortality can be predicted based on clinical and laboratory data (see [Chart 44-3](#)). According to the revised Atlanta Classification, there are three degrees of severity: (1) mild with the absence of organ failure and no local or systemic complications, (2) moderately severe with the presence of transient organ failure or local or systemic complications, and (3) severe acute pancreatitis characterized by persistent organ failure (>48 hours) (Banks, Bollen, Dervenis, et al., 2013; Zorniak et al., 2019). Several risk stratification systems aim to predict persistent organ failure and complications. The Acute Physiology and Chronic Health Evaluation II (APACHE II), Ranson Criteria for Pancreatitis Mortality, and Bedside Index of Severity in Acute Pancreatitis (BISAP) are scoring systems that assess clinical and biochemical factors to determine the severity of acute pancreatitis. Laboratory values such as C-reactive protein, procalcitonin, and blood urea nitrogen (BUN) may also carry some predictive value (Zorniak et al., 2019). Early prediction of the severity of acute pancreatitis is important for guiding early treatment, choosing the optimal level of care, and identifying patients who might benefit from transfer to a center that specializes in the care of this disease (Zorniak et al., 2019).

Chart 44-3

The Ranson Criteria for Pancreatic Mortality

Criteria on Admission to Hospital

Age >55 years
White blood cells (WBCs) >16,000 mm³
Serum glucose >200 mg/dL (>11.1 mmol/L)
Serum lactate dehydrogenase (LDH) >350 IU/L (>350 U/L)
Aspartate aminotransferase (AST) >250 IU/L

Criteria within 48 Hours of Hospital Admission

Fall in hematocrit >10% (>0.10)
Blood urea nitrogen (BUN) increase >5 mg/dL (>1.7 mmol/L)
Serum calcium <8 mg/dL (<2 mmol/L)
Base deficit >4 mEq/L (>4 mmol/L)
Fluid retention or sequestration >6 L
Partial pressure of oxygen (PO₂) <60 mm Hg
Two or fewer signs, 1% mortality; 3 or 4 signs, 15% mortality; 5 or 6 signs, 40% mortality; >6 signs, 100% mortality.

Note: The more risk factors a patient has, the greater the severity and likelihood of complications or death.

Adapted from Ranson, J. H., Rifkind, K. M., Roses, D. F., et al. (1974). Prognostic signs and the role of operative management in acute pancreatitis. *Surgery, Gynecology & Obstetrics*, 139(1), 69–81.

Medical Management

Management of acute pancreatitis is directed toward relieving symptoms and preventing or treating complications. All oral intake is withheld to inhibit stimulation of the pancreas and its secretion of enzymes. Ongoing research has shown positive outcomes with the use of enteral feedings. The current recommendation is that, whenever possible, the enteral route should be used to meet nutritional needs in patients with pancreatitis. This strategy also has been found to prevent infectious complications safely and cost-effectively (McClave, 2019; Mueller, 2017; Olson et al., 2019; Ramanathan & Aadam, 2019; Townsend et al., 2016). Enteral feedings should be started early in the course of acute pancreatitis (Goodchild et al., 2019; McClave, 2019; Mueller, 2017; Olson et al., 2019; Ramanathan & Aadam, 2019). Parenteral nutrition has a role in the nutritional support of patients with severe acute pancreatitis, particularly in those who are unable to tolerate enteral nutrition (Goodchild et al., 2019; Mueller, 2017; Olson et al., 2019). Nasogastric suction may be used to relieve nausea and vomiting and to decrease painful abdominal distention and paralytic ileus (Brunicardi, 2019). Research data do not support the routine use of nasogastric tubes to remove gastric secretions in an effort to limit

pancreatic secretion. Though current literature discourages the use of acid-suppressive therapy, this practice is common for hospitalized patients. Histamine-2 (H_2) antagonists such as cimetidine may be prescribed to decrease pancreatic activity by inhibiting secretion of gastric acid. Proton pump inhibitors such as pantoprazole may be used for patients who do not tolerate H_2 antagonists or for whom this therapy is ineffective (Barbateskovic, Marker, Granholm, et al., 2019; Kavitt, Lipowska, Anyane-Yeboa, et al., 2019).

Pain Management

Adequate administration of analgesia is essential during the course of acute pancreatitis to provide sufficient pain relief and to minimize restlessness, which may stimulate pancreatic secretion further. Pain relief may require parenteral opioids such as morphine, fentanyl, or hydromorphone (Cameron & Cameron, 2020; Goodchild et al., 2019; Olson et al., 2019). The recommendation for pain management is the use of opioids, with assessment for their effectiveness, and altering therapy if pain is not controlled or is increased (Faghah et al., 2019; Goodchild et al., 2019; Olson et al., 2019). There is some evidence that implementing the World Health Organization (WHO) analgesia ladder provides a pragmatic approach to pain management in patients with pancreatitis (Zorniak et al., 2019). This stepwise escalation from low potency to higher potency of nonsteroidal anti-inflammatory drugs (NSAIDs) alone or in combination with opioids may provide an effective method of pain management and lower the potential for opioid dependency (Zorniak et al., 2019). NSAIDs must be avoided or used in caution in patients at risk for bleeding. GI paralysis and ileus are common problems in early acute pancreatitis that can be potentiated and aggravated with the use of high-dose opioids (Zorniak et al., 2019). More research is needed to identify the best option for pain management in the patient with acute pancreatitis (Faghah et al., 2019). Until evidence-based recommendations are developed, guidelines for acute pain management in the perioperative setting should be followed (Rothrock, 2019). Antiemetic agents may be prescribed to prevent vomiting.



Intensive Care

Correction of fluid and blood loss and low albumin levels is necessary to maintain fluid volume and prevent acute kidney injury. The patient is usually acutely ill and is monitored in the intensive care unit, where hemodynamic monitoring and arterial blood gas monitoring are initiated. Antibiotic agents may be prescribed if infection is present. Prophylactic antibiotics are not recommended for patients with acute pancreatitis (Faghah et al., 2019; Goodchild, 2019; Olson et al., 2019). Insulin may be required if hyperglycemia occurs. Intensive insulin therapy (continuous infusion) in the critically ill

patient has undergone much study. The best practice recommendations, which have arisen from many investigations on this complex topic, include targeting a blood glucose level of 140 to 200 mg/dL if insulin therapy is required in critically ill medical and surgical patients. Additionally, clinicians are advised to avoid glucose targets <140 mg/dL because adverse effects are likely to increase with lower blood glucose targets (Horton, 2019).

Respiratory Care

Aggressive respiratory care is indicated because of the high risk of elevation of the diaphragm, pulmonary infiltrates and effusion, and atelectasis. Hypoxemia occurs in a significant number of patients with acute pancreatitis, even with normal x-ray findings. Respiratory care may range from close monitoring of arterial blood gases to the use of humidified oxygen to intubation and mechanical ventilation (see [Chapter 19](#) for further discussion).

Biliary Drainage

Placement of biliary drains (for external drainage) and stents (indwelling tubes) in the pancreatic duct through endoscopy has been performed to reestablish drainage of the pancreas. This has resulted in decreased pain.

Surgical Intervention

Although the acutely ill patient is at high risk for surgical complications, surgery may be performed to assist in the diagnosis of pancreatitis (diagnostic laparotomy); to establish pancreatic drainage; or to resect or débride an infected, necrotic pancreas. The patient who undergoes pancreatic surgery may have multiple drains in place postoperatively, as well as a surgical incision that is left open for irrigation and repacking every 2 to 3 days to remove necrotic debris (see [Fig. 44-6](#)).

Postacute Management

Oral feedings that are low in fat and protein are initiated gradually. Caffeine and alcohol are eliminated from the diet. If the episode of pancreatitis occurred during treatment with thiazide diuretics, corticosteroids, or oral contraceptives, these medications are discontinued. Follow-up may include ultrasound, x-ray studies, or ERCP to determine whether the pancreatitis is resolving and to assess for abscesses and pseudocysts. ERCP may also be used to identify the cause of acute pancreatitis if it is in question and for endoscopic sphincterotomy and removal of gallstones from the common bile duct.

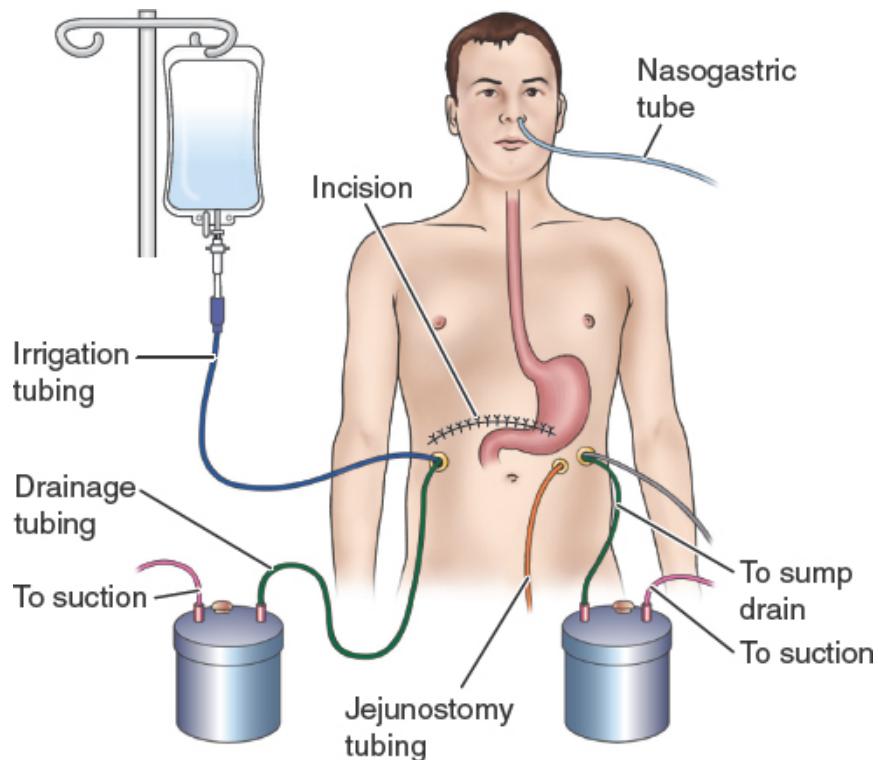


Figure 44-6 • Multiple sump tubes are used after pancreatic surgery. Triple-lumen tubes consist of ports that provide tubing for irrigation, air venting, and drainage.

Nursing Management

Relieving Pain and Discomfort

Because the pathologic process responsible for pain is autodigestion of the pancreas, the objectives of therapy are to relieve pain and decrease secretion of pancreatic enzymes. The pain of acute pancreatitis is often very severe, necessitating the liberal use of analgesic agents. The current recommendation for pain management in this population is parenteral opioids, including morphine, hydromorphone, or fentanyl via patient-controlled analgesia or bolus (Goodchild et al., 2019; Olson et al., 2019). In patients who are critically ill, a continuous infusion may be needed. Because most opioids stimulate spasm of the sphincter of Oddi to some degree, consensus has not been reached on the most effective agent. Ensuring patient comfort, regardless of the opioid prescribed, is the most essential aspect of care. The nurse frequently assesses the pain level and the effectiveness of the pharmacologic (and nonpharmacologic) interventions. Changes may be needed in the regimen for pain management based on the achievement of pain control. Pain assessment tools (see Chapter 9) are available for the nurse to ensure an accurate rating of pain. Nonpharmacologic interventions such as proper positioning, music,

distraction, and imagery may be effective in reducing pain when used along with medications.

Oral feedings are withheld to decrease the secretion of secretin. Parenteral fluids and electrolytes are prescribed to restore and maintain fluid balance. Nasogastric suction may be used to relieve nausea and vomiting or to treat abdominal distention and paralytic ileus. The nurse provides frequent oral hygiene and care to decrease discomfort from the nasogastric tube and relieve dryness of the mouth.

The patient who is acutely ill is maintained on bed rest to decrease the metabolic rate and reduce the secretion of pancreatic and gastric enzymes. If the patient experiences increasing severity of pain, the nurse reports this to the primary provider because the patient may be experiencing hemorrhage of the pancreas or the dose of analgesic medication may be inadequate.

The patient with acute pancreatitis is often confused or delirious because of severe pain, fluid and electrolyte disturbances, and hypoxia. Therefore, the nurse provides frequent and repeated but simple explanations about the need for withholding fluids, maintenance of gastric suction, and bed rest.

Improving Breathing Pattern

The nurse maintains the patient in a semi-Fowler position to decrease pressure on the diaphragm by a distended abdomen and to increase respiratory expansion. Frequent changes of position are necessary to prevent atelectasis and pooling of respiratory secretions. Pulmonary assessment, including monitoring of pulse oximetry or arterial blood gases, is essential to detect changes in respiratory status so that early treatment can be initiated. The nurse instructs the patient in techniques of coughing and deep breathing and in the use of incentive spirometry to improve respiratory function and assists the patient to perform these activities every hour.

Improving Nutritional Status

The nurse assesses the patient's nutritional status and notes factors that alter the patient's nutritional requirements (e.g., temperature elevation, surgery, drainage). Laboratory test results and daily weights are useful to monitor the nutritional status.

Enteral or parenteral nutrition may be prescribed. In addition to administering enteral or parenteral nutrition, the nurse monitors serum glucose levels every 4 to 6 hours. As the acute symptoms subside, oral feedings are gradually reintroduced. Between acute attacks, the patient receives a diet that is high in protein and low in fat (Goodchild et al., 2019; Olson et al., 2019). The patient should avoid heavy meals and alcoholic beverages.

Maintaining Skin Integrity

The patient is at risk for skin breakdown because of poor nutritional status, enforced bed rest, and restlessness, which may result in pressure injuries and breaks in tissue integrity. In addition, the patient who has undergone surgery may have multiple drains or an open surgical incision and is at risk for skin breakdown and infection. The wound, drainage sites, and skin are carefully assessed for signs of infection, inflammation, and breakdown. The nurse carries out wound care as prescribed and takes precautions to protect intact skin from contact with drainage. Consultation with a wound-ostomy-continence (WOC) nurse, a nurse specially educated in appropriate skin, wound, ostomy, and continence care is often helpful in identifying appropriate skin care devices and protocols. The patient must be turned every 2 hours; the use of specialty beds may be indicated to prevent skin breakdown.



Monitoring and Managing Potential Complications

Fluid and electrolyte disturbances are common complications because of nausea, vomiting, movement of fluid from the vascular compartment to the peritoneal cavity, diaphoresis, fever, and the use of gastric suction. The nurse assesses the patient's fluid and electrolyte status by noting skin turgor and moistness of mucous membranes. The nurse weighs the patient daily and carefully measures fluid intake and output, including urine output, nasogastric secretions, and diarrhea. In addition, it is important to assess for other factors that may affect fluid and electrolyte status, including increased body temperature and wound drainage. The patient is assessed for ascites, and abdominal girth is measured daily if ascites is suspected.

Fluids are administered IV and may be accompanied by infusion of blood or blood products to maintain the blood volume and to prevent or treat hypovolemic shock. Emergency medications must be readily available because of the risk of circulatory collapse and shock. The nurse promptly reports decreased blood pressure and reduced urine output, which indicate hypovolemia and shock or acute kidney injury. Low serum calcium and magnesium levels may occur and require prompt treatment.

Pancreatic necrosis is a major cause of morbidity and mortality in patients with acute pancreatitis because of resulting hemorrhage, septic shock, and MODS. The patient may undergo diagnostic procedures for confirmation of pancreatic necrosis. If the patient is found to have pancreatic necrosis with infection, this may require surgical, percutaneous or endoscopic débridement or insertion of multiple drains. Percutaneous or endoscopic catheter drainage is the first step of what is known as the "Step-Up Approach." Catheters are placed via the left or right retroperitoneal approach to drain the infection. Débridement, if required, may be performed through video-assisted retroperitoneal débridement. These interventions, coupled with appropriate, targeted antibiotic therapy, may be the only necessary treatment for some

patients (Paulino, Ramos, & Veloso Gomes, 2019; Rashid, Hussain, Jehanzeb, et al., 2019; Sion & Davis, 2019; Wolbrink, Kolwijk, Ten Oever, et al., 2019). These procedures are considered first-line approaches, with surgery reserved for patients for whom these interventions do not work. Prophylactic antibiotics are not indicated (Paulino et al., 2019; Sion & Davis, 2019; Wolbrink et al., 2019). The patient with pancreatic necrosis with or without infection is usually critically ill and requires expert medical and nursing management, including hemodynamic monitoring in the intensive care unit.

In addition to carefully monitoring vital signs and other signs and symptoms, the nurse is responsible for administering prescribed fluids, medications, and blood products; assisting with supportive management, such as the use of a ventilator; preventing additional complications; and providing physical and psychological care.

Shock and MODS may occur with acute pancreatitis. Hypovolemic shock may occur as a result of hypovolemia and sequestering of fluid in the peritoneal cavity. Hemorrhagic shock may occur with hemorrhagic pancreatitis. Septic shock may occur with bacterial infection of the pancreas. Cardiac dysfunction may occur as a result of fluid and electrolyte disturbances, acid-base imbalances, and release of toxic substances into the circulation.

The nurse closely monitors the patient for early signs of neurologic, cardiovascular, renal, and respiratory dysfunction and must be prepared to respond quickly to rapid changes in the patient's status, treatments, and therapies. In addition, it is important to inform the family about the patient's status and progress and to allow them to spend time with the patient. Management of shock and MODS is discussed in detail in [Chapter 11](#).

Promoting Home, Community-Based, and Transitional Care



Educating Patients About Self-Care

After an episode of acute pancreatitis, the patient is often still weak and debilitated for weeks or months. A prolonged period may be needed to regain strength and return to the previous level of activity. Because of the severity of the acute illness, the patient may not recall education given during the acute phase. Patient education often needs to be repeated and reinforced. The nurse educates the patient about the factors implicated in the onset of acute pancreatitis and about the need to avoid high-fat foods, heavy meals, and alcohol. The patient and family should receive verbal and written instructions about signs and symptoms of acute pancreatitis and possible complications that should be reported promptly to the primary provider.

If acute pancreatitis is a result of biliary tract disease, such as gallstones and gallbladder disease, additional explanations are needed about required dietary

modifications. If the pancreatitis is a result of alcohol abuse, the nurse reinforces the need to avoid all alcohol.

Continuing and Transitional Care

A referral for home, community-based or transitional care is often indicated. This enables the nurse to assess the patient's physical and psychological status and adherence to the therapeutic regimen. The nurse also assesses the home situation and reinforces instructions about fluid and nutrition intake and avoidance of alcohol. Nurses should consider implementing the teach-back method when educating patients and families about this vital information (see [Chapter 3](#) for a discussion of the teach-back method). After the acute attack has subsided, some patients may be inclined to return to their previous drinking habits. The nurse provides specific information about resources and support groups that may be of assistance in avoiding alcohol in the future. Referral to Alcoholics Anonymous as appropriate or other support groups is essential. See the accompanying plan of nursing care in [Chart 44-4](#) for care of the patient with acute pancreatitis.

Chronic Pancreatitis

Chronic pancreatitis is an inflammatory disorder characterized by progressive destruction of the pancreas. As cells are replaced by fibrous tissue with repeated attacks of pancreatitis, pressure within the pancreas increases. The result is obstruction of the pancreatic and common bile ducts and the duodenum. In addition, there is atrophy of the epithelium of the ducts, inflammation, and destruction of the secreting cells of the pancreas.

Alcohol consumption in Western societies and malnutrition worldwide are the major causes of chronic pancreatitis. Patients diagnosed with chronic pancreatitis due to alcohol typically present between the ages of 40 and 60 years (Singh, 2019). Frequently, at that age, patients already report a long history of alcohol abuse. Excessive and prolonged consumption of alcohol accounts for approximately 70% to 80% of all cases of chronic pancreatitis (Papadakis & McPhee, 2020; Srinivasan & Friedman, 2018; Townsend et al., 2016). The incidence of pancreatitis is 50 times greater in people with alcoholism than in those who do not abuse alcohol. Long-term alcohol consumption causes hypersecretion of protein in pancreatic secretions, resulting in protein plugs and calculi within the pancreatic ducts. Alcohol also has a direct toxic effect on the cells of the pancreas. Damage to these cells is more likely to occur and to be more severe in patients whose diets are poor in protein content and either very high or very low in fat.

Smoking is another factor in the development of chronic pancreatitis. Because smoking and alcohol are often associated, it is difficult to separate the

effects of these two factors (Aune et al., 2019; Papadakis & McPhee, 2020; Singh, 2019).

Chart 44-4



PLAN OF NURSING CARE

Care of the Patient with Acute Pancreatitis

NURSING DIAGNOSIS: Acute pain associated with edema, distention of the pancreas, peritoneal irritation, and excess stimulation of pancreatic secretions

GOAL: Relief of pain

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Using a pain scale, assess pain level at baseline, before and after administration of analgesic medications. 2. Administer morphine, fentanyl, or hydromorphone frequently, as prescribed, to achieve level of pain acceptable to patient. Depending on pain severity, nonsteroidal anti-inflammatory drugs (NSAIDs) may be considered alone or in combination with opioids. 3. Maintain the patient NPO (nothing by mouth) as prescribed. 4. Maintain the patient on bed rest. 5. Maintain continuous nasogastric drainage if paralytic ileus or nausea and vomiting, abdominal distention are present. <ol style="list-style-type: none"> a. Measure gastric secretions at specified intervals. b. Observe and record color and viscosity 	<ol style="list-style-type: none"> 1. Baseline assessment and control of pain are important because restlessness increases the body's metabolism, which stimulates the secretion of pancreatic and gastric enzymes. 2. Fentanyl and hydromorphone act by depressing the central nervous system and thereby increasing the patient's pain threshold. 3. Pancreatic secretion is increased by food and fluid intake. 4. Bed rest decreases body metabolism and thus reduces pancreatic and gastric secretions. 5. Nasogastric suction relieves nausea, vomiting, and abdominal distention. Decompression of the intestines (if intestinal intubation is used) also assists in relieving 	<ul style="list-style-type: none"> • Patient rates pain using pain scale • Reports relief of pain, discomfort, and abdominal cramping • Moves and turns without increasing pain and discomfort • Rests comfortably and sleeps for increasing periods • Reports increased feelings of well-being and security with the health care team

of gastric secretions.	respiratory distress.
c. Ensure that the nasogastric tube is patent to permit free drainage.	
6. Report unrelieved pain or increasing intensity of pain.	6. Pain may increase pancreatic enzymes and may also indicate pancreatic hemorrhage.
7. Assist patient to assume positions of comfort; turn and reposition every 2 hours.	7. Frequent turning relieves pressure and assists in preventing pulmonary and vascular complications.
8. Use nonpharmacologic interventions for relieving pain (e.g., relaxation, focused breathing, diversion).	8. The use of nonpharmacologic methods will enhance the effects of analgesic medications.
9. Listen to patient's expression of pain experience.	9. Demonstration of caring can help to decrease anxiety.

NURSING DIAGNOSIS: Discomfort associated with nasogastric tube

GOAL: Relief of discomfort associated with nasogastric intubation used to treat ileus, vomiting, and distention

Nursing Interventions	Rationale	Expected Outcomes
1. Use water-soluble lubricant around external nares.	1. Prevents irritation of nares.	• Exhibits intact skin and tissue of nares at site of nasogastric tube insertion
2. Turn patient at intervals; avoid pressure or tension on nasogastric tube.	2. Relieves pressure of tube on esophageal and gastric mucosa.	• Reports no pain or irritation of nares or oropharynx
3. Provide oral hygiene and gargling solutions without alcohol.	3. Relieves dryness and irritation of oropharynx.	• Exhibits moist, clean mucous membranes of
4. Explain rationale for the use of nasogastric drainage.	4. Assists patient to cooperate with the drainage,	

	nasogastric tube, and suction.	mouth and nasopharynx
		<ul style="list-style-type: none"> • States that thirst is relieved by oral hygiene • Identifies rationale for nasogastric tube and suction
NURSING DIAGNOSIS: Impaired nutritional status associated with inadequate dietary intake, impaired pancreatic secretions, increased nutritional needs secondary to acute illness, and increased body temperature		
GOAL: Improvement in nutritional status		
Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Assess current nutritional status and increased metabolic requirements. 2. Monitor serum glucose levels and administer insulin as prescribed. 3. Administer IV fluid and electrolytes, enteral or parenteral nutrition as prescribed. 4. Provide high-carbohydrate, low-protein, and low-fat diet when tolerated. 5. Instruct patient to eliminate alcohol, and refer to Alcoholics Anonymous, if indicated. 6. Counsel patient to avoid excessive use of 	<ol style="list-style-type: none"> 1. Alteration in pancreatic secretions interferes with normal digestive processes. Acute illness, infection, and fever increase metabolic needs. 2. Impairment of endocrine function of the pancreas leads to increased serum glucose levels. 3. Parenteral administration of fluids and electrolytes, and enteral or parenteral nutrients are essential to provide fluids, calories, electrolytes, and 	<ul style="list-style-type: none"> • Maintains normal body weight • Demonstrates no additional weight loss • Maintains normal serum glucose levels • Reports decreasing episodes of vomiting and diarrhea • Reports return of normal stool characteristics and bowel pattern • Consumes foods high in carbohydrates, low in fat and protein

<p>coffee and spicy foods.</p> <p>7. Monitor daily weights.</p>	<p>nutrients when oral intake is prohibited.</p> <p>4. These foods increase caloric intake without stimulating pancreatic secretions beyond the ability of the pancreas to respond.</p> <p>5. Alcohol intake produces further damage to pancreas and precipitates attacks of acute pancreatitis.</p> <p>6. Coffee and spicy foods increase pancreatic and gastric secretions.</p> <p>7. This provides a baseline and a means to measure weight gain or weight loss.</p>	<ul style="list-style-type: none"> • Explains rationale for high-carbohydrate, low-fat, low-protein diet • Eliminates alcohol from diet • Explains rationale for limiting coffee intake and avoiding spicy foods • Participates in Alcoholics Anonymous as appropriate or other counseling approach • Returns to and maintains desirable weight
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NURSING DIAGNOSIS: Impaired breathing associated with splinting from severe pain, pulmonary infiltrates, pleural effusion, and atelectasis

GOAL: Improvement in respiratory function

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Assess respiratory status (rate, pattern, breath sounds), pulse oximetry, and arterial blood gases. 2. Maintain semi-Fowler position. 3. Instruct and encourage patient to take deep breaths and to cough every hour. Assist patient to turn and change position every 2 hours. 4. Reduce the excessive metabolism of the body. <ul style="list-style-type: none"> a. Administer antibiotics as prescribed. b. Administer nasal oxygen as required for hypoxia. c. Use a hypothermia blanket if necessary. 	<ol style="list-style-type: none"> 1. Acute pancreatitis produces retroperitoneal edema, elevation of the diaphragm, pleural effusion, and inadequate lung ventilation. Intra-abdominal infection and labored breathing increase the body's metabolic demands, which further decreases pulmonary reserve and leads to respiratory failure. 2. Decreases pressure on diaphragm and allows greater lung expansion. 3. Taking deep breaths and coughing will clear the airways and reduce atelectasis. 4. Changing position frequently assists aeration and drainage of all lobes of the lungs. 5. Pancreatitis produces a severe peritoneal and retroperitoneal reaction that causes fever, tachycardia, and accelerated respirations. Supporting the patient with oxygen therapy decreases the workload of the respiratory system and the tissue utilization of oxygen. Reduction of fever and pulse rate 	<ul style="list-style-type: none"> • Demonstrates normal respiratory rate and pattern and full lung expansion • Demonstrates normal breath sounds and absence of adventitious breath sounds • Demonstrates normal arterial blood gases and pulse oximetry • Maintains semi-Fowler position when in bed • Changes position in bed frequently • Coughs and takes deep breaths at least every hour • Demonstrates normal body temperature • Exhibits no signs or symptoms of respiratory

	decreases the metabolic demands on the body.	infection or impairment
		• Is alert and responsive to environment

COLLABORATIVE PROBLEM: Fluid and electrolyte disturbances, hypovolemia, shock

GOAL: Improvement in fluid and electrolyte status, prevention of hypovolemia and shock

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Assess fluid and electrolyte status (skin turgor, mucous membranes, urine output, vital signs, hemodynamic parameters). 2. Assess sources of fluid and electrolyte loss (vomiting, diarrhea, nasogastric drainage, excessive diaphoresis). 3. Combat shock if present. <ol style="list-style-type: none"> a. Administer corticosteroids as prescribed if patient does not respond to conventional treatment. b. Evaluate the amount of urinary output. Attempt to 	<ol style="list-style-type: none"> 1. The amount and type of fluid and electrolyte replacement are determined by the status of the blood pressure, the laboratory evaluations of serum electrolyte and blood urea nitrogen levels, the urinary volume, and the assessment of the patient's condition. 2. Electrolyte losses occur from nasogastric suctioning, severe diaphoresis, and emesis, and as a result of the patient being in a fasting state. 3. Extensive acute pancreatitis may cause peripheral vascular collapse and shock. Blood and plasma may be lost into the abdominal cavity; therefore, there is a decreased blood and plasma volume. The toxins from the bacteria of a necrotic pancreas may cause shock. 	<ul style="list-style-type: none"> • Exhibits moist mucous membranes and normal skin turgor • Exhibits normal blood pressure without evidence of orthostatic hypotension • Excretes adequate urine volume • Exhibits normal, not excessive, thirst • Maintains normal pulse and respiratory rate • Remains alert and responsive • Exhibits normal arterial pressures and blood gases

<p>maintain this at ≥ 0.5 mL/kg/hr and ≥ 400 mL/day.</p> <p>4. Administer blood products, fluids, and electrolytes (sodium, potassium, chloride) as prescribed.</p> <p>5. Administer plasma and blood products as prescribed.</p> <p>6. Keep a supply of IV calcium gluconate or calcium chloride readily available.</p> <p>7. Assess abdomen for ascites formation.</p> <ol style="list-style-type: none"> Measure abdominal girth daily. Weigh patient daily. Assess abdomen for ascites (see Chapter 43, Fig. 43-6). <p>8. Monitor for manifestations of multiple organ dysfunction syndrome: neurologic, cardiovascular, renal, and respiratory dysfunction.</p>	<p>4. Patients with hemorrhagic pancreatitis lose large amounts of blood and plasma, which decreases effective circulation and blood volume.</p> <p>5. Replacement with blood, plasma, or albumin assists in ensuring effective circulating blood volume.</p> <p>6. Calcium may be prescribed to prevent or treat tetany, which may result from calcium losses into retroperitoneal (peripancreatic) exudate.</p> <p>7. During acute pancreatitis, plasma may be lost into the abdominal cavity, which diminishes the blood volume.</p> <p>8. All body systems may fail if pancreatitis is severe and treatment is ineffective.</p>	<ul style="list-style-type: none"> • Exhibits normal electrolyte levels • Exhibits no signs or symptoms of calcium deficit (e.g., tetany, carpopedal spasm) • Exhibits no additional losses of fluids and electrolytes through vomiting, diarrhea, or diaphoresis • Demonstrates no change in weight • Demonstrates no increase in abdominal girth • Demonstrates no fluid wave on palpation of the abdomen • Demonstrates stable organ function without manifestations of failure
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Clinical Manifestations

Chronic pancreatitis is characterized by recurring attacks of severe upper abdominal and back pain, accompanied by vomiting. Attacks are often so painful that opioids, even in large doses, do not provide relief. The risk of opioid dependence is increased in pancreatitis because of the chronic nature and severity of the pain. As the disease progresses, recurring attacks of pain are more severe, more frequent, and of longer duration. Some patients experience continuous severe pain, and others have dull, nagging constant pain. Periods of well-being sometimes follow the episodes of pain (Singh, 2019). In some patients, chronic pancreatitis is painless. The natural history of abdominal pain (character, timing, severity) is variable, and many studies have documented a decrease in pain (“burnout”) over time in a majority of patients (Singh, 2019).

Weight loss is a major problem in chronic pancreatitis. More than 80% of patients experience significant weight loss, which is usually caused by decreased dietary intake secondary to anorexia or fear that eating will precipitate another attack (Kellerman & Rakel, 2018; Singh, 2019). Malabsorption occurs late in the disease, when as little as 10% of pancreatic function remains (Kellerman & Rakel, 2018; Singh, 2019). As a result, digestion, especially of proteins and fats, is impaired. The stools become frequent, frothy, and foul smelling because of impaired fat digestion, which results in stools with a high fat content referred to as **steatorrhea**. As the disease progresses, calcification of the gland may occur, and calcium stones may form within the ducts.

Assessment and Diagnostic Findings

A CT scan is the initial diagnostic test that should be performed for patients in whom there is a suspicion of chronic pancreatitis. The presence of either calcifications, pancreatic ductal changes, or both of these findings can substantiate the diagnosis of chronic pancreatitis (Singh, 2019). MRCP is considered for further evaluation, should the CT findings be equivocal in patients with known risk factors. Transabdominal ultrasonography is frequently used as a screening method for patients with abdominal symptoms. EUS and laparoscopic ultrasound are capable of detecting very small abnormalities in the pancreas. EUS is frequently used early in the evaluation of patients with pancreatic disease, and MRCP is increasingly being used in select patients who are candidates for the most invasive imaging method, ERCP. EUS evaluates for parenchymal and ductal abnormalities that are useful for diagnosis and staging (Singh, 2019). The staging of disease is important in the care of patients, and a combination of imaging methods is usually used to confirm the stage (Brunicardi, 2019; Singh, 2019). ERCP provides details

about the anatomy of the pancreas and the pancreatic and biliary ducts. It is also helpful in obtaining tissue for analysis and differentiating pancreatitis from other conditions, such as carcinoma (Singh, 2019).

A glucose tolerance test evaluates pancreatic islet cell function and provides necessary information for making decisions about surgical resection of the pancreas. An abnormal glucose tolerance test may indicate the presence of diabetes associated with pancreatitis. Acute exacerbations of chronic pancreatitis may result in increased serum amylase levels. Steatorrhea is best confirmed by laboratory analysis of fecal fat content (Kellerman & Rakel, 2018).

Medical Management

The management of chronic pancreatitis depends on its probable cause in each patient. Treatment is directed toward preventing and managing acute attacks, relieving pain and discomfort, and managing exocrine and endocrine insufficiency of pancreatitis (Singh, 2019; Papadakis & McPhee, 2020).

Nonsurgical Management

Nonsurgical approaches may be indicated for the patient who refuses surgery, is a poor surgical risk, or when the disease and symptoms do not warrant surgical intervention. Endoscopy to remove pancreatic duct stones, correct strictures with stenting, and drain cysts may be effective in select patients to manage pain and relieve obstruction via ERCP (Singh, 2019).

Management of abdominal pain and discomfort is similar to that of acute pancreatitis; however, the focus is usually on the use of nonopioid methods to manage pain and the implementation of the WHO's three-step ladder for the treatment of chronic pain. This involves initiating monotherapy and, if ineffective, instituting combination therapy with peripherally acting and centrally acting medications. Pain management in the early stages may respond to nonopioid analgesics, but if the pain becomes more constant or debilitating, introduction of opioid analgesics is appropriate. The abuse of opioids is possible in these patients, and may be more likely in patients with depression, higher pain intensity, and history of alcohol misuse or abuse (Singh, 2019). Adjunct means of pain modulation, such as use of antioxidants, antidepressants, and nonopioid agents as well as avoidance of smoking and alcohol, are recommended before starting opioids for pain control (Singh, 2019). Antioxidants assist in the relief of pain and in improving quality of life and are often given to patients with chronic pancreatitis (Jalal, Campbell, & Hopper, 2019; Singh, 2019). An EUS with guided placement of a celiac nerve block is a potential option for managing the chronic, unrelieved pain of this disease (Singh, 2019). Yoga and other mindfulness-based therapies may be effective nonpharmacologic methods for pain reduction and for relief of other

coexisting symptoms of chronic pancreatitis (Jalal et al., 2019). Persistent, unrelieved pain is often the most difficult aspect of management (Jalal et al., 2019; Singh, 2019). The primary provider, nurse, and dietitian emphasize to the patient and family the importance of avoiding alcohol and foods that have produced abdominal pain and discomfort in the past. The health care team stresses to the patient that no other treatment is likely to relieve pain if the patient continues to consume alcohol.

Diabetes resulting from dysfunction of the pancreatic islet cells is treated with diet, insulin, or oral antidiabetic agents. The hazard of severe hypoglycemia with alcohol consumption is stressed to the patient and family. Pancreatic enzyme replacement is indicated for the patient with malabsorption and steatorrhea.

Surgical Management

Chronic pancreatitis is not often managed by surgery. However, surgery may be indicated to relieve persistent abdominal pain and discomfort, restore drainage of pancreatic secretions, and reduce the frequency of acute attacks of pancreatitis and hospitalization (Brunicardi, 2019; Jalal et al., 2019; Singh, 2019). The type of surgery performed depends on the anatomic and functional abnormalities of the pancreas, including the location of disease within the pancreas, the presence of diabetes, exocrine insufficiency, biliary stenosis, and pseudocysts of the pancreas. Other considerations for surgery selection include the patient's likelihood for continued use of alcohol and the likelihood that the patient will be able to manage the endocrine or exocrine changes that are expected after surgery.

Pancreaticojejunostomy (also referred to as Roux-en-Y), with a side-to-side anastomosis or joining of the pancreatic duct to the jejunum, allows drainage of the pancreatic secretions into the jejunum. Pain relief occurs within 6 months in more than 85% of the patients who undergo this procedure, but pain returns in a substantial number of patients as the disease progresses (Brunicardi, 2019; Cameron & Cameron, 2020; Yeo, 2019).

Other surgical procedures may be performed for different degrees and types of underlying disorders. These procedures include revision of the sphincter of the ampulla of Vater, internal drainage of a pancreatic cyst into the stomach (see later discussion), insertion of a stent, and wide resection or removal of the pancreas. A Whipple's resection (pancreaticoduodenectomy) can be carried out to relieve the pain of chronic pancreatitis (see later discussion under Tumors of the Head of the Pancreas). In an effort to provide permanent pain relief and avoid endocrine and exocrine insufficiency that ensue with major resections of the pancreas, some procedures combine limited resection of the head of the pancreas with a pancreaticojejunostomy. These procedures, known as the Beger or Frey operations, remove most of the head of the pancreas except for a

shell of pancreatic tissue posteriorly (Brunicardi, 2019; Cameron & Cameron, 2020; Yeo, 2019).

When chronic pancreatitis develops as a result of gallbladder disease, surgery is performed to explore the common duct and remove the stones; usually, the gallbladder is removed at the same time. In addition, an attempt is made to improve the drainage of the CBD and the pancreatic duct by dividing the sphincter of Oddi, a muscle that is located at the ampulla of Vater (this surgical procedure is known as a sphincterotomy). A T-tube usually is placed in the common bile duct, requiring a drainage system to collect the bile postoperatively. Nursing care after such surgery is similar to that indicated after other biliary tract surgery.

Approximately two thirds of all patients with chronic pancreatitis can be managed with endoscopic or laparoscopic intervention (Jalal et al., 2019; Singh, 2019). Endoscopic and laparoscopic procedures such as distal pancreatectomy, longitudinal decompression of the pancreatic duct, nerve denervation, and stenting have been performed in patients with jaundice or recurrent inflammation and are being refined. Minimally invasive procedures to treat chronic pancreatitis may prove to be successful adjuncts in the management of this complex disorder (Jalal et al., 2019; Singh, 2019).

Patients who undergo surgery for chronic pancreatitis may experience weight gain and improved nutritional status; this may result from reduction in pain associated with eating rather than from correction of malabsorption. However, morbidity and mortality after these surgical procedures are high because of the poor physical condition of the patient before surgery and the frequent presence of cirrhosis. Even after undergoing these surgical procedures, the patient is likely to continue to have pain and impaired digestion secondary to pancreatitis.

Pancreatic Cysts

As a result of the local necrosis that occurs because of acute pancreatitis, collections of fluid may form close to the pancreas. These fluid collections become walled off by fibrous tissue and are called *pancreatic pseudocysts*. Pseudocysts are amylase-rich fluid collections contained within a wall of fibrous granulation tissue and develop within 4 to 6 weeks after an episode of acute pancreatitis. They are a result of pancreatic necrosis, which produces a pancreatic ductal leak into pancreatic tissue weakened by extravasating enzymes (Feldman et al., 2016; Goldman & Schafer, 2019). Pseudocysts are distinguished from true cysts by the characteristics of the lining of the walls of these anomalies. The lining of pseudocysts consists of fibrous granulation tissue, whereas true cysts have epithelium-lined walls (Bansal, Gupta, Singh, et al., 2019; Feldman et al., 2016; Goldman & Schafer, 2019; Papadakis &

McPhee, 2020). Pseudocysts are the most common type of pancreatic “cyst.” Less common cysts occur as a result of congenital anomalies or secondary to chronic pancreatitis or trauma to the pancreas.

Diagnosis of pancreatic cysts and pseudocysts is made by ultrasound, CT scan, and ERCP. ERCP may be used to identify the anatomy of the pancreas and evaluate the patency of pancreatic drainage. Pancreatic pseudocysts may be of considerable size. When pancreatic pseudocysts enlarge, they impinge on and displace the adjacent stomach or the colon because of the location of pseudocysts behind the posterior peritoneum. Eventually, through pressure or secondary infection, they produce symptoms and require drainage.

Drainage into the GI tract or through the skin and abdominal wall may be established. In the latter instance, the drainage is likely to be profuse and destructive to tissue because of the enzyme contents. Hence, steps (including application of skin ointment) must be taken to protect the skin near the drainage site from excoriation. A suction apparatus may be used to continuously aspirate digestive secretions from the drainage tract so that skin contact with the digestive enzymes is avoided. Expert nursing attention is required to ensure that the suction tube does not become dislodged and suction is not interrupted. Consultation with a WOC nurse is indicated to identify appropriate strategies for maintaining drainage and protecting the skin.

Cancer of the Pancreas

Pancreatic cancer is the fourth leading cause of cancer death in men in the United States and the fifth leading cause of cancer death in women. It is very rare before the age of 45 years, and the majority of patients present in or beyond the sixth decade of life (Feldman et al., 2016; Papadakis & McPhee, 2020; Yeo, 2019). The incidence of pancreatic cancer increases with age, peaking in the seventh and eighth decades for both men and women (American Cancer Society [ACS], 2020). The frequency of pancreatic cancer has decreased slightly over the past 25 years among non-Caucasian men. There is a slight male preponderance; and in the United States, incidence is highest in African American males (ACS, 2020; Feldman et al., 2016). Exposure to industrial chemicals or toxins in the environment, and a diet high in fat, meat, or both are associated risk factors (ACS, 2020; Feldman et al., 2016; Papadakis & McPhee, 2020).

The risk of pancreatic cancer is greater in those with a history of increased pack years of cigarette smoking and in those with high alcohol intake. Diabetes, chronic pancreatitis, hereditary pancreatitis, and obesity are also associated with pancreatic cancer (Rawla, 2019; Yeo, 2019). The pancreas can also be the site of metastasis from other tumors.

Cancer may develop in the head, body, or tail of the pancreas; clinical manifestations vary depending on the site and whether functioning insulin-secreting pancreatic islet cells are involved. Approximately 70% of pancreatic cancers originate in the head of the pancreas and give rise to a distinctive clinical picture (Feldman et al., 2016; Rawla, 2019). Functioning islet cell tumors, whether benign (adenoma) or malignant (adenocarcinoma), are responsible for the syndrome of hyperinsulinism. The symptoms are typically nonspecific, and patients usually do not seek medical attention until late in the disease. Only about 7% of cases are diagnosed in early stages; 80% to 85% of patients have advanced, unresectable tumor when first detected. As a result, pancreatic carcinoma has only a 7% survival rate at 5 years regardless of the stage of disease at diagnosis or treatment (ACS, 2020; Rawla, 2019; Yeo, 2019).

Clinical Manifestations

Pain, jaundice, or both are present in more than 80% of patients and, along with weight loss, are considered classic signs of pancreatic carcinoma (Brunicardi, 2019; Feldman et al., 2016; Yeo, 2019). However, they often do not appear until the disease is far advanced. Other signs include rapid, profound, and progressive weight loss as well as vague upper or midabdominal pain or discomfort that is unrelated to any GI function and is often difficult to describe. Such discomfort radiates as a boring pain in the midback and is unrelated to posture or activity. It is often progressive and severe, requiring the use of opioids. It is often more severe at night and is accentuated when lying supine. Relief may be obtained by sitting up and leaning forward.

Malignant cells from pancreatic cancer are often shed into the peritoneal cavity, increasing the likelihood of metastasis. The formation of ascites is common. An important sign, if present, is the onset of symptoms of insulin deficiency: glucosuria, hyperglycemia, and abnormal glucose tolerance. Therefore, diabetes may be an early sign of carcinoma of the pancreas. Meals often aggravate epigastric pain, which usually occurs before the appearance of jaundice and pruritus.

Assessment and Diagnostic Findings

Spiral (helical) CT is more than 85% to 90% accurate in the diagnosis and staging of pancreatic cancer and currently is the most useful preoperative imaging technique. MRI/MRCP may also be used. EUS is useful in identifying small tumors and in performing fine-needle aspiration biopsy of the primary tumor or lymph nodes (Goldman & Schafer, 2019; Papadakis & McPhee, 2020). EUS can be superior to CT in localizing these small tumors, which can

produce dramatic symptoms despite their size (<1 cm) (Brunicardi, 2019). ERCP may also be used in the diagnosis of pancreatic carcinoma.

Cells obtained during ERCP are sent to the laboratory for analysis. GI x-ray findings may demonstrate deformities in adjacent organs caused by the impinging pancreatic mass.

A histologic diagnosis is not usually required in patients who are candidates for surgery. The tissue diagnosis is made at the time of the surgical procedure. Percutaneous fine-needle aspiration biopsy of the pancreas, which is used to diagnose pancreatic tumors, is also used to confirm the diagnosis in patients whose tumors are not resectable so that a palliative plan of care can be determined. This may eliminate the stress and postoperative pain of ineffective surgery. In this procedure, a needle is inserted through the anterior abdominal wall into the pancreatic mass, guided by CT, ultrasound, ERCP, or other imaging techniques. The aspirated material is examined for malignant cells. Although percutaneous biopsy is a valuable diagnostic tool, it has some potential drawbacks: a false-negative result if small tumors are missed and the risk of seeding of cancer cells along the needle track. Low-dose radiation to the site may be used before the biopsy to reduce this risk.

PTC is another procedure that may be performed to identify obstructions of the biliary tract by a pancreatic tumor. Several tumor markers (e.g., cancer antigen 19-9, carcinoembryonic antigen, DU-PAN-2) may be used in the diagnostic workup, but they are nonspecific for pancreatic carcinoma. These tumor markers are useful as indicators of disease progression.

Angiography, CT scans, and laparoscopy may be performed to determine whether the tumor can be removed surgically. Intraoperative ultrasonography has been used to determine whether there is metastatic disease to other organs.

Medical Management

If the tumor is resectable and localized (typically tumors in the head of the pancreas), the surgical procedure to remove it is usually extensive (see later discussion). However, total excision of the lesion often is not possible for two reasons: (1) extensive growth of tumor before diagnosis, and (2) probable widespread metastases (especially to the liver, lungs, and bones). More often, treatment is limited to palliative measures.

Although pancreatic tumors may be resistant to standard radiation therapy, the patient may be treated with radiation and chemotherapy (5-fluorouracil, leucovorin, and gemcitabine). Currently, gemcitabine is the standard of care for patients with metastatic pancreatic cancer and has been found to lengthen survival (ACS, 2020; Feldman et al., 2016). The targeted anticancer drug erlotinib has demonstrated a slight improvement in advanced pancreatic cancer survival when used in combination with gemcitabine (ACS, 2020; Brunicardi, 2019). Agents such as S-1 (an oral fluoropyrimidine), or the use of

nanoparticle albumin-bound paclitaxel with gemcitabine therapy, combined with radiation or surgery may also result in improved survival (ACS, 2020; Brunicardi, 2019; Kamisawa, Wood, Itoi, et al., 2016). Irinotecan, in combination with fluorouracil and leucovorin, was approved as treatment for metastatic pancreatic adenocarcinoma that has progressed following treatment with a gemcitabine-based therapy. 5-Fluorouracil or capecitabine, a similar but orally administered drug, are used as radiosensitizers during radiation therapy (Brunicardi, 2019). Single-agent gemcitabine may be used in patients who are not well enough to tolerate combination therapy (ACS, 2020; Brunicardi, 2019; O'Reilly, Fou, Hasler, et al., 2018).

If the patient undergoes surgery, intraoperative radiation therapy may be used to deliver a high dose of radiation to the tumor with minimal injury to other tissues; this may also be helpful in relief of pain. Interstitial implantation of radioactive sources has also been used, although the rate of complications is high. A large biliary stent inserted percutaneously or by endoscopy may be used to relieve jaundice (Brunicardi, 2019; O'Reilly et al., 2018).

Nursing Management

Pain management and attention to nutritional requirements are important nursing measures that improve the level of patient comfort. Skin care and nursing measures are directed toward relief of pain and discomfort associated with jaundice, anorexia, and profound weight loss. Specialty mattresses are beneficial and protect bony prominences from pressure. Pain associated with pancreatic cancer may be severe and may require liberal use of opioids; patient-controlled analgesia should be considered for the patient with severe, escalating pain.

There is growing evidence that symptoms frequently appear together in a phenomenon known as a symptom cluster. A symptom cluster is two (or more) symptoms occurring at the same time. The symptoms included in a cluster may have shared underlying mechanisms or outcomes, and pancreatic cancer is one of the diseases that appears to have associated symptom clusters. It may be beneficial to investigate these symptoms as a cluster, rather than individually (Miaskowski, Barsevick, Berger, et al., 2017).

Symptom clusters associated with pancreatic cancer have been identified in several studies and include affective, GI, gustatory, and discomfort-related symptoms (Burrell, Yeo, Smeltzer, et al., 2018a, 2018b). See the Nursing Research Profile in [Chart 44-5](#).

Knowledge and awareness regarding symptom clusters is important to nursing care for patients with pancreatic cancer. Improving nursing assessments and interventions based upon this evidence may improve the quality of life and survival in patients with pancreatic cancer undergoing surgical resection for this disease (Burrell et al., 2018a, 2018b). Because of the

poor prognosis and likelihood of short survival, end-of-life preferences are discussed and honored. If appropriate, the nurse refers the patient to hospice care. See Chapters 12 and 13 for care of the patient with cancer and end-of-life care, respectively.

Promoting Home, Community-Based, and Transitional Care



Educating Patients About Self-Care

Specific education for the patient and family varies with the stage of disease and the treatment choices made by the patient. If the patient elects to receive chemotherapy, the nurse focuses on prevention of side effects and complications of the agents used. If surgery is performed to relieve obstruction and establish biliary drainage, education addresses management of the drainage system and monitoring for complications. The nurse educates the family about changes in the patient's status that should be reported to the primary provider.

Continuing and Transitional Care

A referral for home, community-based, or transitional care is indicated to help the patient and family deal with the physical problems and discomforts associated with pancreatic cancer and the psychological impact of the disease. The nurse assesses the patient's physical status, fluid and nutritional status, skin integrity, and the adequacy of pain management. The nurse educates the patient and family on strategies to prevent skin breakdown and relieve pain, pruritus, and anorexia. It is important to discuss and arrange palliative care (hospice services) as indicated in an effort to relieve patient discomfort, assist with care, and comply with the patient's end-of-life decisions and wishes.

Tumors of the Head of the Pancreas

Tumors of the head of the pancreas comprise 60% to 80% of all pancreatic tumors (Goldman & Schafer, 2019; Yeo, 2019). Tumors in this region of the pancreas obstruct the common bile duct where the duct passes through the head of the pancreas to join the pancreatic duct and empty at the ampulla of Vater into the duodenum. The tumors producing the obstruction may arise from the pancreas, the common bile duct, or the ampulla of Vater (Yeo, 2019).

Chart 44-5



NURSING RESEARCH PROFILE

Symptom Clusters in Patients with Pancreatic Cancer

Burrell, S. A., Yeo, T. P., Smeltzer, S. C., et al. (2018a). Symptom clusters in patients with pancreatic cancer undergoing surgical resection: Part I. *Oncology Nursing Forum*, 45(4), e36–e52.

Purpose

The purpose of this study was to describe patient-reported symptoms and symptom clusters in patients with pancreatic cancer undergoing surgical resection.

Design

The study recruited 143 patients with stage II pancreatic cancer undergoing surgical resection alone or with subsequent adjuvant chemoradiation or chemotherapy to participate in a nested, longitudinal, exploratory study through convenient sampling techniques. The Functional Assessment in Cancer Therapy–Hepatobiliary questionnaire was used to assess 17 pancreatic cancer symptoms preoperatively and at 3, 6, and 9 months postoperatively. Exploratory and confirmatory factor analyses were used to identify symptom clusters.

Findings

Fatigue, trouble sleeping, poor appetite, trouble digesting food, and weight loss were consistently reported as the most prevalent and severe symptoms. Sixteen distinct symptom clusters were identified within 9 months of surgery. Four core symptom clusters persisted over time: affective, GI, gustatory, and discomfort.

Nursing Implications

The findings in this study may be used by nurses to provide anticipatory patient and family education and guidance as they begin and continue with their treatment.

Nurses and other health care providers will be knowledgeable about symptoms and symptom clusters in this population, which may guide assessments and interventions.

Clinical Manifestations

The obstructed flow of bile produces jaundice, clay-colored stools, and dark urine. Malabsorption of nutrients and fat-soluble vitamins may result if the

tumor obstructs the entry of bile to the GI tract. Abdominal discomfort or pain and pruritus may be noted, along with anorexia, weight loss, and malaise. If these signs and symptoms are present, cancer of the head of the pancreas is suspected.

The jaundice of this disease must be differentiated from that due to a biliary obstruction caused by a gallstone in the common duct. Jaundice caused by a gallstone is usually intermittent and occurs more commonly in women and in people with obesity and who have had previous symptoms of gallbladder disease.

Assessment and Diagnostic Findings

Diagnostic studies may include duodenography, angiography by hepatic or celiac artery catheterization, pancreatic scanning, PTC, ERCP, and percutaneous needle biopsy of the pancreas. Results of a biopsy of the pancreas may aid in the diagnosis.

Medical Management

Before extensive surgery can be performed, a period of preparation is necessary because the patient's nutritional status and physical condition are often quite compromised. Various liver and pancreatic function studies are performed. A diet high in protein along with pancreatic enzymes, which aid digestion, is often prescribed. Preoperative preparation includes adequate hydration, correction of prothrombin deficiency with vitamin K, and treatment of anemia to minimize postoperative complications. Enteral or parenteral nutrition and blood component therapy are frequently required.

A biliary drainage procedure may be performed, usually with a catheter via percutaneous access, to relieve the jaundice and, perhaps, to provide time for a thorough diagnostic evaluation. Total pancreatectomy (removal of the pancreas) may be performed if there is no evidence of direct extension of the tumor to adjacent tissues or regional lymph nodes. A pancreaticoduodenectomy (Whipple's procedure or resection) is used for potentially resectable cancer of the head of the pancreas (see Fig. 44-7) (Brunicardi, 2019; Yeo, 2019). This procedure involves removal of the gallbladder, a portion of the stomach, duodenum, proximal jejunum, head of the pancreas, and distal common bile duct. Reconstruction involves anastomosis of the remaining pancreas and stomach to the jejunum (Cameron & Cameron, 2020; Townsend et al., 2016; Yeo, 2019). A pylorus-preserving pancreaticojejunostomy (PPPD) may also be performed for tumors of the head of the pancreas and may reduce postgastrectomy symptoms and improve overall GI function (Yeo, 2019). The result of these procedures is removal of the tumor, allowing flow of bile into the jejunum. If the tumor cannot be

excised, the jaundice may be relieved by diverting the bile flow into the jejunum by anastomosing the jejunum to the gallbladder, a procedure known as **cholecystojejunostomy** (Brunicardi, 2019; Yeo, 2019).

The postoperative management of patients who have undergone a pancreatectomy or a pancreaticoduodenectomy is similar to the management of patients after extensive GI or biliary surgery. The patient's physical status is often suboptimal, increasing the risk of postoperative complications. Hemorrhage, vascular collapse, and hepatorenal failure remain the major postoperative complications. The mortality rate associated with these procedures has decreased because of advances in nutritional support and improved surgical techniques. A nasogastric tube with suction and parenteral nutrition allow the GI tract to rest while promoting adequate nutrition.

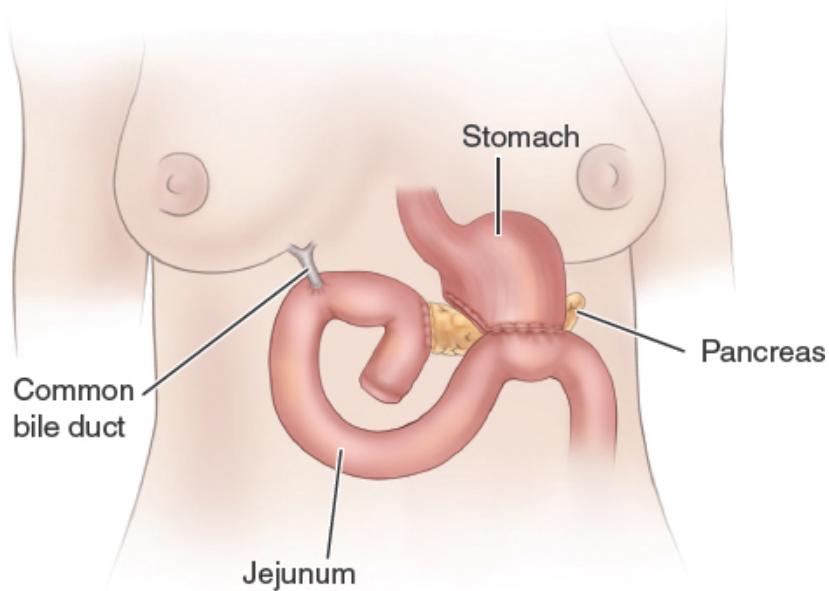
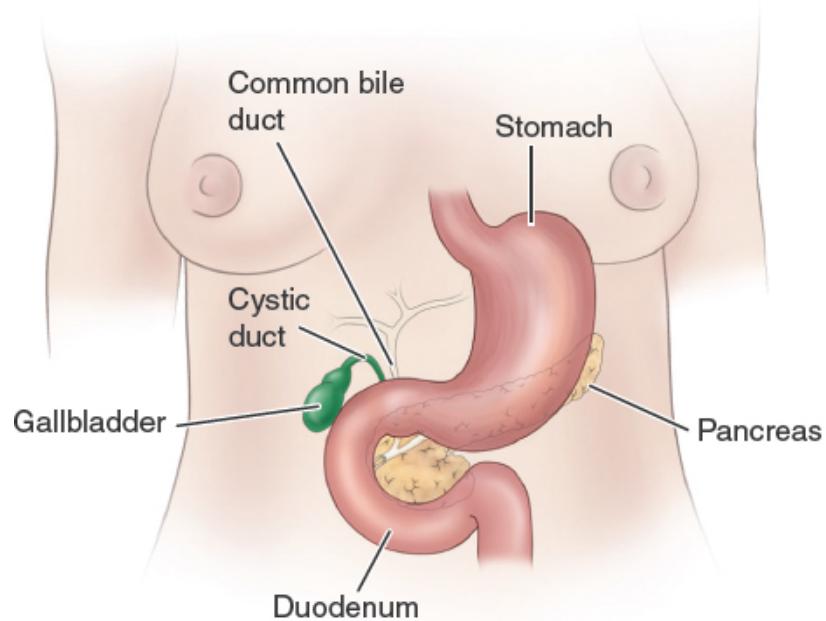


Figure 44-7 • Pancreatoduodenectomy (Whipple's procedure or resection). End result of resection of carcinoma of the head of the pancreas or the ampulla of Vater. The common duct is sutured to the side of the jejunum (choledochojejunostomy), and the remaining portion of the pancreas and the end of the stomach are sutured to the side of the jejunum.



Nursing Management

Preoperatively and postoperatively, nursing care is directed toward promoting patient comfort, preventing complications, and assisting the patient to return to and maintain as normal and comfortable a life as possible. The nurse closely monitors the patient in the intensive care unit after surgery; in the immediate postoperative period, multiple IV and arterial lines are used for fluid and blood replacement and hemodynamic monitoring, and a mechanical ventilator may be used. It is important to note and report changes in vital signs, arterial blood gases and pressures, pulse oximetry, laboratory values, and urine output. The nurse must also consider the patient's compromised nutritional status and risk of bleeding. Depending on the type of surgical procedure performed, malabsorption syndrome and diabetes are likely; the nurse must address these issues during acute and long-term patient care.

Although the patient's physiologic status is the focus of the health care team in the immediate postoperative period, the patient's psychological and emotional states must be considered, along with those of the family. The patient has undergone a major high-risk surgery and is critically ill; anxiety and depression may affect recovery. The immediate and long-term outcomes of this extensive surgical resection are uncertain, and the patient and family require emotional support and understanding in the critical and stressful preoperative and postoperative periods.

Promoting Home, Community-Based, and Transitional Care



Educating Patients About Self-Care

The patient who has undergone this extensive surgery requires careful and thorough preparation for self-care at home. The nurse educates the patient and family about strategies to relieve pain and discomfort, along with strategies to manage drains, if present, and to care for the surgical incision. The patient and family members may require education about the use of appropriate analgesic medications, parenteral nutrition, wound care, skin care, increasing activity, and management of drainage.

The nurse may need to educate the patient and family about the need for modifications in the diet because of malabsorption and hyperglycemia resulting from the surgery. It is important to educate the patient and family about the continuing need for pancreatic enzyme replacement, a low-fat diet, and vitamin supplementation. The nurse describes—verbally and in writing—the signs and symptoms of complications and educates the patient and family about indicators of complications that should be reported promptly.

Discharge of the patient to a long-term care or rehabilitation facility may be warranted after surgery as extensive as pancreatectomy or pancreaticoduodenectomy, particularly if the patient's preoperative status is not

optimal. Information about the education that has been provided is shared with the long-term care staff so that instructions can be clarified and reinforced. During the recovery or long-term phase of care, the patient and family receive further education about self-care in the home.

Continuing and Transitional Care

A referral for home, community-based, or transitional care may be indicated when the patient returns home. The nurse assesses the patient's physical and psychological status and the ability of the patient and family to manage needed care. The nurse provides needed physical care and monitors the adequacy of pain management. In addition, it is important to assess the patient's nutritional status and monitor the use of enteral or parenteral nutrition, if used. The nurse discusses the use of hospice services with the patient and family and makes a referral if indicated.

Pancreatic Islet Tumors

At least two types of tumors of the pancreatic islet cells are known: those that secrete insulin (insulinoma) and those in which insulin secretion is not increased (nonfunctioning islet cell cancer). All of these types of tumors combined are termed *neuroendocrine tumors* (NETs). Insulinomas produce hypersecretion of insulin and cause an excessive rate of glucose metabolism. The resulting hypoglycemia may produce symptoms of weakness, mental confusion, and seizures. These symptoms may be relieved almost immediately by oral or IV administration of glucose. The 5-hour glucose tolerance test is helpful to diagnose insulinoma and to distinguish a diagnosis of NET from other causes of hypoglycemia.

Surgical Management

If a tumor of the islet cells (a type of NET) has been diagnosed, surgical treatment with removal of the tumor is usually recommended (Brunicardi, 2019; Yeo, 2019). The tumors may be benign adenomas, or they may be malignant. Complete removal usually results in almost immediate relief of symptoms. In some patients, symptoms may be produced by simple hypertrophy of this tissue rather than a tumor of the islet cells. In such cases, a partial pancreatectomy (removal of the tail and part of the body of the pancreas) is performed.

Nursing Management

In preparing the patient for surgery, the nurse must be alert for symptoms of hypoglycemia and be ready to administer glucose as prescribed if symptoms occur. Postoperatively, the nursing management is the same as after other upper abdominal surgical procedures, with special emphasis on monitoring serum glucose levels. Patient education is determined by the extent of surgery and alterations in pancreatic function.

Hyperinsulinism

Hyperinsulinism is caused by overproduction of insulin by the pancreatic islets. Symptoms resemble those of excessive doses of insulin and are attributable to the same mechanism: an abnormal reduction in blood glucose levels. Clinically, it is characterized by episodes during which the patient experiences unusual hunger, nervousness, sweating, headache, and faintness; in severe cases, seizures and episodes of unconsciousness may occur. The findings at the time of surgery or at autopsy may indicate hyperplasia (overgrowth) of the islets of Langerhans or a benign or malignant tumor involving the islets that is capable of producing large amounts of insulin (see preceding discussion). Occasionally, tumors of nonpancreatic origin produce an insulinlike material that can cause severe hypoglycemia and may be responsible for seizures coinciding with blood glucose levels that are too low to sustain normal brain function (i.e., lower than 30 mg/dL [1.6 mmol/L]) (Goldman & Schafer, 2019; Kellerman & Rakel, 2018).

All of the symptoms that accompany spontaneous hypoglycemia are relieved by the oral or parenteral administration of glucose. Surgical removal of the hyperplastic or neoplastic tissue from the pancreas is the only successful method of treatment. About 15% of patients with spontaneous or functional hypoglycemia eventually develop diabetes.

Ulcerogenic Tumors

Some tumors of the islets of Langerhans are associated with hypersecretion of gastric acid that produces ulcers in the stomach, duodenum, and jejunum. This is referred to as **Zollinger-Ellison syndrome**. The hypersecretion is so excessive that even after partial gastric resection, enough acid is produced to cause further ulceration. If a marked tendency to develop gastric and duodenal ulcers is noted, an ulcerogenic tumor of the islets of Langerhans is considered (Brunicardi, 2019; Yeo, 2019). The clinical manifestations include nausea, vomiting, diarrhea, and burning discomfort or pain in the upper abdomen. The diagnostic test for this disorder includes measuring a blood gastrin level. Imaging tests may include CT scan or MRI, EUS or upper endoscopy.

Scintigraphy and positron emission tomography (PET)/CT are sensitive and specific tests for this disease (Brunicardi, 2019; Yeo, 2019).

These tumors, which may be benign or malignant, are treated by excision, if possible. Frequently, however, removal is not possible because of extension beyond the pancreas and because the tumors are often quite small and difficult to locate. Acid hypersecretion in patients with gastrinoma can be managed with proton pump inhibitors and this is often the first-line treatment intervention. Highly selective vagotomy may make management easier in some patients and should be considered in those with surgically untreatable or unresectable gastrinoma. Total gastrectomy for Zollinger-Ellison is not indicated (Brunicardi, 2019; Yeo, 2019). Embolization or radiofrequency ablation may also be used to control the tumor.

The many disorders of the biliary and pancreatic systems result in a host of clinical and biochemical abnormalities presented in detail in this chapter. The recognition and management of these illnesses pose a challenge to nurses. There is now much evidence about the prevention, diagnosis, and treatment of these disorders. However, there is still much to learn as the medical and nursing communities strive to provide optimum care to patients afflicted with biliary and pancreatic disorders.

CRITICAL THINKING EXERCISES

1 pq A 57-year-old man has been having symptoms over the past 2 months including burning discomfort or pain in his upper abdomen, nausea, and diarrhea. He thought that he had acid reflux with heartburn so he started an over-the-counter histamine antagonist. His appetite had decreased and he had lost several pounds. The patient's physician performed a complete physical examination and thinks that the patient may have Zollinger-Ellison syndrome. What laboratory testing might you anticipate? What diagnostic imaging will most likely be performed? What medication or medications may be prescribed? Since the patient continues to be symptomatic, surgery is recommended. What patient education can you provide to best prepare the patient for the surgical intervention and the postoperative care?

2 ebp A 58-year-old man presents to the emergency department with complaints of severe midepigastic pain. He is also nauseated and vomiting. His vital signs reveal hypotension and tachycardia and he is mildly febrile. His history is significant for alcohol abuse and hypertension. What laboratory and imaging studies do you expect to be performed? His CT scan revealed noninfected pancreatic necrosis. An evidence-based approach for managing pancreatic necrosis was implemented. Would surgery be included in this initial approach? What first-line intervention would you expect to be implemented in this case? What classification of pancreatitis is this patient experiencing? After 1 week in the hospital, the patient's fever is higher, he is having more abdominal pain and a repeat CT scan reveals multiple fluid collections and worsening edema consistent with infected pancreatic necrosis. What is the evidence for new therapies to effectively treat this patient?

3 ipc A 55-year-old woman has been experiencing upper abdominal pain for several weeks. She believes that she has gallbladder disease but is concerned because her mother died from pancreatic cancer. She visits her primary provider who prescribes diagnostic and laboratory studies. Which laboratory tests would you expect to be assessed? An MRI reveals a small mass at the head of the pancreas. 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

- Al-Anon Family Groups Headquarters, www.al-anon.alateen.org
- Alcoholics Anonymous World Services (AAWS), www.aa.org
- American Gastroenterological Association (AGA), www.gastro.org
- Endocrine Society, www.endo-society.org
- National Pancreas Foundation (NPF), www.pancreasfoundation.org

45 Assessment and

Management of Patients with Endocrine Disorders

LEARNING OUTCOMES

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

- 1.** Describe the functions of each of the endocrine glands and their hormones.
- 2.** Differentiate diagnostic studies and clinical manifestations for endocrine disorders.
- 3.** Demonstrate knowledge of management strategies for endocrine disorders.
- 4.** Explain nursing interventions in the care of patients with endocrine disorders.
- 5.** Use the nursing process as a framework for care of the patient with an endocrine disorder.

NURSING CONCEPTS

Cellular Regulation

Family

Infection

Inflammation

Metabolism

Mobility

Nutrition

Stress

GLOSSARY

acromegaly: progressive enlargement of peripheral body parts resulting from excessive secretion of growth hormone

Addison's disease: chronic adrenocortical insufficiency due to inadequate adrenal cortex function

Addisonian crisis: acute adrenocortical insufficiency; characterized by hypotension, cyanosis, fever, nausea/vomiting, and signs of shock

adrenalectomy: surgical removal of one or both adrenal glands

adrenocorticotropic hormone (ACTH): hormone secreted by the anterior pituitary, essential for growth and development

androgens: male sex hormones

basal metabolic rate: chemical reactions occurring when the body is at rest

calcitonin: hormone secreted by the thyroid gland; participates in calcium regulation

Chvostek sign: spasm of the facial muscles produced by sharply tapping over the facial nerve in front of the parotid gland and anterior to the ear; suggestive of latent tetany in patients with hypocalcemia

corticosteroids: hormones produced by the adrenal cortex or their synthetic equivalents

Cushing's syndrome: group of symptoms produced by an oversecretion of adrenocorticotropic hormone; characterized by truncal obesity, "moon face," acne, abdominal striae, and hypertension

diabetes insipidus: condition in which abnormally large volumes of dilute urine are excreted as a result of deficient production of vasopressin

euthyroid: state of normal thyroid hormone production

exophthalmos: abnormal protrusion of one or both eyeballs

glucocorticoids: steroid hormones secreted by the adrenal cortex in response to adrenocorticotropic hormone; produce a rise of liver glycogen and blood glucose

goiter: enlargement of the thyroid gland

Graves disease: a form of hyperthyroidism; characterized by a diffuse goiter and exophthalmos

hormones: chemical transmitter substances produced in one organ or part of the body and carried by the bloodstream to other cells or organs on which they have a specific regulatory effect

mineralocorticoids: steroid hormones secreted by the adrenal cortex

myxedema: severe hypothyroidism; can be with or without coma

negative feedback: regulating mechanism in which an increase or decrease in the level of a substance decreases or increases the function of the organ producing the substance

pheochromocytoma: adrenal medulla tumor

syndrome of inappropriate antidiuretic hormone (SIADH): excessive secretion of antidiuretic hormone from the pituitary gland despite low serum osmolality level

thyroidectomy: surgical removal of all or part of the thyroid gland

thyroiditis: inflammation of the thyroid gland; may lead to chronic hypothyroidism or may resolve spontaneously

thyroid-stimulating hormone (TSH): released from the pituitary gland; causes stimulation of the thyroid, resulting in release of T_3 and T_4

thyroid storm: life-threatening condition of the thyroid due to untreated hyperthyroidism

thyrotoxicosis: condition produced by excessive endogenous or exogenous thyroid hormone

thyroxine (T_4): thyroid hormone; active iodine compound formed and stored in the thyroid; deiodinated in peripheral tissues to form triiodothyronine; maintains body metabolism in a steady state

triiodothyronine (T_3): thyroid hormone; formed and stored in the thyroid; released in smaller quantities, biologically more active, and with faster onset of action than T_4 ; widespread effect on cellular metabolism

Trousseau sign: carpopedal spasm induced when blood flow to the arm is occluded using a blood pressure cuff or tourniquet, causing ischemia to the distal nerves; suggestive sign for latent tetany in hypocalcemia

vasopressin: antidiuretic hormone secreted by the posterior pituitary

The endocrine system plays a vital role in orchestrating transportation of chemicals across cell membranes, growth and development, metabolism, fluid and electrolyte balance, acid-base balance, adaptation, and reproduction (Norris, 2019). This interconnected network of glands is closely linked with the nervous and immune systems, regulating the functions of multiple body organs. The hypothalamus is responsible for this interrelationship. The pituitary gland, because of its status as the master gland, plays an important role in the regulation of endocrine hormones; its primary function is to secrete hormones into the bloodstream, which in turn affects endocrine glands such as the thyroid. Disorders of the endocrine system are common and are manifested as hyper- and hypofunction.

Nursing interventions are essential in the management of patients with endocrine disorders. This chapter focuses on the anatomy and physiology of the endocrine system; the most common endocrine disorders of the pituitary, thyroid, parathyroid, and adrenal glands; clinical manifestations; diagnostic studies; medical management; and nursing interventions. The unique endocrine and exocrine functions of the pancreas, pancreatic function, and

associated pancreatic disorders are discussed in [Chapter 44](#) and reproductive structures, including the ovaries and testes, are discussed in Chapters 50 and 53, respectively.

ASSESSMENT OF THE ENDOCRINE SYSTEM

Anatomic and Physiologic Overview

The endocrine system involves the release of chemical transmitter substances known as **hormones**. These substances regulate and integrate body functions by acting on local or distant target sites. Hormones are generally produced by the endocrine glands but may also be produced by specialized tissues such as those found in the gastrointestinal (GI) system, the kidney, and white blood cells. The GI mucosa produces hormones (e.g., gastrin, enterogastrone, secretin, cholecystokinin) that are important in the digestive process; the kidneys produce erythropoietin, a hormone that stimulates the bone marrow to produce red blood cells; and the white blood cells produce cytokines (hormonelike proteins) that actively participate in inflammatory and immune responses.

The endocrine system has a unique relationship with the immune and the nervous systems. Chemicals such as neurotransmitters (e.g., epinephrine) released by the nervous system can also function as hormones when needed. The immune system responds to the introduction of foreign agents by means of chemical messengers (cytokines), and is also subject to regulation by adrenal corticosteroid hormones (Norris, 2019).

Glands of the Endocrine System

The endocrine system is composed of the pituitary gland, thyroid gland, parathyroid glands, adrenal glands, pancreatic islets, ovaries, and testes. See [Figure 45-1](#) for major hormonal secreting glands of the endocrine system. Most hormones secreted from endocrine glands are released directly into the bloodstream. However, exocrine glands, such as sweat glands, secrete their products through ducts onto epithelial surfaces or into the GI tract.

Function and Regulation of Hormones

Hormones help regulate organ function in concert with the nervous system. The rapid action by the nervous system is balanced by slower hormonal action. This dual regulatory system permits precise control of organ functions in response to changes within and outside the body.

The endocrine glands are composed of secretory cells arranged in minute clusters known as acini. A rich blood supply provides a vehicle for the hormones produced by the endocrine glands to enter the bloodstream rapidly.

The amount of circulating hormones depends on their unique function and the body's needs. In the healthy physiologic state, hormone concentration in the bloodstream is maintained at a relatively constant level. To prevent accumulation, these hormones must be inactivated continuously by a **negative feedback** system so that when the hormone concentration increases, further production of that hormone is inhibited. Conversely, when the hormone concentration decreases, the rate of production of that hormone increases.

Classification and Action of Hormones

Hormones are classified into four categories according to their structure: (1) amines and amino acids (e.g., epinephrine, norepinephrine, and thyroid hormones); (2) peptides, polypeptides, proteins, and glycoproteins (e.g., thyrotropin-releasing hormone [TRH], follicle-stimulating hormone [FSH], and growth hormone [GH]); (3) steroids (e.g., **corticosteroids**, which are hormones produced by the adrenal cortex or their synthetic equivalents); and (4) fatty acid derivatives (e.g., eicosanoid, retinoids) (Norris, 2019). Although most hormones released by endocrine glands can be transported to distant target sites for action, some hormones never enter the bloodstream because they act locally in the area where they are released; this is called *paracrine action* (e.g., the effect of sex hormones on the ovaries). Others may act on the actual cells from which they were released; this is called *autocrine action* (e.g., the effect of insulin from pancreatic beta cells on those cells) (Norris, 2019).

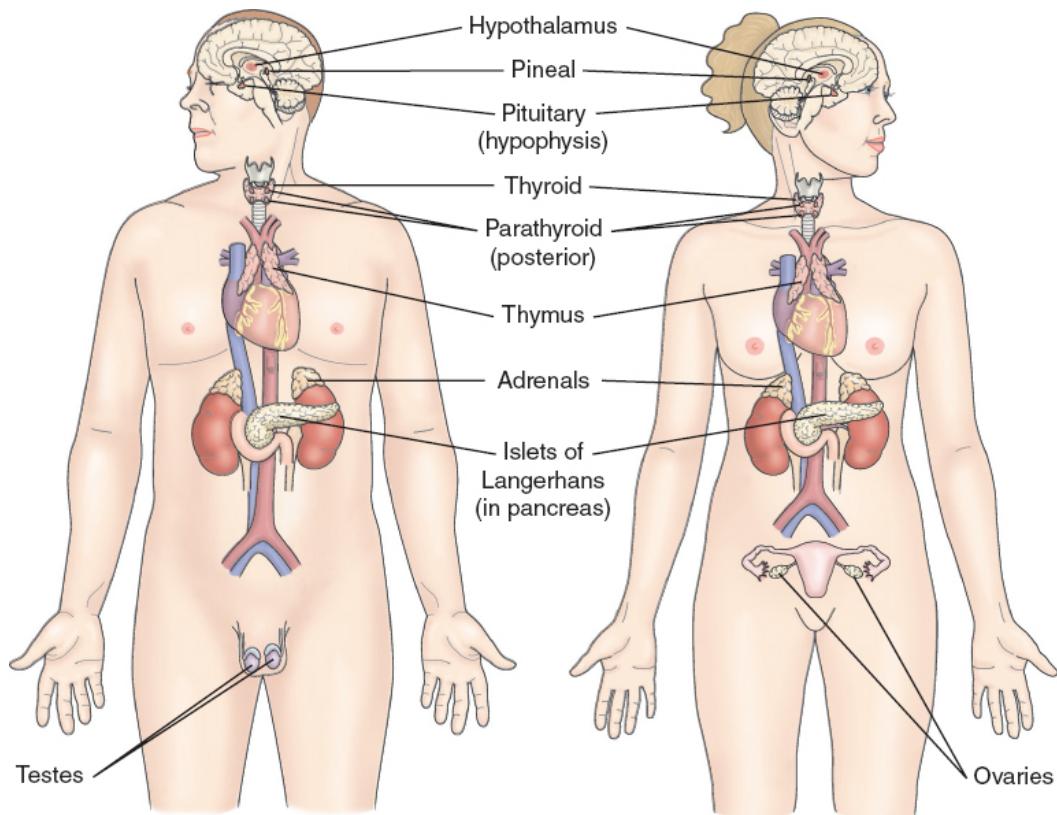


Figure 45-1 • Major hormone-secreting glands of the endocrine system.

Hormones can alter the function of the target tissue by interacting with chemical receptors located either on the cell membrane or in the interior of the cell. For example, *peptide* and *protein hormones* interact with receptor sites on the cell surface, resulting in stimulation of the intracellular enzyme adenyl cyclase. This causes increased production of cyclic 3',5'-adenosine monophosphate (AMP). The cyclic AMP inside the cell alters enzyme activity. Thus, cyclic AMP is the “second messenger” that links the peptide hormone at the cell surface to a change in the intracellular environment. Some protein and peptide hormones also act by changing membrane permeability and act within seconds or minutes. The mechanism of action for *amine hormones* is similar to that for peptide hormones (Norris, 2019).

Steroid hormones, because of their smaller size and higher lipid solubility, penetrate cell membranes and interact with intracellular receptors. The steroid–receptor complex modifies cell metabolism and the formation of messenger ribonucleic acid (mRNA) from deoxyribonucleic acid (DNA). The mRNA then stimulates protein synthesis within the cell. Steroid hormones require several hours to exert their effects, because they exert their action by the modification of protein synthesis.

Assessment

The nursing assessment of the patient with endocrine dysfunction includes a health history and physical examination that evaluates the effects of endocrine disorders on the patient.

Health History

Although specific endocrine disorders are often accompanied by specific clinical symptoms, more general manifestations may also occur. A thorough health history and review of systems are necessary for diagnosis and management of these disorders. Patients should be asked if they have experienced changes in the following areas: energy level, tolerance to heat or cold, weight, thirst, frequency of urination, bowel function, body proportions, muscle mass, fat and fluid distribution, secondary sexual characteristics (e.g., loss or growth of hair), menstrual cycle, memory, concentration, sleep patterns, mood, vision, joint pain, and sexual dysfunction. Documentation is important regarding the severity of these changes, the length of time the patient has experienced these changes, the way in which these changes have affected the patient's ability to carry out activities of daily living, the effect of the changes on the patient's self-perception, and family history.

Physical Assessment

The physical examination should include vital signs; head-to-toe inspection; and palpation of skin, hair, and thyroid. Findings should be compared with previous findings, if available. Physical, psychological, and behavioral changes should be noted. Examples of changes in physical characteristics on examination may include appearance of facial hair in women, "moon face," "buffalo hump," **exophthalmos** (abnormal protrusion of one or both eyeballs), vision changes, edema, thinning of the skin, obesity of the trunk, thinness of the extremities, increased size of the feet and hands, edema, and hypo- or hyperreflexia. The patient may also exhibit changes in mood and behavior such as nervousness, lethargy, and fatigue (Jensen, 2019).

Diagnostic Evaluation

A variety of diagnostic studies are used to evaluate the endocrine system. The nurse educates the patient about the purpose of the prescribed studies, what to expect, and any possible side effects related to these examinations prior to testing. The nurse notes trends in results that provide information about disease progression as well as the patient's response to therapy.

Blood Tests

Blood tests determine the levels of circulating hormones, the presence of autoantibodies, and the effect of a specific hormone on other substances (e.g., the effect of insulin on blood glucose levels). The serum levels of a specific hormone may provide information to determine the presence of hypo- or hyperfunction of the endocrine system and the site of dysfunction. An example of a specific hormone that is amenable to analyzing by blood testing is thyroid hormone (i.e., T₃ and T₄; see later discussion). Radioimmunoassay tests are frequently used to detect antigen levels which give additional information about hormone levels and levels of other substances (Fischbach & Fischbach, 2018).

Urine Tests

Urine tests are used to measure the amount of hormones or the end products of hormones excreted by the kidneys. One-time specimens or, in some disorders, 24-hour urine specimens are collected to measure hormones or their metabolites. For example, urinary levels of free catecholamines (norepinephrine, epinephrine, and dopamine) may be measured in patients with suspected **pheochromocytoma**, a tumor of the adrenal medulla. Several disadvantages related to urine tests that must be considered are that patients may be unable to urinate at scheduled intervals and that some medications or disease states may affect the test results (Norris, 2019).

Additional Diagnostic Studies

Stimulation tests are used to confirm hypofunction of an endocrine organ. The tests determine how an endocrine gland responds to the administration of stimulating hormones that are normally produced or released by the hypothalamus or pituitary gland. If the endocrine gland responds to this stimulation, the specific disorder may be in the hypothalamus or pituitary. Failure of the endocrine gland to respond to this stimulation helps identify the problem as being in the endocrine gland itself.

Suppression tests are used to detect hyperfunction of an endocrine organ. They determine if the organ is not responding to the negative feedback mechanisms that normally control secretion of hormones from the hypothalamus or pituitary gland. Suppression tests measure the effect of a given exogenous dose of the hormone on the endogenous secretion of the hormone or on the secretion of stimulation hormones from the hypothalamus or pituitary gland.

Imaging studies include radioactive scanning, magnetic resonance imaging (MRI), computed tomography (CT), ultrasonography, positron emission

tomography (PET), and dual-energy x-ray absorptiometry (DXA) (Norris, 2019).

Genetic screening is becoming more routine in the assessment of endocrine disorders (see Chart 45-1). DNA testing can be used for the identification of specific genes associated with endocrine disorders, selective targeting for drug development, and increased understanding of the function of the endocrine system. Genetic screening is used to determine the presence of a gene mutation that may predispose a person to a certain condition, the implications of which must be considered by the patient.

Chart 45-1



GENETICS IN NURSING PRACTICE

Metabolic and Endocrine Disorders

Metabolic and endocrine disorders that are influenced by genetic factors are complex and typically impact multiple body systems. Some examples of genetic metabolic and endocrine disorders include:

- Diabetes
- Disorders in amino acids (e.g., phenylketonuria, homocystinuria, maple syrup urine disease)
- Disorders of carbohydrate metabolism (e.g., galactosemia)
- Disorders of fatty acid oxidation (e.g., medium-chain acyl-CoA dehydrogenase deficiency)
- Disorders of glycogen storage (e.g., Pompe disease, Von Gierke disease, Danon disease, Cori disease, Anderson disease, McArdle disease)
- Disorders of lysosomal storage disease (e.g., Tay Sachs, Gaucher, Niemann-Pick, Fabry disease)
- Disorders of mucopolysaccharides (e.g., Hurler syndrome, Hunter disease, Morquio syndrome)
- Disorders in urea cycle (e.g., ornithine transcarbamylase deficiency)
- Hemochromatosis
- McCune-Albright syndrome
- Multiple endocrine neoplasia type I and type II

Nursing Assessments

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

Family History Assessment

- Assess family history for relatives with early-onset hepatic, pancreatic, or endocrine disease.
- Inquire about family members with diabetes and their ages at onset.
- Assess family history of other related genetic conditions such as cystic fibrosis, alpha₁-antitrypsin deficiency, and hemochromatosis.

Patient Assessment

- Assess for physical symptoms such as mucosal neuromas, hypertrophied lips, skeletal abnormalities, and marfanoid appearance.
- Assess for signs of arthritis and hemochromatosis (bronze pigmentation of the skin).
- Assess for history of seizures, sweet smell to urine, jaundice, lethargy, vomiting, dehydration, acidosis, neutropenia, hepatomegaly, high ammonia levels

Genetics Resources

Association for Glycogen Storage Disease, www.agdsus.org
 Society for Inherited Metabolic Disorders, www.simd.org
 See Chapter 6, Chart 6-7, for components of genetic counseling.

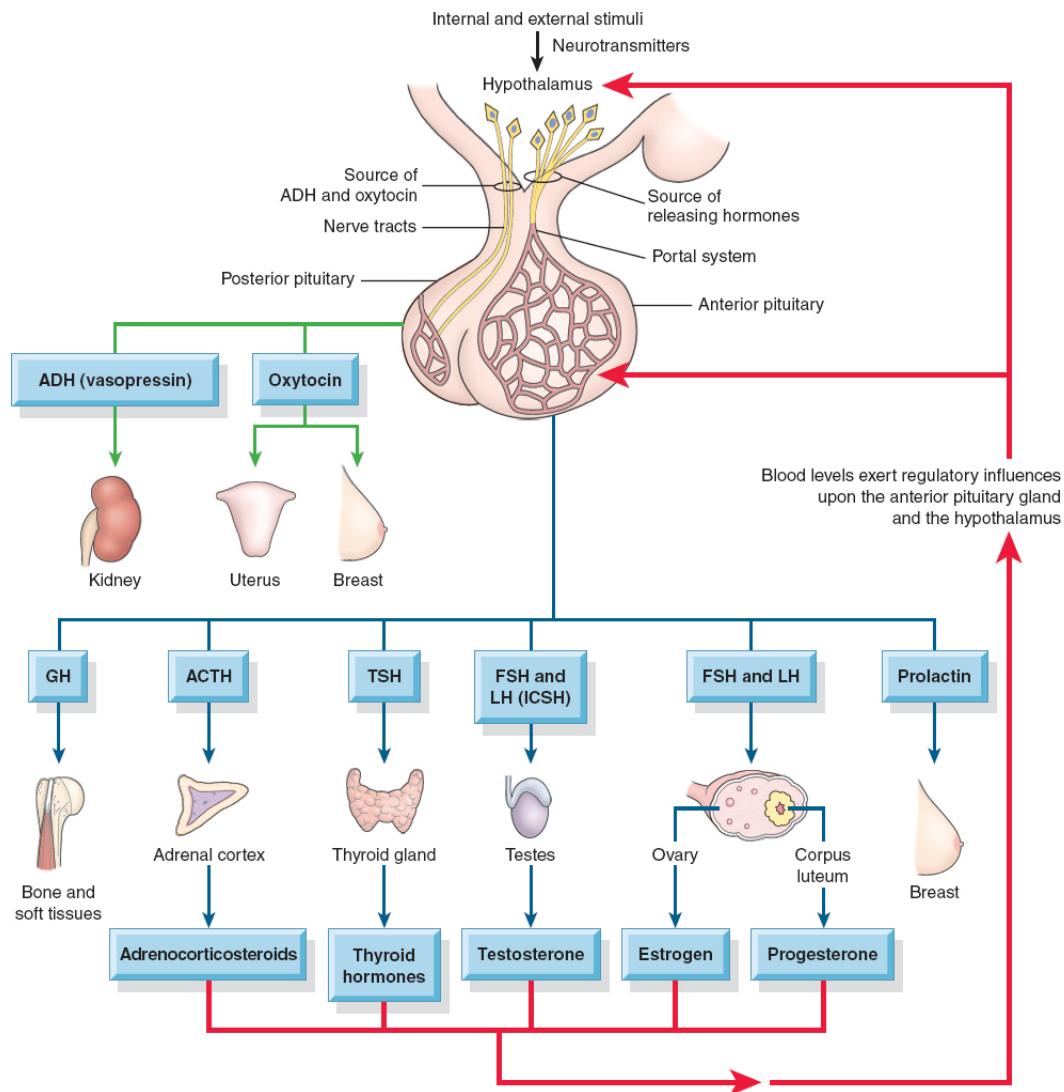


Figure 45-2 • The pituitary gland, the relationship of the brain to pituitary action, and the hormones secreted by the anterior and posterior pituitary lobes. ADH, antidiuretic hormone; GH, growth hormone; ACTH, adrenocorticotrophic hormone; TSH, thyroid-stimulating hormone; FSH, follicle-stimulating hormone; LH, luteinizing hormone; ICSH, interstitial cell-stimulating hormone.

THE PITUITARY GLAND

Anatomic and Physiologic Overview

The pituitary gland, or hypophysis, is commonly referred to as the master gland because of the influence it has on secretion of hormones by other endocrine glands (see Fig. 45-2). The round structure, about 1.27 cm (1/2 inch) in diameter, is located on the inferior aspect of the brain and is divided into anterior and posterior lobes. The gland is controlled by the hypothalamus, which is an adjacent area of the brain that is connected to the pituitary by the pituitary stalk.

Anterior Pituitary

The major hormones of the anterior pituitary gland are FSH, luteinizing hormone (LH), prolactin (PRL), **adrenocorticotropic hormone (ACTH)**, **thyroid-stimulating hormone (TSH)**, and GH (also referred to as somatotropin). The secretion of these major hormones is controlled by releasing factors secreted by the hypothalamus. These releasing factors reach the anterior pituitary by way of the bloodstream in a special circulation called the *pituitary portal blood system*. Other hormones include melanocyte-stimulating hormone and beta-lipotropin; the function of lipotropin is poorly understood.

The hormones released by the anterior pituitary enter the general circulation and are transported to their target organs. The main function of TSH, ACTH, FSH, and LH is the release of hormones from other endocrine glands. Imbalanced ACTH secretion characterizes both Addison's disease (hypoproduction) and Cushing's syndrome (hyperproduction). PRL acts on the breast to stimulate milk production. Hormones that stimulate other organs and tissues are discussed in conjunction with their target organs.

GH, the most abundant anterior pituitary hormone, regulates growth in children and energy and metabolism in adults. GH increases protein synthesis in many tissues, increases the breakdown of fatty acids in adipose tissue, and increases blood glucose levels. Other hormones, such as thyroid hormone and insulin, are required for the GH system to function as well. Secretion of GH is increased by deep sleep, stress, exercise, fasting, malnutrition, hypoglycemia, trauma, hypovolemic shock, and sepsis, and is decreased in the presence of obesity, depression, and hypothyroidism (Norris, 2019).

Posterior Pituitary

The important hormones secreted by the posterior lobe of the pituitary gland are **vasopressin**, also referred to as antidiuretic hormone (ADH), and oxytocin. These hormones are synthesized in the hypothalamus and travel from the hypothalamus to the posterior pituitary gland for storage. Vasopressin controls the excretion of water by the kidney; its secretion is stimulated by an increase in the osmolality of the blood or by a decrease in blood pressure. Oxytocin

secretion is stimulated during pregnancy and at childbirth. Oxytocin also facilitates milk ejection during lactation and increases the force of uterine contractions during labor and delivery.

Pathophysiology

Abnormalities of pituitary function are caused by over- or undersecretion of any of the hormones produced or released by the gland. Abnormalities of the anterior and posterior portions of the gland may occur independently. Hypopituitarism, or hypofunction of the pituitary gland, can result from disease of the pituitary gland itself or disease of the hypothalamus; the outcome is essentially the same. Hypopituitarism can result from radiation therapy to the head and neck area. The total destruction of the pituitary gland by trauma, tumor, or vascular lesion removes all stimuli that are normally received by the thyroid, the gonads, and the adrenal glands. This leads to extreme weight loss, emaciation, atrophy of all endocrine glands and organs, hair loss, impotence, amenorrhea, hypometabolism, and hypoglycemia. Coma and death occur if the missing hormones are not replaced (Norris, 2019).

Anterior Pituitary

Oversecretion (hypersecretion) of the anterior pituitary gland most commonly involves ACTH or GH and results in **Cushing's syndrome** (group of symptoms produced by an oversecretion of ACTH) or acromegaly, respectively. **Acromegaly**, a disorder caused by an excess of GH in adults, results in enlargement of peripheral body parts and soft tissue, after the fusion of the epiphyseal plates has occurred (Hickey & Silverstein, 2019), without an increase in height with an incidence of between 0.2 and 1.1 cases per 100,000 people (Lavrentaki, Paluzzi, Wass, et al., 2017). Although rare, oversecretion of GH in children before the fusion of epiphyseal growth plates result in pituitary gigantism; a person may grow to be 7 or even 8 feet tall. Conversely, insufficient secretion of GH during childhood can result in generalized limited growth and pituitary dwarfism. Undersecretion (hyposecretion) commonly involves all of the anterior pituitary hormones and is termed **panhypopituitarism**. In this condition, the thyroid gland, the adrenal cortex, and the gonads atrophy (shrink) because of loss of the tropic-stimulating hormones. Hypopituitarism may result from destruction of the anterior lobe of the pituitary gland (Norris, 2019).

Posterior Pituitary

The most common disorder related to posterior lobe dysfunction is **diabetes insipidus** (DI), a condition in which abnormally large volumes of dilute urine are excreted as a result of deficient production of vasopressin. DI may occur following surgical treatment of a brain tumor, secondary to nonsurgical brain tumors, traumatic brain injury, infections of the nervous system, post hypophysectomy (removal of the pituitary), failure of renal tubules to respond to ADH, and the use of specific medications (Hollar & Silverstein, 2019).

Pituitary Tumors

Almost all pituitary tumors are benign and are slow growing. The tumors may be primary or secondary and functional or nonfunctional (Norris, 2019). Functional tumors secrete pituitary hormones, whereas nonfunctional tumors do not. Pituitary tumors can cause clinical sequelae from the pressure that they exert on adjoining tissues, from the endocrine dysfunction that they cause, or from their dysfunctional effects on target organs. The principal types of pituitary tumors represent an overgrowth of eosinophilic cells, basophilic cells, or chromophobic cells (i.e., cells with no affinity for either eosinophilic or basophilic stains).

Clinical Manifestations

Eosinophilic tumors that develop early in life result in gigantism. The affected person may be more than 7 feet tall and large in all proportions, yet so weak and lethargic that they can hardly stand. If the disorder begins during adult life, the excessive skeletal growth occurs only in the feet, the hands, the superciliary ridge, the molar eminences, the nose, and the chin, giving rise to the clinical picture called *acromegaly*. However, enlargement involves all tissues and organs of the body, and many of these patients suffer from severe headaches and visual disturbances because the tumors exert pressure on the optic nerves (Norris, 2019). Assessment of central vision and visual fields may reveal loss of color discrimination, diplopia (double vision), or blindness in a portion of a field of vision. Decalcification of the skeleton, muscular weakness, and endocrine disturbances, similar to those occurring in patients with hyperthyroidism, also are associated with this type of tumor.

Basophilic tumors give rise to Cushing's syndrome with features largely attributable to hyperadrenalinism, including masculinization and amenorrhea in females, truncal obesity, hypertension, osteoporosis, and polycythemia.

Chromophobic tumors represent 90% of pituitary tumors. These tumors usually produce no hormones but destroy the rest of the pituitary gland, causing hypopituitarism. People with this disease often have obesity, are somnolent, and exhibit fine, scanty hair; dry, soft skin; a pasty complexion;

and small bones. They also experience headaches, loss of libido, and visual defects progressing to blindness. Other signs and symptoms include polyuria, polyphagia, a lowering of the **basal metabolic rate** (chemical reactions occurring when the body is at rest), and a subnormal body temperature (Sadiq & Silverstein, 2019b).

Assessment and Diagnostic Findings

Diagnostic evaluation requires a careful history and physical examination, including assessment of visual acuity and visual fields. CT and MRI scans are used to diagnose the presence and extent of pituitary tumors. Serum levels of pituitary hormones may be obtained along with measurements of hormones of target organs (e.g., thyroid, adrenal) to assist in diagnosis.

Medical Management

Hypophysectomy, or surgical removal of the pituitary gland through a transsphenoidal approach, is the usual treatment. Stereotactic radiation therapy, which requires the use of a neurosurgery-type stereotactic frame, may be used to deliver external-beam radiation therapy precisely to the pituitary tumor with minimal effect on normal tissue (see [Chapter 12](#)). Other treatments include conventional radiation therapy, bromocriptine, and octreotide. These medications inhibit the production or release of GH and may bring about marked improvement of symptoms. Octreotide and lanreotide may also be used preoperatively to improve the patient's clinical condition and to shrink the tumor (American Association of Neurological Surgeons, 2019).

Surgical Management

Hypophysectomy is the treatment of choice in patients with Cushing's disease resulting from excessive production of ACTH by a pituitary tumor. Hypophysectomy may also be performed on occasion as a palliative measure to relieve bone pain secondary to metastasis of malignant lesions of the breast and prostate.

Several approaches are used to remove or destroy the pituitary gland, including surgical removal by transfrontal, subcranial, or oronasal-transsphenoidal approaches; irradiation; and cryosurgery. The transsphenoidal approach and the nursing management of a patient undergoing cranial surgery are discussed in [Chapter 61](#). Features or symptoms of acromegaly are unaffected by surgical removal of the tumor.

The absence of the pituitary gland alters the function of many body systems. Menstruation ceases and infertility occurs after total or near-total ablation of the pituitary gland. Replacement therapy with corticosteroids and thyroid hormone is necessary.

Diabetes Insipidus

Diabetes insipidus (DI) is a rare disorder that occurs due to injury to the hypothalamus or pituitary gland with a deficiency of ADH (vasopressin) that results in excretion of large volumes of dilute urine and extreme thirst. DI is characterized as central, nephrogenic, or dipsogenic, as well as gestational (Hollar & Silverstein, 2019).

The primary etiology for central DI is head trauma but other causes include surgery, infection, inflammation, brain tumors, or cerebral vascular disease; it also may be idiopathic. Nephrogenic DI etiologic factors include kidney injury, medications such as lithium, hypokalemia, and hypercalcemia. Dipsogenic DI is caused by a defect in the hypothalamus and may be the result of damage to the pituitary gland from a head injury, surgery, infection, inflammatory process, or a tumor (National Institute of Diabetes and Digestive and Kidney Diseases, 2019). DI must be differentiated from diabetes, which may also cause polydipsia and excessive urination.

Clinical Manifestations

Without the action of ADH on the distal nephron of the kidney, an enormous daily output (greater than 250 mL per hour) of very dilute urine with a specific gravity of 1.001 to 1.005 occurs (Hollar & Silverstein, 2019). The urine contains no abnormal substances such as glucose or albumin. Because of the intense thirst, the patient tends to drink 2 to 20 L of fluid daily and craves cold water. In adults, the onset of DI may be insidious or abrupt.

The disease cannot be controlled by limiting fluid intake, because the high-volume loss of urine continues even without fluid replacement. Attempts to restrict fluids cause the patient to experience an insatiable craving for fluid and to develop hypernatremia and severe dehydration.

Assessment and Diagnostic Findings

The fluid deprivation test is carried out by withholding fluids for 8 to 12 hours or until 3% to 5% of the body weight is lost. The patient is weighed frequently during the test. Plasma and urine osmolality studies are performed at the beginning and end of the test. The inability to increase the specific gravity and osmolality of the urine is characteristic of DI. The patient continues to excrete large volumes of urine with low specific gravity and experiences weight loss, increasing serum osmolality, and elevated serum sodium levels. The patient's condition needs to be monitored frequently during the test, and the test is terminated if tachycardia, excessive weight loss, or hypotension develops.

Other diagnostic procedures include concurrent measurements of plasma levels of ADH and plasma and urine osmolality as well as a trial of

desmopressin therapy and intravenous (IV) infusion of hypertonic saline solution. If the diagnosis is confirmed and the cause (e.g., head injury) is not obvious, the patient is carefully assessed for tumors that may be causing the disorder.

Medical Management

The objectives of therapy are to replace ADH (which is usually a long-term therapeutic program), ensure adequate fluid replacement, and identify and correct the underlying intracranial pathology. Nephrogenic causes require different management approaches (Hollar & Silverstein, 2019).

Pharmacologic Therapy

Desmopressin, a synthetic vasopressin without the vascular effects of natural ADH, is the drug of choice for central DI. The drug may be given orally or intranasally (Norris, 2019). Vasopressin causes vasoconstriction; therefore, it must be used cautiously in patients with coronary artery disease. Chlorpropamide and thiazide diuretics are also used in mild forms of the disease because they potentiate the action of vasopressin but are used with caution due to the risk for hypoglycemia (Norris, 2019).

If the DI is renal in origin, the previously described treatments are ineffective. Thiazide diuretics, mild salt depletion, and prostaglandin inhibitors (e.g., indomethacin and aspirin) are used to treat the nephrogenic form of DI.

Nursing Management

Ongoing physical assessment and patient education are the pillars of skilled nursing management of the patient with a diagnosis of DI. Initially, the nurse reviews the patient history and physical assessment and monitors for clinical manifestations of dehydration. Severe dehydration can lead to decreased cardiac output and, therefore, decreased perfusion of the vital organs, specifically the brain and kidneys. Ongoing monitoring of vital signs as well as intake and output (I&O) is essential. The nurse is responsible to educate the patient, family, and other caregivers about follow-up care, prevention of complications, and emergency measures. Specific verbal and written instructions should include the dose, actions, side effects, and administration of all medications and the signs and symptoms of hyponatremia. The nurse should demonstrate and observe a return demonstration of medication administration to ensure that the patient received the prescribed dosage. The patient should be advised to wear a medical identification bracelet and carry required medication and information about DI at all times.

Syndrome of Inappropriate Antidiuretic Hormone Secretion

The **syndrome of inappropriate antidiuretic hormone (SIADH)** results from a failure of the negative feedback system that regulates the release and inhibition of ADH (Norris, 2019). Patients with SIADH cannot excrete a dilute urine, retain fluids, and develop a sodium deficiency known as dilutional hyponatremia. SIADH is often of nonendocrine origin; for instance, the syndrome may occur in patients with bronchogenic carcinoma in which malignant lung cells synthesize and release ADH. SIADH has also occurred in patients with severe pneumonia, pneumothorax, and other disorders of the lungs, as well as malignant tumors that affect other organs (Norris, 2019).

Disorders of the central nervous system, such as head injury, brain surgery or tumor, and infection, are thought to produce SIADH by direct stimulation of the pituitary gland (Norris, 2019). Some medications (e.g., vincristine, phenothiazines, tricyclic antidepressants, thiazide diuretics) and nicotine have been implicated in SIADH; they either directly stimulate the pituitary gland or increase the sensitivity of renal tubules to circulating ADH.

Medical Management

SIADH is generally self-limiting and treatment is focused on eliminating the underlying cause, if possible, and restricting fluid intake (Parham & Silverstein, 2019). Because retained water is excreted slowly through the kidneys, the extracellular fluid volume contracts and the serum sodium concentration gradually increases toward normal. Diuretic agents such as furosemide may be used along with fluid restriction. In severe hyponatremia sometimes a hypertonic NaCl (3%) may be prescribed and administered IV (Norris, 2019).

Nursing Management

Close monitoring of fluid I&O, daily weight, urine and blood chemistries, and neurologic status is indicated for the patient at risk for SIADH. Supportive measures and explanations of procedures and treatments assist the patient in managing this disorder.

THE THYROID GLAND

The thyroid gland—the largest endocrine gland—is a butterfly-shaped organ located in the lower neck, anterior to the trachea (Fig. 45-3). It consists of two lateral lobes connected by an isthmus. The gland is about 5 cm long and 3 cm wide and weighs about 30 g. The blood flow to the thyroid is very high (about 5 mL/min per gram of thyroid tissue), approximately five times the blood flow

to the liver. The thyroid gland produces three hormones: **thyroxine (T₄)**, **triiodothyronine (T₃)**, and **calcitonin**. Thyroxine and triiodothyronine are needed by all body cells for metabolism (Moore, 2018).

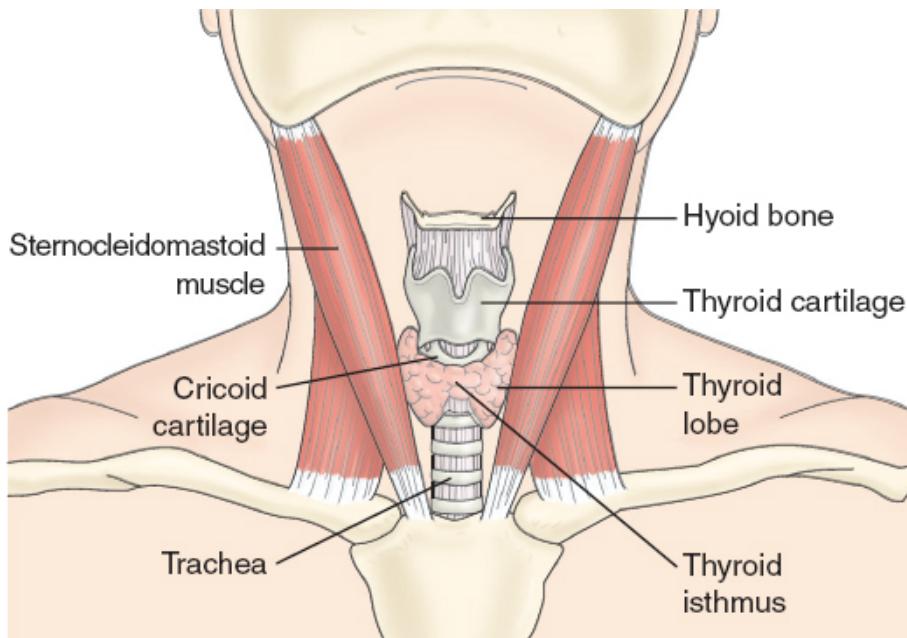


Figure 45-3 • The thyroid gland and surrounding structures.

Anatomic and Physiologic Overview

Various hormones and chemicals are responsible for normal thyroid function. Key among them are thyroid hormone, calcitonin, and iodine.

Thyroid Hormone

Thyroid hormone is comprised of T₄ and T₃, two separate hormones produced by the thyroid gland. Both are amino acids that contain iodine molecules bound to the amino acid structure; T₄ contains four iodine atoms in each molecule, and T₃ contains three. These hormones are synthesized and stored bound to proteins in the cells of the thyroid gland until needed for release into the bloodstream. Three thyroid-binding hormones—thyroxine-binding globulin (TBG), transthyretin (formerly known as thyroid-binding prealbumin), and albumin—bind and transport T₃ and T₄ (Norris, 2019).

Synthesis of Thyroid Hormone

Iodine is essential to the thyroid gland for synthesis of its hormones. The major use of iodine in the body is by the thyroid, and the major derangement in

iodine deficiency is alteration of thyroid function. Iodide is ingested in the diet and absorbed into the blood in the GI tract. The thyroid gland is extremely efficient at taking up iodide from the blood and concentrating it within the cells, where iodide ions are converted to iodine molecules, which react with tyrosine (an amino acid) to form the thyroid hormones (Norris, 2019).

Regulation of Thyroid Hormone

The secretion of T_3 and T_4 by the thyroid gland is controlled by TSH (also called *thyrotropin*) from the anterior pituitary gland. TSH controls the rate of thyroid hormone release through a negative feedback mechanism. In turn, the level of thyroid hormone in the blood determines the release of TSH. If the thyroid hormone concentration in the blood decreases, the release of TSH increases, which causes increased output of T_3 and T_4 . The term **euthyroid** refers to thyroid hormone production that is normal.

TRH, secreted by the hypothalamus, exerts a modulating influence on the release of TSH from the pituitary. Environmental factors, such as a decrease in temperature, may lead to increased secretion of TRH, resulting in elevated secretion of thyroid hormones. Figure 45-4 shows the hypothalamic–pituitary–thyroid axis, which regulates thyroid hormone production.

Function of Thyroid Hormone

The main function of thyroid hormone is to control cellular metabolic activity. T_4 , a relatively weak hormone, maintains body metabolism in a steady state. T_3 is about five times as potent as T_4 and has a more rapid metabolic action. These hormones accelerate metabolic processes by increasing the level of specific enzymes that contribute to oxygen consumption and altering the responsiveness of tissues to other hormones. The thyroid hormones influence cell replication, are important in brain development, and are necessary for normal growth. Thyroid hormones affect virtually every major organ system and tissue function, including the basal metabolic rate, tissue thermogenesis, serum cholesterol levels, and vascular resistance (Norris, 2019).

Calcitonin

Calcitonin, or thyrocalcitonin, is another important hormone secreted by the thyroid gland. The hormone is secreted in response to high plasma levels of calcium, and it reduces the plasma level of calcium by increasing its deposition in bone.

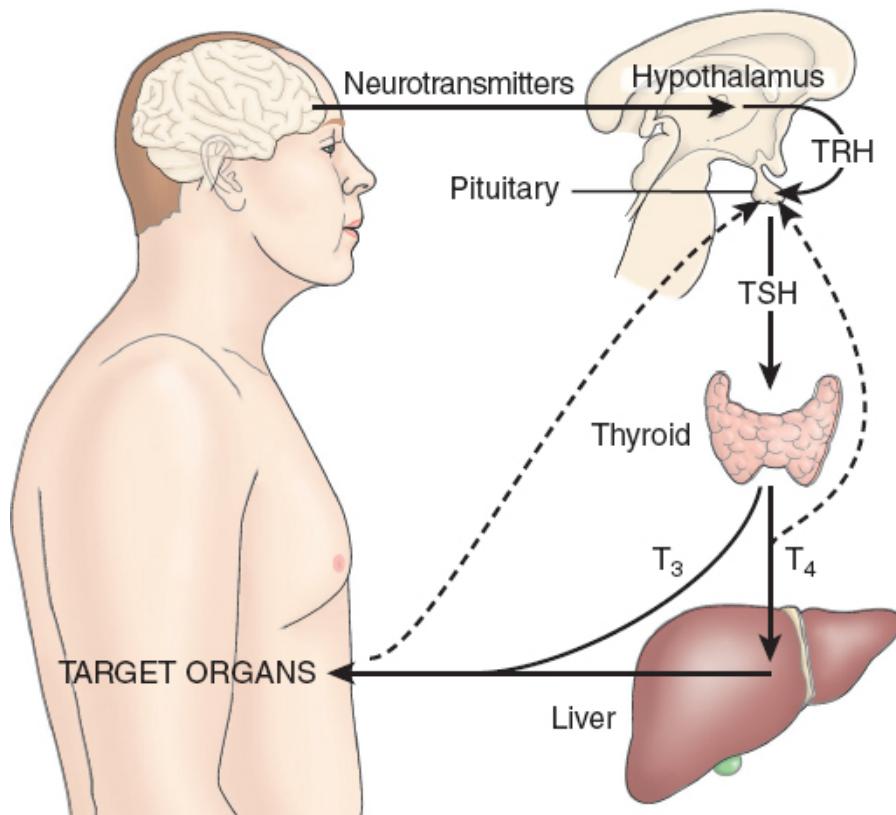


Figure 45-4 • The hypothalamic–pituitary–thyroid axis. Thyroid-releasing hormone (TRH) from the hypothalamus stimulates the pituitary gland to secrete thyroid-stimulating hormone (TSH). TSH stimulates the thyroid to produce thyroid hormone (triiodothyronine [T_3] and thyroxine [T_4]). High circulating levels of T_3 and T_4 inhibit further TSH secretion and thyroid hormone production through a negative feedback mechanism (*dashed lines*).

Pathophysiology

Congenital hypothyroidism, which occurs when there is inadequate secretion of thyroid hormone during fetal and neonatal development, results in intellectual disability and stunted physical growth because of general depression of metabolic activity (Norris, 2019). In adults, hypothyroidism manifests as lethargy, slow mentation, weight gain, constipation, cold intolerance, and generalized slowing of body functions (Singh & Clutter, 2019).

Hyperthyroidism (oversecretion of thyroid hormones) is manifested by a greatly increased metabolic rate. Many of the other characteristics of hyperthyroidism result from the increased response to circulating catecholamines (epinephrine and norepinephrine).

Oversecretion of thyroid hormones is usually associated with an enlarged thyroid gland known as a **goiter**. Goiter also commonly occurs with iodine deficiency. In this latter condition, lack of iodine results in low levels of circulating thyroid hormones, which causes increased release of TSH; the elevated TSH causes overproduction of thyroglobulin (a precursor of T₃ and T₄) and hypertrophy of the thyroid gland.

Assessment

Physical Examination

The thyroid gland is inspected and palpated routinely in all patients. Inspection begins with identification of landmarks. The lower neck region between the sternocleidomastoid muscles is inspected for swelling or asymmetry. The patient is instructed to extend the neck slightly and swallow. Thyroid tissue rises normally with swallowing. The thyroid is then palpated for size, shape, consistency, symmetry, and the presence of tenderness.

The health care provider may examine the thyroid from an anterior or a posterior position. In the posterior position, both hands encircle the patient's neck. The thumbs rest on the nape of the neck, while the index and middle fingers palpate for the thyroid isthmus and the anterior surfaces of the lateral lobes. When palpable, the isthmus is perceived as firm and of a rubber-band consistency.

The left lobe is examined by positioning the patient so that the neck flexes slightly forward and to the left. The thyroid cartilage is then displaced to the left with the fingers of the right hand. This maneuver displaces the left lobe deep into the sternocleidomastoid muscle, where it can be more easily palpated. The left lobe is then palpated by placing the left thumb deep into the posterior area of the sternocleidomastoid muscle, while the index and middle fingers exert opposite pressure in the anterior portion of the muscle. Having the patient swallow during the maneuver may assist the examiner to locate the thyroid as it ascends in the neck. The procedure is reversed to examine the right lobe. The isthmus is the only portion of the thyroid that is normally palpable. If a patient has a very thin neck, two thin, smooth, nontender lobes may also be palpable.

If palpation discloses an enlarged thyroid gland, both lobes are auscultated using the diaphragm of the stethoscope. Auscultation identifies the localized audible vibration of a bruit. This is indicative of increased blood flow through the thyroid gland associated with hyperthyroidism and necessitates referral to a primary provider. Other abnormal findings that require referral for further evaluation may include a soft texture (Graves disease), firmness (Hashimoto thyroiditis or malignancy), and tenderness (thyroiditis) (Jensen, 2019).

Diagnostic Evaluation

Assessment measures in addition to palpation and auscultation include thyroid function tests, such as laboratory measurement of thyroid hormones, thyroid scanning, biopsy, and ultrasonography. The most widely used tests are serum immunoassay for TSH and free T₄. Free T₄ levels correlate with metabolic status; they are elevated in hyperthyroidism and decreased in hypothyroidism (Fischbach & Fischbach, 2018). Ultrasound, CT, and MRI may be used to clarify or confirm the results of other diagnostic studies.

Thyroid Tests

Serum Thyroid-Stimulating Hormone

Measurement of the serum TSH concentration is the primary screening test of thyroid function. The ability to detect minute changes in serum TSH makes it possible to distinguish subclinical thyroid disease from euthyroid states in patients with low or high normal values. Measurement of TSH is also used for monitoring thyroid hormone replacement therapy and for differentiating between disorders of the thyroid gland itself and disorders of the pituitary or hypothalamus.

The American Thyroid Association recommends that pregnant women be screened for thyroid disease (Alexander, Pearce, Brent, et al., 2017); however, there is lack of consensus among professional organizations regarding routine screening of adults for thyroid disease. The US Preventive Services Task Force (USPSTF) does not recommend routine screening of adults (USPSTF, 2019).

Serum Free T₄

Serum free T₄ is a direct measurement of free (unbound) thyroxine, the only metabolically active fraction of T₄. The range of free T₄ in serum is normally 0.7 to 2.0 ng/dL (10 to 26 pmol/L) (Fischbach & Fischbach, 2018). When measured by the dialysis method, free T₄ is not affected by variations in protein binding and is the procedure of choice for monitoring the changes in T₄ secretion during treatment for hyperthyroidism.

Serum T₃ and T₄

Measurement of total T₃ or T₄ includes protein-bound and free hormone levels that occur in response to TSH secretion. T₄ is 70% bound to TBG; T₃ is bound less firmly. Only 0.03% of T₄ and 0.3% of T₃ are unbound. Serious systemic illnesses, medications (e.g., oral contraceptives, corticosteroids, carbamazepine, salicylates), and protein wasting as a result of nephrosis or the use of androgens may interfere with accurate test results. Normal range for T₄

is 5.4 to 11.5 $\mu\text{g}/\text{dL}$ (57 to 148 nmol/L) (Fischbach & Fischbach, 2018). Although serum T_3 and T_4 levels generally increase or decrease together, the T_3 level appears to be a more accurate indicator of hyperthyroidism or severity of the disorder, as T_4 levels are often within normal range. The normal range for serum T_3 is 260 to 480 pg/dL (4.0 to 7.4 pmol/L) (Fischbach & Fischbach, 2018).

T₃ Resin Uptake Test

The T_3 resin uptake test is an indirect measure of unsaturated TBG. Its purpose is to determine the amount of thyroid hormone bound to TBG and the number of available binding sites. This provides an index of the amount of thyroid hormone already present in the circulation. Normally, TBG is not fully saturated with thyroid hormone, and additional binding sites are available to combine with radioiodine-labeled T_3 added to the blood specimen. The normal T_3 uptake value is 25% to 35% (relative uptake fraction, 0.25 to 0.35), which indicates that about one third of the available sites of TBG are occupied by thyroid hormone. If the number of free or unoccupied binding sites is low, as in hyperthyroidism, the T_3 uptake is greater than 35% (0.35). If the number of available sites is high, as occurs in hypothyroidism, the test result is less than 25% (0.25).

T_3 uptake is useful in evaluating thyroid hormone levels in patients who have received diagnostic or therapeutic doses of iodine. The test results may be altered by the use of estrogens, androgens, salicylates, phenytoin, anticoagulants, or corticosteroids (Fischbach & Fischbach, 2018).

Thyroid Antibodies

Autoimmune thyroid diseases include both hypo- and hyperthyroid conditions. Results of testing by immunoassay techniques for antithyroid antibodies are positive in chronic autoimmune thyroid disease (90%), Hashimoto thyroiditis (100%), Graves disease (80%), and other organ-specific autoimmune diseases, such as systemic lupus erythematosus (SLE) and rheumatoid arthritis. Antithyroid antibody titers are normally present in 5% to 10% of the population and increase with age.

Radioactive Iodine Uptake

The radioactive iodine uptake test measures the rate of iodine uptake by the thyroid gland. The patient is given a tracer dose of iodine 123 (^{123}I) or another radionuclide, and a count is made over the thyroid gland with a scintillation counter, which detects and counts the gamma rays released from the breakdown of ^{123}I in the thyroid. The radioactive iodine uptake test is a simple test with reliable results. The test measures the proportion of the given dose

that is present in the thyroid gland at a specific time after its administration. Since the test is affected by the patient's intake of iodide or thyroid hormone, a careful preliminary clinical history is essential in evaluating results. Normal values vary from one geographic region to another and with the intake of iodine. Patients with hyperthyroidism exhibit a high uptake of the ^{123}I (in some patients, as high as 90%), whereas patients with hypothyroidism exhibit a very low uptake.

Fine-Needle Aspiration Biopsy

The use of a small-gauge needle to sample the thyroid tissue for biopsy is a safe and accurate method of detecting malignancy and is often the initial test for evaluation of thyroid masses. Results are reported as benign, malignant, suspicious, or nondiagnostic/insufficient (Ross, Cooper, & Mulder, 2019). Within the malignancy category, masses are reported as a follicular neoplasm or a follicular lesion.

Thyroid Scan, Radioscan, or Scintiscan

In a thyroid scan, a scintillation detector or gamma camera moves back and forth across the area to be studied in a series of parallel tracks, and a visual image is made of the distribution of radioactivity in the area being scanned. The most commonly used isotopes of iodine are ^{123}I and ^{131}I (Fischbach & Fischbach, 2018).

Scans are helpful in determining the location, size, shape, and anatomic function of the thyroid gland, particularly when thyroid tissue is substernal or large (Fischbach & Fischbach, 2018). Identifying areas of increased function ("hot" areas) or decreased function ("cold" areas) can assist in diagnosis. Although most areas of decreased function do not represent malignancies, lack of function increases the likelihood of malignancy, particularly if only one nonfunctioning area is present. Scanning of the entire body, to obtain the total body profile, may be carried out in a search for a functioning thyroid metastasis (i.e., a lesion that produces thyroid hormones).

Serum Thyroglobulin

Thyroglobulin (Tg) can be measured reliably in the serum by radioimmunoassay. Clinically, it is used to detect persistence or recurrence of thyroid carcinoma.

Nursing Implications

Since thyroid tests involve the use of iodine, determining if the patient has any allergies to iodine or is taking medications that contain iodine is essential. The relationship between having an allergy to shellfish and having an allergy to

iodine is a long held belief; however, an allergy to shellfish is due to specific proteins in the shellfish and not iodine (American College of Allergy, Asthma and Immunology, 2019). Patients should be asked if they have had a reaction to iodine previously and to shellfish so that the radiologist can determine what precautions need to be taken, if any (American College of Allergy, Asthma and Immunology, 2019). Patients should be asked about obvious sources of iodine-containing medications such as contrast agents and those used to treat thyroid disorders such as radioactive iodine. They should also be asked whether they eat kelp or seaweed. Numerous medications may also affect test results because they affect the thyroid. Chart 45-2 gives a list of select medications that may interfere with accurate testing of thyroid gland function (Fischbach & Fischbach, 2018). This information should be documented in the patient's electronic health record (EHR) and communicated clearly to personnel conducting the test.

Chart 45-2 PHARMACOLOGY

Select Medications That May Alter Thyroid Test Results

- amiodarone
- aspirin
- cimetidine
- diazepam
- estrogens
- furosemide
- glucocorticoids
- heparin
- lithium
- phenytoin and other anticonvulsants
- propranolol

Adapted from Morton, P. G., & Fontaine, D. K. (2018). *Critical care nursing: A holistic approach* (10th ed.). Philadelphia, PA: Wolters Kluwer.

Hypothyroidism

Hypothyroidism results from suboptimal levels of thyroid hormone. Thyroid deficiency can affect all body functions and can range from mild, subclinical forms to **myxedema** (severe deficiency discussed later), an advanced life-threatening form. The most common cause of hypothyroidism in adults is autoimmune thyroiditis (Hashimoto disease), in which the immune system attacks the thyroid gland. Symptoms of hyperthyroidism may later be followed

by those of hypothyroidism and myxedema. Hypothyroidism also commonly occurs in patients with previous hyperthyroidism that has been treated with radioiodine or antithyroid medications or **thyroidectomy** (surgical removal of all or part of the thyroid gland). Testing of thyroid function is recommended for all patients who receive radiation therapy to the neck. See Chart 45-3 for other causes of hypothyroidism.

More than 95% of patients with hypothyroidism have primary or thyroidal hypothyroidism, which refers to dysfunction of the thyroid gland itself. If the cause of the thyroid dysfunction is failure of the pituitary gland, the hypothalamus, or both, the hypothyroidism is known as central hypothyroidism. If the cause is entirely a pituitary disorder, it may be referred to as pituitary or secondary hypothyroidism. If the cause is a disorder of the hypothalamus resulting in inadequate secretion of TSH due to decreased stimulation of TRH, it is referred to as hypothalamic or tertiary hypothyroidism. If thyroid deficiency is present at birth, it is referred to as neonatal hypothyroidism. In such instances, the mother may also have thyroid deficiency. The term *myxedema* refers to the accumulation of mucopolysaccharides in subcutaneous and other interstitial tissues. Although myxedema occurs in long-standing hypothyroidism, the term is used appropriately only to describe the extreme symptoms of severe hypothyroidism (Chaker, Bianco, Janklaas, et al., 2017).

Chart 45-3

Causes of Hypothyroidism

- Autoimmune disease (Hashimoto thyroiditis, post-Graves disease)
- Atrophy of thyroid gland with aging
- Infiltrative diseases of the thyroid (amyloidosis, scleroderma, lymphoma)
- Iodine deficiency, iodine excess, and iodine compounds
- Medications (e.g., Lithium)
- Radioactive iodine (^{131}I)
- Therapy for hyperthyroidism
- Thyroidectomy
- Radiation to head and neck in treatment for head and neck cancers, lymphoma

Clinical Manifestations

Presenting clinical manifestations in adults frequently reflect the decrease in metabolism resulting from the decrease in thyroid function. Clinical manifestations include complaints of fatigue and lethargy that may interfere with activities of daily living, weight gain without an increased intake of calories, cold intolerance, dry skin, and, in some patients, a deepening of the

voice. Other clinical manifestations are related to gender, age, and duration of the decrease in thyroid function (see Fig. 45-5). These include cardiovascular-related manifestations such as bradycardia and changes in electrical conduction of the heart which will be noted on the electrocardiogram (ECG). In women, changes in the menstrual cycle will be noted (Chaker et al., 2017).

Severe hypothyroidism results in a subnormal body temperature and pulse rate. The patient usually begins to gain weight even without an increase in food intake, although they may be cachectic. The skin becomes thickened because of an accumulation of mucopolysaccharides in the subcutaneous tissues. The hair thins and falls out, and the face becomes expressionless and masklike. The patient often complains of being cold even in a warm environment.

At first, the patient may be irritable and may complain of fatigue, but as the condition progresses, the emotional responses are subdued. The mental processes become dulled, and the patient appears apathetic. Speech is slow, the tongue enlarges, the hands and feet increase in size, and deafness may occur. The patient frequently reports constipation.

Advanced hypothyroidism may produce personality and cognitive changes characteristic of dementia. Inadequate ventilation and sleep apnea can occur with severe hypothyroidism. Pleural effusion, pericardial effusion, and respiratory muscle weakness may also occur (Chaker et al., 2017).

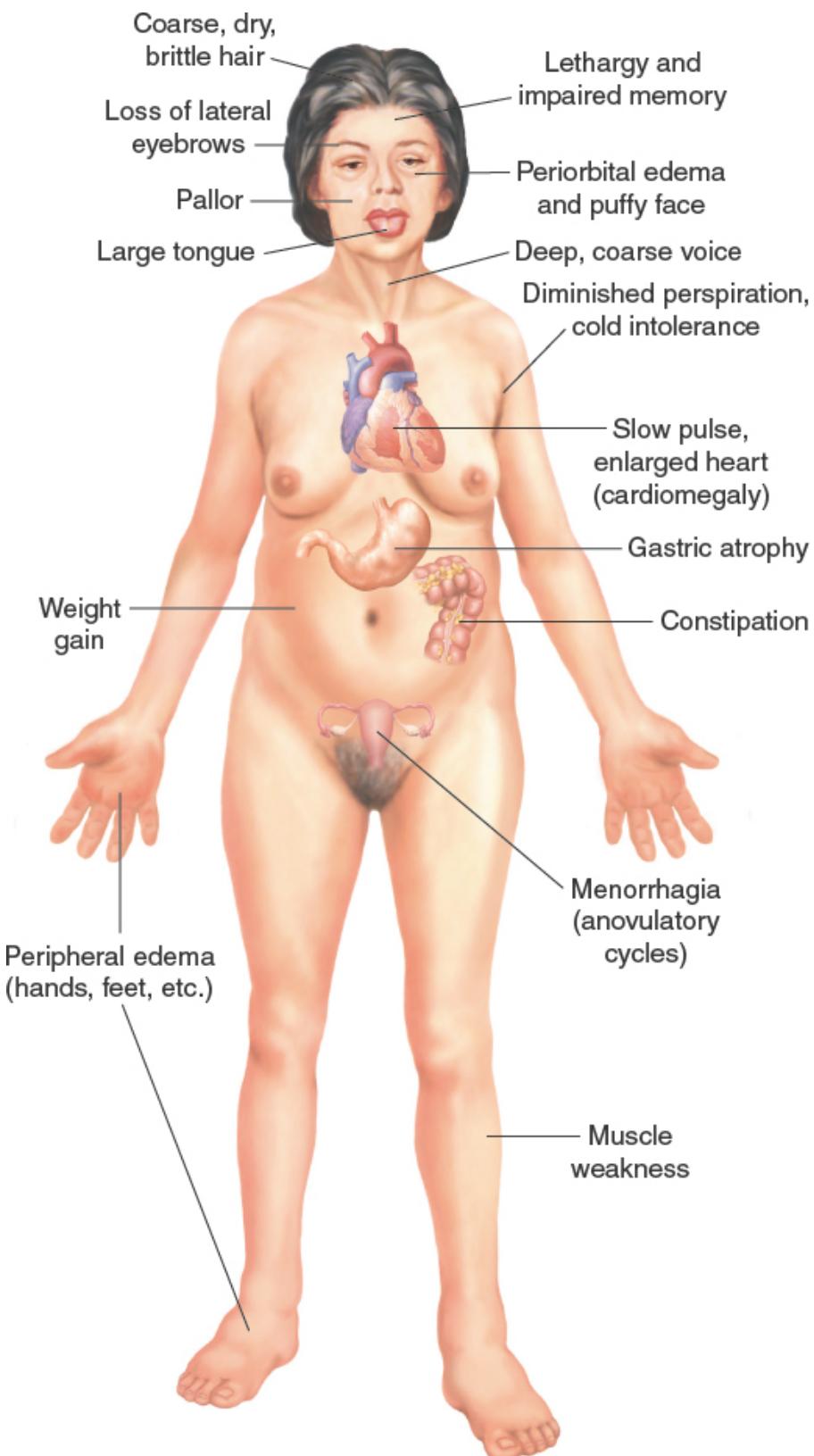


Figure 45-5 • Clinical manifestations of hypothyroidism. Reprinted with permission from Norris, T. L. (2019). *Porth's pathophysiology*:

Concepts of altered health states (10th ed., Fig. 41.5). Philadelphia, PA: Wolters Kluwer.

Severe hypothyroidism is associated with an elevated serum cholesterol level, atherosclerosis, coronary artery disease, and poor left ventricular function. The patient with advanced hypothyroidism is hypothermic and abnormally sensitive to sedative, opioid, and anesthetic agents, which must be given with extreme caution.

Patients with unrecognized hypothyroidism who are undergoing surgery are at increased risk for intraoperative hypotension, postoperative heart failure, and altered mental status.

Myxedema coma is a rare life-threatening condition and a decompensated state of severe hypothyroidism in which the patient is hypothermic and unconscious. This condition may develop with undiagnosed hypothyroidism and may be precipitated by infection or other systemic disease or by use of sedatives or opioid analgesic agents. Patients may also experience myxedema coma if they forget to take their thyroid replacement medication. The condition occurs most often among older women in the winter months and appears to be precipitated by cold. However, the disorder can affect any age group.

In myxedema coma, the patient may initially show signs of depression, diminished cognitive status, lethargy, and somnolence (Chaker et al., 2017).

Increasing lethargy may progress to stupor. The patient's respiratory drive is depressed, resulting in alveolar hypoventilation, progressive carbon dioxide retention, narcosis, and coma. In addition, patients with myxedema coma can also exhibit hyponatremia, hypoglycemia, hypoventilation, hypotension, bradycardia, and hypothermia. These symptoms, along with cardiovascular collapse and shock, require aggressive and intensive supportive and hemodynamic therapy if the patient is to survive. Although there has been a decline in mortality rates over the past two decades due to early intervention and improved therapies, the mortality rate remains at 40% despite treatment; proper diagnosis and prompt treatment is essential (Eledrisi, 2018).

Medical Management

The objectives in the management of hypothyroidism are to restore a normal metabolic state by replacing the missing hormone, as well as prevention of disease progression and complications.

Pharmacologic Therapy

Synthetic levothyroxine is the drug of choice for the treatment of hypothyroidism (Drake, 2018). The general range is 75 to 150 mcg per day and treatment is generally started at the lower dose and titrated slowly until desired levels of serum TSH concentration are achieved (Singh & Clutter,

2019). Older adult patients generally require a lower dose; normal TSH levels are often achieved with 50 mcg per day (Singh & Clutter, 2019). Some patients on thyroid hormone replacement may complain of continued clinical manifestations despite normal TSH levels (Chaker et al., 2017).

Prevention of Cardiac Dysfunction

Any patient who has had hypothyroidism for a long period usually has associated elevated serum cholesterol, atherosclerosis, and coronary artery disease. As long as metabolism is subnormal and the tissues (including the myocardium) require relatively little oxygen, a reduction in blood supply is tolerated without overt symptoms of coronary artery disease. When thyroid hormone is given, the oxygen demand increases, but oxygen delivery cannot be increased unless, or until, the atherosclerosis improves. This occurs very slowly, if at all. The occurrence of angina and acute coronary syndrome (see Chapter 23) is the signal that the oxygen needs of the myocardium exceed its blood supply. Angina or arrhythmias can occur when thyroid replacement is initiated because thyroid hormones enhance the cardiovascular effects of catecholamines.



Quality and Safety Nursing Alert

The nurse must monitor for signs and symptoms of cardiac dysfunction, which can occur in response to therapy in patients with severe, long-standing hypothyroidism or myxedema coma, especially during the early phase of treatment. Acute coronary syndrome must be aggressively treated to avoid morbid complications (e.g., myocardial infarction).

If angina or arrhythmias occur, thyroid hormone administration must be discontinued immediately. Later, when it can be resumed safely, it should be prescribed cautiously at a lower dosage and with close monitoring by the primary provider and the nurse.

Prevention of Medication Interactions

Oral thyroid hormones interact with many other medications. They increase the effect of warfarin and the cardiovascular effects of adrenergic agents (bronchodilators and vasopressors). In addition, the dosage of insulin and oral hypoglycemic medications used to treat diabetes may require adjustment. Caution is also needed in patients who are concomitantly taking estrogen, which may necessitate an increased dosage of the oral thyroid hormone. Absorption may be affected by any supplement or food that contains calcium, iron, magnesium or zinc (Vallerand & Sanoski, 2018).

Even in small IV doses, hypnotic and sedative agents may induce profound somnolence, lasting far longer than anticipated and leading to narcosis (stuporlike condition). Furthermore, they are likely to cause respiratory depression, which can easily be fatal because of decreased respiratory reserve and alveolar hypoventilation. The dose of these medications should be one half or one third of that typically prescribed for patients of similar age and weight with normal thyroid function.

Supportive Therapy

Severe hypothyroidism and myxedema coma require prompt, aggressive management to maintain vital functions. Arterial blood gases may be measured to determine carbon dioxide retention and to guide the use of assisted ventilation to combat hypoventilation. Oxygen saturation levels should be monitored using pulse oximetry. Fluids are given cautiously because of the danger of water intoxication. Passive rewarming with a blanket is recommended versus active rewarming such as application of external heat (e.g., heating pads). The latter should be avoided to prevent increased oxygen demands and hypotension.

Nursing Management

Nursing care of the patient with hypothyroidism and myxedema is summarized in the plan of nursing care in Chart 45-4. In patients with hypothyroidism, the effects of analgesic, sedative, and anesthetic agents are prolonged. The nurse should carefully monitor patients who are prescribed these agents for adverse effects. Older patients are at increased risk because of age-related changes in liver and renal function.

Chart 45-4  **PLAN OF NURSING CARE**

Care of the Patient with Hypothyroidism

NURSING DIAGNOSIS: Impaired breathing associated with depressed ventilation

GOAL: Improved respiratory status and maintenance of normal breathing pattern

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none">1. Assess respiratory rate, depth, pattern, pulse oximetry, and arterial blood gases.2. Encourage deep breathing, coughing, and the use of incentive spirometry.3. Verify with the provider orders to administer any hypnotic and sedative until euthyroid state achieved. If these medications are needed, monitor for adverse side effects.4. Maintain patient airway through suction and ventilator support if needed (see Chapter 19 for care of patients requiring mechanical ventilation).	<ol style="list-style-type: none">1. Identifies patient's baseline to monitor further changes and evaluate effectiveness of interventions.2. Prevents atelectasis and promotes adequate ventilation.3. Patients with hypothyroidism are susceptible to respiratory depression with the use of hypnotics and sedatives.4. The use of artificial airway and ventilator support may be necessary.	<ul style="list-style-type: none">• Shows improved respiratory status and normal respiratory rate, depth, and pattern• Takes deep breaths, coughs and uses incentive spirometry• Explains rationale for cautious use of medications• Maintains adequate oxygenation

NURSING DIAGNOSIS: Risk for impaired cardiac function associated with

altered metabolism

GOAL: Improved cardiac status and maintenance of adequate cardiac output

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none">1. Assess heart rate and rhythm and blood pressure2. Monitor serum cholesterol value and complaints of anginal pain.3. Monitor ECG for the presence of any arrhythmias, especially after thyroid hormone replacement therapy is initiated.	<ol style="list-style-type: none">1. Identifies patient's baseline to monitor further changes and evaluate effectiveness of interventions2. The presence of atherosclerosis and cardiac disease prior to onset of hypothyroidism/myxedema may contribute to decreased perfusion3. Initiation of thyroid therapy enhances the cardiovascular effects of catecholamines.	<ul style="list-style-type: none">• Shows improved cardiac status and maintenance of normal cardiac pattern• Reports free of anginal pain• Maintains normal sinus rhythm

NURSING DIAGNOSIS: Risk for impaired thermoregulation

GOAL: Maintenance of normal body temperature

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none">1. Provide extra layer of clothing or extra blanket.2. Avoid and discourage the use of external heat source (e.g., heating pads, electric or	<ol style="list-style-type: none">1. Minimizes heat loss2. Reduces risk of peripheral vasodilation and vascular collapse3. Detects decreased body temperature and onset of myxedema coma4. Increases patient's level of comfort and decreases	<ul style="list-style-type: none">• Experiences relief of discomfort and cold intolerance• Maintains baseline body temperature• Reports adequate feeling of

warming blankets).	further heat loss	warmth and lack of chilling
3. Monitor patient's body temperature and report decreases from patient's baseline value.		<ul style="list-style-type: none"> • Uses extra layer of clothing or extra blanket • Explains rationale for avoiding external heat source
4. Protect from exposure to cold and drafts.		

NURSING DIAGNOSIS: Acute confusion associated with altered cardiovascular and respiratory status and depression

GOAL: Improved thought process

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Orient patient to time, place, date, and events around them. 2. Provide stimulation through conversation and nonthreatening activities. 3. Explain to patient and family that change in cognitive and mental functioning is a result of disease process. 4. Monitor cognitive and mental processes and 	<ol style="list-style-type: none"> 1. Provides reality orientation to patient 2. Provides stimulation within patient's level of tolerance for stress 3. Reassures patient and family about the cause of the cognitive changes and that a positive outcome is possible with appropriate treatment 4. Permits evaluation of the effectiveness of treatment 	<ul style="list-style-type: none"> • Shows improved cognitive functioning • Identifies time, place, date, and events correctly • Responds appropriately when stimulated • Responds spontaneously as treatment becomes effective • Interacts spontaneously with family and environment • Explains that change in mental and cognitive

response of these to medication and other therapy.

processes is a result of disease processes

- Takes medications as prescribed to prevent decrease in cognitive processes

NURSING DIAGNOSIS: Activity intolerance associated with insufficient physiologic or psychological energy

Goal: Increased participation in activities and increased independence

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none">1. Promote independence in self-care activities.<ol style="list-style-type: none">a. Space activities to promote rest and exercise as tolerated.b. Assist with self-care activities when patient is fatigued.c. Provide stimulation through conversation and nonstressful activities.d. Monitor patient's response to increasing activities.	<ol style="list-style-type: none">1. Encouragement needed in patients with decreased energy Encourages activities while allowing time for adequate restb. Permits patient to participate to the extent possible in self-care activitiesc. Promotes interest without stressing the patientd. Guards against over- and underexertion by the patient	<ul style="list-style-type: none">• Participates in self-care activities• Reports increased level of energy• Displays interest and awareness in environment• Participates in activities and events in environment• Participates in family events and activities• Reports free of chest pain, increased fatigue, or breathlessness with increased level of activity

NURSING DIAGNOSIS: Constipation associated with diminished gastrointestinal peristalsis

GOAL: Return of normal bowel function

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Encourage increased fluid intake within limits of fluid restriction. 2. Provide foods high in fiber. 3. Instruct patient about foods with high water content. 4. Monitor bowel function. 5. Encourage increased mobility within patient's exercise tolerance. 6. Encourage patient to use laxatives and enemas sparingly. 	<ol style="list-style-type: none"> 1. Promotes passage of soft stools 2. Increases bulk of stools and more frequent bowel movements 3. Provides rationale for patient to increase fluid intake 4. Permits detection of constipation and return to normal bowel pattern 5. Promotes evacuation of the bowel 6. Minimizes patient's dependence on laxatives and enemas and encourages normal pattern of bowel evacuation 	<ul style="list-style-type: none"> • Reports normal bowel function • Identifies and consumes foods high in fiber • Drinks recommended amount of fluid each day • Participates in gradually increasing exercises • Uses laxatives as prescribed and avoids excessive dependence on laxatives and enemas

NURSING DIAGNOSIS: Lack of knowledge about the therapeutic regimen for lifelong thyroid replacement therapy

GOAL: Knowledge and acceptance of the prescribed therapeutic regimen

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Explain rationale for thyroid hormone replacement. 	<ol style="list-style-type: none"> 1. Provides rationale for patient to use thyroid hormone replacement as prescribed 2. Provides encouragement to patient by identifying 	<ul style="list-style-type: none"> • Describes therapeutic regimen correctly • Explains rationale for

2. Describe desired effects of medication to patient.	improved physical status and well-being that will occur with thyroid hormone therapy and return to a euthyroid state	thyroid hormone replacement
3. Assist patient to develop schedule and checklist to ensure self-administration of thyroid replacement.	3. Increases chances that medication will be taken as prescribed	• Identifies positive outcomes of thyroid hormone replacement
4. Describe signs and symptoms of over- and underdose of medication.	4. Serves as check for patient to determine if therapeutic goals are met	• Administers medication to self as prescribed
5. Explain the necessity for long-term follow-up to patient and family.	5. Increases likelihood that hypo- or hyperthyroidism will be detected and treated	• Identifies adverse side effects that should be reported promptly to primary provider: recurrence of symptoms of hypothyroidism and occurrence of symptoms of hyperthyroidism
		• Restates need for periodic/long-term follow-up visits to primary provider

COLLABORATIVE PROBLEM: Myxedema and myxedema coma**GOAL:** Evidence of progression to pre-coma baseline without incurring additional complications

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none">1. Monitor patient for increasing severity of signs and symptoms of hypothyroidism:<ol style="list-style-type: none">a. Decreased level of consciousnessb. Decreased vital signs (blood pressure, respiratory rate, temperature, pulse rate)c. Increasing difficulty in awakening or arousing patient2. Assist in ventilator support if respiratory depression and failure occur.3. Administer prescribed medications (e.g., thyroxine) with extreme caution.4. Turn and reposition patient at least every 2 hours.5. Avoid the use of hypnotic,	<ol style="list-style-type: none">1. Extreme hypothyroidism may lead to myxedema, myxedema coma, and slowing of all body systems if untreated2. Ventilator support is necessary to maintain adequate oxygenation and maintenance of airway3. The slow metabolism and atherosclerosis of myxedema may result in angina with administration of thyroxine4. Minimizes risks associated with immobility5. Altered metabolism of these agents greatly increases the risks of their use in myxedema	<ul style="list-style-type: none">• Exhibits reversal of myxedema and myxedema coma• Responds appropriately to questions and surroundings• Vital signs return to normal or near-normal ranges• Respiratory status improves with adequate spontaneous ventilatory effort• Reports free of angina or other indicators of cardiac insufficiency• Experiences minimal or no complications caused by immobility

sedative and analgesic agents.



Quality and Safety Nursing Alert

Medications are given to the patient with hypothyroidism with extreme caution because of the potential for altered metabolism and excretion, as well as depressed metabolic rate and respiratory status.

Promoting Home, Community-Based, and Transitional Care



Educating Patients About Self-Care

The patient and family require education and support to manage this complex disorder at home. Oral and written instructions should be provided regarding the following:

- The importance of life-long therapy and the need to take thyroid medication everyday
- Desired actions and side effects of medications
- Correct medication administration (“Take first thing in the morning with a full glass of water on an empty stomach.”)
- Importance of continuing to take the medications as prescribed even after symptoms improve
- When to seek medical attention
- Importance of nutrition and diet to promote weight loss and normal bowel patterns
- Importance of periodic follow-up testing

The patient and family should be educated that the symptoms observed during the course of the disorder will disappear with effective treatment (see Chart 45-5).

Continuing and Transitional Care

If indicated, a referral is made for home, community-based or transitional care. The nurse monitors the patient's recovery and ability to cope with changes, and assesses the patient's physical and cognitive status and the patient's and family's understanding of previous education. The nurse documents and reports to the patient's primary provider subtle signs and symptoms that may indicate either inadequate or excessive thyroid hormone.

Chart 45-5**HOME CARE CHECKLIST****The Patient with Hypothyroidism (Myxedema)**

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

- State the impact of hypothyroidism and treatment on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- State the need to avoid extreme cold temperature until condition is stable.
- State precipitating factors and interventions for complications (hyperthyroidism, myxedema coma).
- State the continuing potential effects of hypothyroidism on the body.
- State the potential for menstrual irregularities and potential for pregnancy in women.
- State the importance of avoiding infection.
- Relate how to reach primary provider with questions or complications.
- State time and date of follow-up medical appointments, therapy, and testing.
- Identify sources of support (e.g., friends, relatives, faith community).
- Identify the contact details for support services for patients and their caregivers/families.
- Identify the need for health promotion, disease prevention, and screening activities.

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

**Gerontologic Considerations**

The prevalence of hypothyroidism increases with age, most often among women (Calsolaro, Niccolai, Pasqualetti, et al., 2019). The higher prevalence of hypothyroidism among older adults may be related to age-related alterations in immune function and complicated by multiple comorbidities.

Most patients with primary hypothyroidism present with long-standing mild to moderate hypothyroidism. Subclinical disease is common among older women and can be asymptomatic or mistaken for other medical conditions. Subtle symptoms of hypothyroidism, such as fatigue, muscle aches, and mental confusion, may be attributed to the normal aging process by patients, families, and health care providers; therefore, these symptoms require close attention (Calsolaro et al., 2019). In addition, signs and symptoms of

hypothyroidism in older adults are often atypical, and manifestations of hypothyroidism and hyperthyroidism may blur. Patients may have few or no symptoms until dysfunction is severe. Depression, apathy, and decreased mobility or activity may be the major initial symptoms and may be accompanied by significant weight loss. Constipation affects one fourth of older patients.

In those with mild to moderate hypothyroidism, thyroid hormone replacement is individually tailored and must be started with low dosages and increased gradually to prevent serious cardiovascular side effects (Calsolaro et al., 2019). Angina, for example, may occur with rapid thyroid replacement in the presence of coronary artery disease secondary to the hypothyroid state. Heart failure and tachyarrhythmias may worsen during the transition from the hypothyroid state to the normal metabolic state. Dementia may become more apparent during early thyroid hormone replacement in older patients with concomitant dementia.

Older patients with severe hypothyroidism and atherosclerosis may become confused and agitated if their metabolic rate is increased too quickly. Marked clinical improvement follows the administration of hormone replacement; such medication must be continued for life, even though signs of hypothyroidism disappear within 3 to 12 weeks.

Older patients require periodic follow-up monitoring of serum TSH levels, because poor adherence with therapy may occur or the patient may take the medications erratically. A careful history can identify the need for further education about the importance of the medication.

Hyperthyroidism

Hyperthyroidism, a common endocrine disorder, is a form of **thyrotoxicosis** resulting from an excessive synthesis and secretion of endogenous or exogenous thyroid hormones by the thyroid (Norris, 2019). The most common causes are Graves disease, toxic multinodular goiter, and toxic adenoma. Other causes include **thyroiditis** (inflammation of the thyroid gland) and excessive ingestion of thyroid hormone.

Graves disease is an autoimmune disorder that results from an excessive output of thyroid hormones caused by abnormal stimulation of the thyroid gland by circulating immunoglobulins. This disease affects women eight times more frequently than men, with onset usually between the second and fourth decades. The disorder may appear after an emotional shock, stress, or an infection, but the exact significance of these relationships is not understood (Norris, 2019).

Clinical Manifestations

Patients with hyperthyroidism exhibit a characteristic group of signs and symptoms (see Fig. 45-6). Clinical manifestations are related to the increase in metabolic rate and increased oxygen consumption. The patient may appear anxious, seem restless and irritable, and exhibit fine tremors of the hands. The patient will be tachycardic and complain of palpitations. Heat intolerance will be noted with increased perspiration. Additional clinical manifestations include an increase in appetite, diarrhea, weight loss, and thin skin. Patients with Graves disease may present with exophthalmos and may exhibit reduced blinking and lid retraction. Treatment may not reverse ocular manifestations (Medford, 2019). Women experience changes in menstruation including oligomenorrhea (Lee & Khadri, 2018).

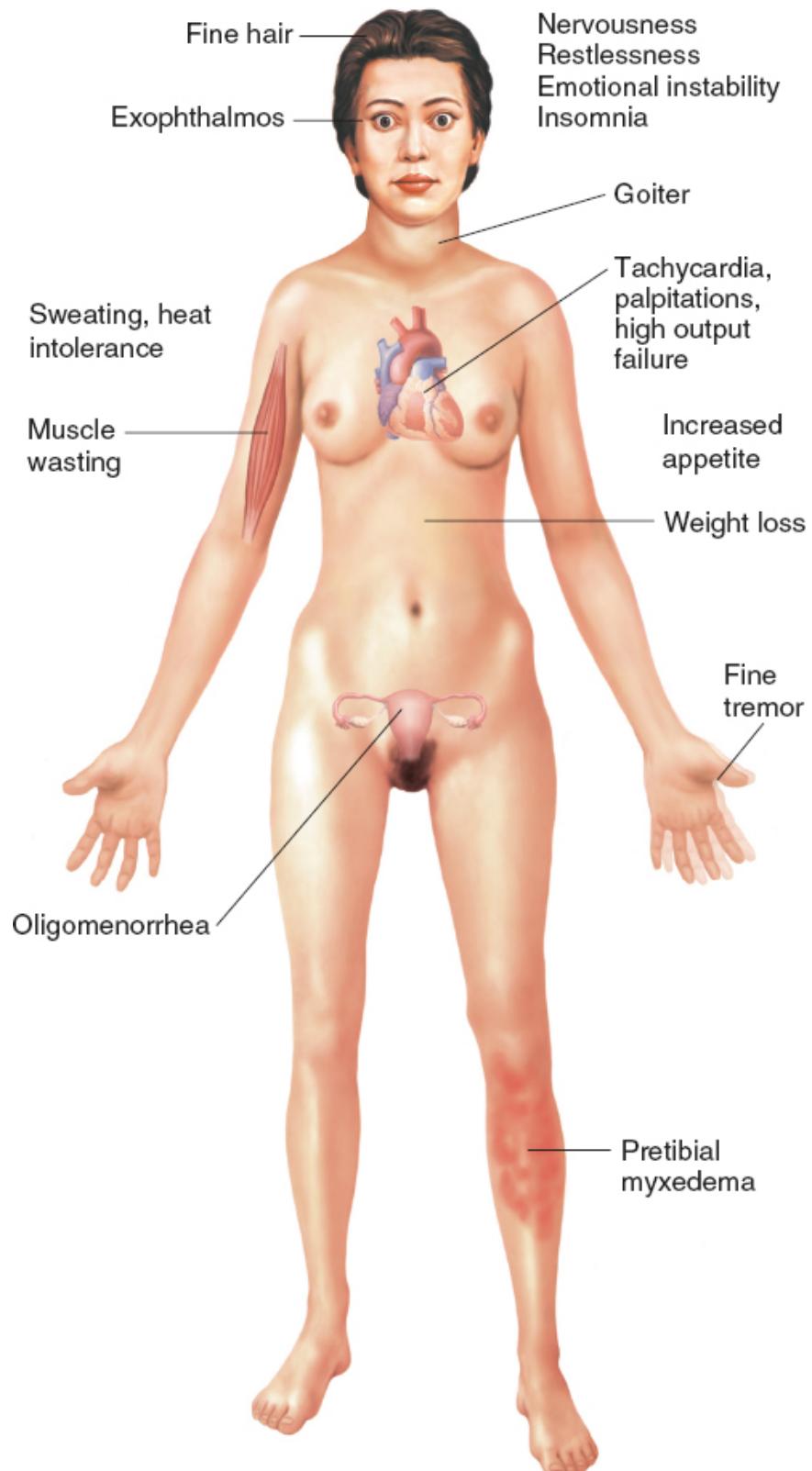


Figure 45-6 • Clinical manifestations of hyperthyroidism. Reprinted with permission from Norris, T. L. (2019). *Porth's pathophysiology*:

Concepts of altered health states (10th ed., Fig. 41.5). Philadelphia, PA: Wolters Kluwer.

Cardiac effects may include sinus tachycardia or arrhythmias, decreased cardiac output, increased pulse pressure, and palpitations; these changes may be related to increased sensitivity to catecholamines or to changes in neurotransmitter turnover. Myocardial hypertrophy and heart failure may occur if the hyperthyroidism is severe and untreated.

The course of the disease may be mild, characterized by remissions and exacerbations, and terminate with spontaneous recovery in a few months or years. Conversely, it may progress relentlessly, with the untreated person becoming emaciated, intensely nervous, delirious, and even disoriented; eventually, the heart fails.

Symptoms of hyperthyroidism may occur with the release of excessive amounts of thyroid hormone as a result of inflammation after irradiation of the thyroid or destruction of thyroid tissue by tumor. Such symptoms may also occur with excessive administration of thyroid hormone for the treatment of hypothyroidism. Long-standing use of thyroid hormone in the absence of close monitoring may be a cause of symptoms of hyperthyroidism. It is also likely to result in premature osteoporosis, particularly in women.

Assessment and Diagnostic Findings

The thyroid gland is enlarged to some extent. It is soft and may pulsate; a thrill often can be palpated, and a bruit is heard over the thyroid arteries (Norris, 2019). These are signs of greatly increased blood flow through the thyroid gland. In advanced cases, the diagnosis is made on the basis of the symptoms, a decrease in serum TSH, increased free T₄, and an increase in radioactive iodine uptake.

Medical Management

Appropriate treatment of hyperthyroidism depends on the underlying cause and often consists of a combination of therapies, including antithyroid agents, radioactive iodine, and surgery. Treatment of hyperthyroidism is directed toward reducing thyroid hyperactivity to relieve symptoms and preventing complications. The use of radioactive iodine is the most common form of treatment for Graves disease. Beta-adrenergic blocking agents (e.g., propranolol, atenolol, metoprolol) are used as adjunctive therapy for symptomatic relief, particularly in transient thyroiditis (Lee & Khadori, 2018). The three treatments (radioactive iodine therapy, antithyroid medications [e.g., thionamides], and surgery) share the same complications: relapse or recurrent hyperthyroidism and permanent hypothyroidism. The rate

of relapse increases in patients who have had very severe disease, a long history of dysfunction, ocular and cardiac symptoms, large goiter, or relapse after previous treatment. Patients with Graves disease may sustain remission for up to 12 to 18 months but often experience recurrence within 12 months of treatment (Lee & Khadori, 2018).

Pharmacologic Therapy

Two forms of pharmacotherapy are available for treating hyperthyroidism and controlling excessive thyroid activity: (1) the use of irradiation by administration of the radioisotope ^{131}I for destructive effects on the thyroid gland and (2) antithyroid medications that interfere with the synthesis of thyroid hormones and other agents that control manifestations of hyperthyroidism.

Radioactive Iodine Therapy

Radioactive iodine has been used to treat toxic adenomas, toxic multinodular goiter, and most varieties of thyrotoxicosis and is considered the treatment of choice because a single dose is effective in treating 80% to 90% of cases (Bauerle & Clutter, 2019). Radioactive iodine is contraindicated during pregnancy because it crosses the placenta. Women of childbearing age should be given a pregnancy test 48 hours before administration of radioactive iodine. They should also be instructed to not conceive for at least 6 months following treatment. In addition, breast-feeding for up to 6 weeks prior to radioactive iodine treatment is contraindicated (Lee & Khadori, 2018).

The goal of radioactive iodine therapy (^{131}I) is to eliminate the hyperthyroid state with the administration of sufficient radiation in a single dose (Lee & Khadori, 2018). Almost all of the iodine that enters and is retained in the body becomes concentrated in the thyroid gland. Therefore, the radioactive isotope of iodine is concentrated in the thyroid gland, where it destroys thyroid cells without jeopardizing other radiosensitive tissues. Over a period of several weeks, thyroid cells exposed to the radioactive iodine are destroyed, resulting in reduction of the hyperthyroid state and inevitably hypothyroidism.

The use of an ablative dose of radioactive iodine initially causes an acute release of thyroid hormone from the thyroid gland and may cause increased symptoms. The patient is observed for signs of **thyroid storm** (see Chart 45-6), a life-threatening condition manifested by cardiac arrhythmias, fever, and neurologic impairment (Norris, 2019) which may lead to heart failure, circulatory collapse and dangerous elevation of body temperature, all related to the increase in metabolism. Beta-blockers are used to control these symptoms.

Thyroid hormone replacement is started 4 to 18 weeks after the antithyroid medications have been stopped based on the results of thyroid function tests. TSH measurements can be misleading in the early months following treatment

with radioactive iodine. Therefore, serum free T₄ is the principal test measured at 3 to 6 weeks following administration of radioactive iodine and then every 1 to 2 months until normal thyroid function is established. If TSH and free T₄ are both persistently low, the total T₃ then must be measured to differentiate between persistent hyperthyroidism (T₃ elevated) or transient hypothyroidism (T₃ normal or low). Once a normal thyroid state has been established, TSH should be measured every 6 to 12 months for life (Fischbach & Fischbach, 2018).

A major advantage of treatment with radioactive iodine is that it avoids many of the side effects associated with antithyroid medications. However, some patients may elect to be treated with antithyroid medications rather than radioactive iodine for a variety of reasons, including fear of radiation.

Chart 45-6

Thyroid Storm (Thyrotoxic Crisis, Thyrotoxicosis)

Thyroid storm (thyrotoxic crisis) is a form of severe hyperthyroidism, usually of abrupt onset. Untreated, it is almost always fatal, but with proper treatment the mortality rate is reduced substantially. The patient with thyroid storm or crisis is critically ill and requires astute observation and aggressive and supportive nursing care during and after the acute stage of illness.

Clinical Manifestations

Thyroid storm is characterized by:

- Hyperpyrexia (high fever), $>38.5^{\circ}\text{C}$ ($>101.3^{\circ}\text{F}$)
- Extreme tachycardia (>130 bpm)
- Exaggerated symptoms of hyperthyroidism with disturbances of a major system—for example, gastrointestinal (weight loss, diarrhea, abdominal pain) or cardiovascular (edema, chest pain, dyspnea, palpitations)
- Altered neurologic or mental state, which frequently appears as delirium psychosis, somnolence, or coma

Life-threatening thyroid storm is usually precipitated by stress, such as injury, infection, thyroid and nonthyroid surgery, tooth extraction, insulin reaction, diabetic ketoacidosis, pregnancy, digitalis intoxication, abrupt withdrawal of antithyroid medications, extreme emotional stress, or vigorous palpation of the thyroid. These factors can precipitate thyroid storm in the partially controlled or completely untreated patient with hyperthyroidism. Current methods of diagnosis and treatment for hyperthyroidism have greatly decreased the incidence of thyroid storm, making it uncommon today.

Management

Immediate objectives are reduction of body temperature and heart rate and prevention of vascular collapse. Measures to accomplish these objectives include:

- A hypothermia mattress or blanket, ice packs, a cool environment, hydrocortisone, and acetaminophen. Salicylates (e.g., aspirin) are not used because they displace thyroid hormone from binding proteins and worsen the hypermetabolism.
- Humidified oxygen is given to improve tissue oxygenation and meet the high metabolic demands. Arterial blood gas levels or pulse oximetry may be used to monitor respiratory status.
- IV fluids containing dextrose are given to replace liver glycogen stores that have been decreased in the patient who is hyperthyroid.
- Propylthiouracil or methimazole is given to impede formation of thyroid hormone and block conversion of T_4 to T_3 , the more active form of thyroid hormone.
- Hydrocortisone is prescribed to treat shock or adrenal insufficiency.

- Iodine is given to decrease output of T_4 from the thyroid gland. For cardiac problems such as atrial fibrillation, arrhythmias, and heart failure, sympatholytic agents may be given. Propranolol, combined with digitalis, has been effective in reducing severe cardiac symptoms.

T_3 , triiodothyronine; T_4 , thyroxine.

Adapted from Morton, P. G., & Fontaine, D. K. (2018). *Critical care nursing: A holistic approach* (10th ed.). Philadelphia, PA: Wolters Kluwer; Comerford, K. C., & Durkin, M. T. (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer.

Patients who receive radioactive iodine should be informed that they can contaminate their household and other people through saliva, urine, or radiation emitting from their body. They should avoid sexual contact, sleeping in the same bed with other people, having close contact with children and pregnant women, and sharing utensils and cups. The patient should follow the instructions provided regarding the time restrictions for these cautions because they are dose related (Fischbach & Fischbach, 2018).

Antithyroid Medications

Antithyroid medications (thionamides) are summarized in Table 45-1. The objective of pharmacotherapy is to inhibit one or more stages in thyroid hormone synthesis or hormone release. Antithyroid agents block the utilization of iodine by interfering with the iodination of tyrosine and the coupling of iodotyrosines in the synthesis of thyroid hormones. This prevents the synthesis of thyroid hormone. The most commonly used antithyroid drugs in the United States are methimazole or propylthiouracil. The medications are used until the patient is euthyroid (i.e., neither hyperthyroid nor hypothyroid). These medications block extrathyroidal conversion of T_4 to T_3 (Bauerle & Clutter, 2019).

Prior to initiating therapy with these drugs, baseline blood tests are performed, including complete blood count (white blood cell [WBC] count with differential) and liver profile (transaminases and bilirubin) (Bauerle & Clutter, 2019). The therapeutic dose is determined on the basis of clinical criteria, including changes in pulse rate, pulse pressure, body weight, size of the goiter, and results of laboratory studies. The patient should be instructed to take the medication in the morning on an empty stomach 30 minutes before eating to avoid decrease in absorption associated with some foods such as walnuts, soybean flour, cottonseed meal, and dietary fiber. Because antithyroid medications do not interfere with release or activity of previously formed thyroid hormones, it may take several weeks until symptom relief occurs. At that time, the maintenance dose is established, and the medication is gradually tapered over several months.

Toxic complications of antithyroid medications are relatively uncommon; nevertheless, the importance of periodic follow-up is emphasized, because medication sensitization, fever, rash, urticaria, or even agranulocytosis and thrombocytopenia (decrease in granulocytes and platelets) may develop (Bauerle & Clutter, 2019). With any sign of infection, especially pharyngitis and fever or the occurrence of mouth ulcers, the patient is advised to stop the medication, notify the primary provider immediately, and undergo hematologic studies (Bauerle & Clutter, 2019). Propylthiouracil is recommended during the first trimester of pregnancy rather than methimazole due to the teratogenic effects of methimazole. Due to risk of hepatotoxicity, propylthiouracil should be discontinued after the first trimester and the patient should be switched to methimazole for the remainder of the pregnancy and when breast-feeding (Bauerle & Clutter, 2019).

TABLE 45-1 Pharmacologic Agents Used to Treat Hyperthyroidism

Agent	Action	Nursing Considerations
Propylthiouracil	Blocks synthesis of hormones (conversion of T ₄ to T ₃)	Monitor cardiac parameters. Observe for conversion to hypothyroidism. Must be given by mouth. Watch for rash, nausea, vomiting, agranulocytosis, SLE.
Methimazole	Inhibits synthesis of thyroid hormone	More toxic than propylthiouracil. Watch for rash and other symptoms as for propylthiouracil.
Sodium iodide	Suppresses release of thyroid hormone	Given 1 h after propylthiouracil or methimazole. Watch for edema, hemorrhage, gastrointestinal upset.
Potassium iodide	Suppresses release of thyroid hormone	Discontinue for rash. Watch for signs of toxic iodonism.
Saturated solution of potassium iodide (SSKI)	Suppresses release of thyroid hormone	Mix with juice or milk. Given by straw to prevent staining of teeth.
Beta-blocker (e.g., propranolol)	Beta-adrenergic blocking agent	Monitor cardiac status. Hold for bradycardia or decreased cardiac output. Use with caution in patients with heart failure.

SLE, systemic lupus erythematosus; T₃, triiodothyronine; T₄, thyroxine.

Adapted from Bauerle, K. T., & Clutter, W. E. (2019). Hyperthyroidism. In T. J. Braranski, J. B. McGill, & J. M. Silverstein (Eds.). *The Washington manual endocrinology subspecialty consult* (4th ed.). Philadelphia, PA: Wolters Kluwer; Comerford, K. C., & Durkin, M. T. (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer.

Discontinuation of antithyroid medications before therapy is complete usually results in relapse within 6 months. It is important that the possibility of relapse be discussed so that a treatment strategy will be in place if relapse occurs.

Adjunctive Therapy

Additional medications may be necessary. Potassium iodide (SSKI) may be used in combination with antithyroid agents or beta-adrenergic blockers to prepare the patient with hyperthyroidism for surgery. The drugs reduce the effects of hyperthyroidism quickly and help to prevent the onset of thyroid

storm. The usual dosage for SSKI is 5 drops every 6 hours. The usual dose of propylthiouracil is 200 mg every 6 hours and the usual dose of propranolol is 60 to 80 mg orally every 6 hours to prevent tachycardia. The patient will need to continue to take the propylthiouracil and any cardiac medication until the free T₄ and T₃ levels are near normal (Bauerle & Clutter, 2019).

Surgical Management

Surgery to remove thyroid tissue is reserved for special circumstances, for example, in pregnant women who are allergic to antithyroid medications, in patients with large goiters, or in patients who are unable to take antithyroid agents. Surgery for treatment of hyperthyroidism is performed soon after the thyroid function has returned to normal (4 to 6 weeks).

The surgical removal of about five sixths of the thyroid tissue (subtotal thyroidectomy) reliably results in a prolonged remission in most patients with exophthalmic goiter. Its use today is reserved for patients with obstructive symptoms, some pregnant women, and for patients with a need for rapid normalization of thyroid function. Before surgery, an antithyroid medication is given until signs of hyperthyroidism have disappeared. A beta-adrenergic blocking agent (e.g., propranolol) may be used to reduce the heart rate and other signs and symptoms of hyperthyroidism. Medications that may prolong clotting (e.g., aspirin) are stopped several weeks before surgery to reduce the risk of postoperative bleeding. Patients receiving iodine medication must be monitored for evidence of iodine toxicity, which requires immediate withdrawal of the medication. Symptoms of toxicity include mucosa membranes stained brown, burning pain in the mouth and esophagus, laryngeal edema, and shock (Fischbach & Fischbach, 2018).



Gerontologic Considerations

Although hyperthyroidism is much less common in older adults than hypothyroidism, patients 65 years and older need careful assessment to avoid missing subtle signs and symptoms. This age group may present with atypical vague and nonspecific signs and symptoms of thyroid disease such as anorexia and weight loss with absence of ocular signs, or isolated atrial fibrillation (Morton & Fontaine, 2018). New or worsening heart failure or angina is more likely to occur in older patients rather than in younger patients. Symptoms such as tachycardia, fatigue, mental confusion, weight loss, change in bowel habits, and depression can be attributed to age and other illnesses that are common in older adults (Eliopoulos, 2018). The older patient may complain of difficulty climbing stairs or rising from a chair because of muscle weakness. Evaluation for thyroid disease with a serum TSH measurement is indicated in older patients who have unexplained physical or mental deterioration

(Samuels, 2018). Free T₄ and T₃ should be included in the initial screening when hyperthyroidism is highly suspected. Once thyrotoxicosis is confirmed, additional tests such as radioactive iodine uptake and thyroid scan are prescribed to differentiate between causes such as Graves disease, toxic nodular goiter, acute thyroiditis, and other disorders. Toxic nodular goiter is the most common cause of thyrotoxicosis in older patients. Patients have the option of treatment using antithyroid medications, radioactive iodine, and surgery. Radioactive iodine is generally recommended for treatment of thyrotoxicosis caused by toxic nodular goiter in older patients unless an enlarged thyroid gland is pressing on the airway. Long-term use of certain antithyroid medications such as propylthiouracil is not recommended for treatment of hyperthyroidism in older adults due to potential side effects (Samuels, 2018).

The use of beta-adrenergic blocking agents (e.g., propranolol and atenolol) may be indicated to decrease the cardiovascular and neurologic signs and symptoms of thyrotoxicosis. These agents must be used with extreme caution in older patients to minimize adverse effects on cardiac function that may produce heart failure. The dosage of other medications used to treat other chronic illnesses in older patients may also need to be modified because of the altered rate of metabolism associated with hyperthyroidism.

The Patient with Hyperthyroidism

Assessment

The health history and examination focus on symptoms related to accelerated or exaggerated metabolism. These include the patient's and family's reports of irritability and increased emotional reaction and the impact that these changes have had on the patient's interactions with family, friends, and coworkers. The history includes other stressors and the patient's ability to cope with stress.

The nurse initially and periodically assesses the patient's nutritional status and the presence of symptoms related to the hypermetabolic state. This hypermetabolic state may affect the cardiovascular system, causing changes in vital signs including heart rate and rhythm, blood pressure, heart sounds, and peripheral pulses. Other specific changes may include alteration in vision and appearance of the external eye. Because emotional changes are associated with hyperthyroidism, the patient's emotional state and psychological status are evaluated, as well as such symptoms as irritability, anxiety, sleep disturbances, apathy, and lethargy, all of which may occur with hyperthyroidism (Bauerle & Clutter, 2019). The family may also provide information about recent changes in the patient's emotional status.

Diagnosis

NURSING DIAGNOSES

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

- Risk for impaired cardiac function associated with alteration in heart rate and rhythm.
- Impaired nutritional status associated with exaggerated metabolic rate, excessive appetite, and increased GI activity
- Difficulty coping associated with irritability, hyperexcitability, apprehension, and emotional instability
- Situational low self-esteem associated with changes in appearance, excessive appetite, and weight loss
- Risk for impaired thermoregulation

COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS

Potential complications may include the following:

- Thyrotoxicosis or thyroid storm
- Hypothyroidism

Planning and Goals

The major goals for the patient may include maintenance of adequate cardiac function, maintenance of adequate nutritional status, improved coping ability, improved self-esteem, maintenance of normal body temperature, and absence of complications.

Nursing Interventions

MAINTAINING ADEQUATE CARDIAC OUTPUT

Due to the effects of hyperthyroidism on the cardiac system, it is important to critically assess vital signs, especially heart rate and rhythm and body temperature. Nurses need to be alert to complaints of palpitations which may be reported as “my heart is racing.” The nurse monitors the patient for signs of heart failure (dyspnea, jugular vein distention, crackles, and peripheral edema; see [Chapter 25](#)).

In the presence of thyroid storm the patient should be placed on a cardiac monitor in order to adequately monitor for arrhythmias. Monitoring of electrolytes and strict I&O assessment is also essential. The nurse is prepared to administer beta-blockers as prescribed. Acetaminophen is the drug of choice to reduce elevated body temperature because medications containing salicylates may result in higher levels of unbound thyroid hormone. A cooling blanket as well as a cool environment may also be needed.

IMPROVING NUTRITIONAL STATUS

Hyperthyroidism affects all body systems, including the GI system. Rapid movement of food through the GI tract may result in nutritional imbalance and weight loss. In addition, the patient will report an increased appetite and should be encouraged to eat small frequent nutritious meals. If necessary, the patient is referred to a nutritionist or dietician to develop a meal plan to address dietary concerns.

Foods and fluids are selected to replace fluid lost through diarrhea and diaphoresis and to control the diarrhea that results from increased peristalsis. To reduce diarrhea, highly seasoned foods, coffee, tea, cola, and alcohol are discouraged while high-calorie, high-protein foods are encouraged. A quiet atmosphere during mealtime may aid digestion. The patient should be encouraged to record weight and dietary intake.

ENHANCING COPING MEASURES

The patient with hyperthyroidism needs reassurance that the emotional reactions being experienced are a result of the disorder and that with effective treatment those symptoms will be controlled. Because of the negative effect that these symptoms can have on family and friends, they too need reassurance that the symptoms are expected to disappear with treatment. A calm, unhurried approach is beneficial for the patient. Stressful experiences should be minimized and a quiet, uncluttered

environment should be maintained. The patient should be instructed to balance periods of activity with rest.

If a thyroidectomy is planned, the patient needs to know that pharmacologic therapy is necessary to prepare the thyroid gland for surgical treatment. The nurse provides education and reminds the patient to take the medications as prescribed. Because of hyperexcitability and shortened attention span, the patient may require repetition of this education and written instructions.

IMPROVING SELF-ESTEEM

The patient with hyperthyroidism is likely to experience changes in appearance, appetite, and weight. These factors, along with the patient's inability to cope well with family and the illness, may result in loss of self-esteem. The nurse conveys an understanding of the patient's concern about these problems and promotes the use of effective coping strategies. The patient and family should be reassured that these changes are a result of the thyroid dysfunction and are, in fact, out of the patient's control. The nurse refers the patient to professional counseling as necessary.

If the patient experiences ocular changes secondary to hyperthyroidism, eye care and protection may be necessary. The nurse educates the patient about instillation of eye drops or ointment prescribed to soothe the eyes and protect the exposed corneas. Smoking should be highly discouraged, and smoking cessation strategies are recommended. The patient may be embarrassed by the need to eat large meals. The nurse explains the need for increased food consumption to caregivers and family members in order to address the possibility of their commenting on the patient's increased appetite.

MAINTAINING NORMAL BODY TEMPERATURE

The patient with hyperthyroidism frequently finds a normal room temperature too warm because of an exaggerated metabolic rate and increased heat production. If the patient is hospitalized, the environment should be maintained at a cool, comfortable temperature, and the bedding and clothing should be changed as needed. Cool baths and cool or cold fluids may also provide relief.

MONITORING AND MANAGING POTENTIAL COMPLICATIONS

The nurse closely monitors the patient with hyperthyroidism for signs and symptoms that may be indicative of thyroid storm. Cardiac and respiratory functions are assessed by measuring vital signs and cardiac output, electrocardiographic (ECG) monitoring, arterial blood gases, and pulse oximetry. Assessment continues after treatment is initiated because of the potential effects of treatment on cardiac function. Oxygen is given to prevent hypoxia, to improve tissue oxygenation, and to meet the high metabolic demands. IV fluids may be necessary to maintain blood glucose

levels and to replace lost fluids. Antithyroid medications (methimazole or propylthiouracil) may be prescribed to reduce thyroid hormone levels. In addition, beta-blockers and digitalis may be prescribed to treat cardiac symptoms. If shock develops, treatment strategies must be implemented (see [Chapter 11](#)).

Hypothyroidism is likely to occur with any of the treatments used for hyperthyroidism. Therefore, the nurse periodically monitors the patient. Most patients report a greatly improved sense of well-being after treatment of hyperthyroidism, and some fail to continue to take prescribed thyroid replacement therapy. Therefore, part of patient and family education is instruction about the importance of continuing therapy indefinitely after discharge and a discussion of the consequences of failing to take medication.

PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE



Educating Patients About Self-Care. The nurse educates the patient with hyperthyroidism about how and when to take prescribed medication and provides education about the essential role of the medication in the broader therapeutic plan. Because of the hyperexcitability and decreased attention span associated with hyperthyroidism, the nurse provides a written plan for the patient to take home and use. The type and amount of information given depend on the patient's stress and anxiety levels. The patient and family members receive verbal and written education about the actions and possible side effects of the medications as well as adverse effects that should be reported if they occur (see [Chart 45-7](#)).

Chart 45-7



HOME CARE CHECKLIST

The Patient with Hyperthyroidism

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

- State the impact of hyperthyroidism, treatment on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- State that emotional lability is part of disease process.
- Identify the potential for menstrual irregularities and pregnancy, and increased risk of osteoporosis in women.
- State that long-term treatment and follow-up is necessary.
- Describe the potential benefits and risks of surgical intervention or radioactive iodine therapy.
- State the name, dose, side effects, frequency, and schedule for all medications.
- Explain the purpose, dose, route, schedule, side effects, and precautions of treatment of hyperthyroidism (antithyroid medications, radioactive iodine).
- State the need to contact primary provider before taking over-the-counter medications.
- State changes in lifestyle (e.g., diet, activity) necessary to maintain health.
- Identify the need for increased dietary intake until weight stabilizes.
- Identify foods to be avoided.
- Identify the need for planned rest periods and methods to improve sleep patterns.
- Identify areas of stress and management techniques.
- Identify rationale for smoking cessation and steps to stop use of any tobacco product.
- State precipitating factors and interventions for complications (hypothyroidism, thyroid storm).
- Relate how to reach primary provider with questions or complications.
- State time and date of follow-up medical appointments, therapy, and testing.
 - Identify sources of support (e.g., friends, relatives, faith community).
 - Identify the contact details for support services for patients and their caregivers/families.
- Identify the need for health promotion, disease prevention, and screening activities.

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

If a total or subtotal thyroidectomy is anticipated, the patient needs to be educated about what to expect. Information is repeated as the time of surgery approaches. The nurse also advises the patient to avoid stressful situations that may precipitate thyroid storm.

Continuing and Transitional Care. Referral for home, community-based or transitional care, if indicated, allows the nurse to assess the home and family environment, as well as the patient's and family's understanding of the importance of adhering to the therapeutic regimen and the recommended follow-up monitoring. The nurse reinforces to the patient and family the importance of long-term follow-up because of the risk of hypothyroidism after thyroidectomy or treatment with antithyroid medications or radioactive iodine. The patient is assessed for changes indicating return to normal thyroid function and signs and symptoms of hyperthyroidism and hypothyroidism. Furthermore, the patient and family are reminded about the importance of health promotion activities and recommended health screening.

Evaluation

Expected patient outcomes may include:

1. Improved cardiac status
 - a. Vital signs within normal limits
 - b. Absence of dyspnea, crackles, and peripheral edema
 - c. Reports absence of palpitations
2. Improved nutritional status
 - a. Reports adequate dietary intake and decreased hunger
 - b. Identifies high-calorie, high-protein foods; identifies foods to be avoided
 - c. Avoids the use of alcohol and stimulants
 - d. Stops smoking
 - e. Reports decreased episodes of diarrhea
3. Demonstrates effective coping methods in dealing with family, friends, and coworkers
 - a. Explains reasons for irritability and emotional instability
 - b. Avoids stressful situations, events, and people
 - c. Participates in relaxing, nonstressful activities
4. Achieves increased self-esteem
 - a. Verbalizes feelings about self and illness
 - b. Describes feelings of frustration and loss of control
 - c. Describes reasons for increased appetite
5. Maintains normal body temperature
6. Absence of complications

- a. Serum thyroid hormone and TSH levels within normal limits
- b. Identifies signs and symptoms of thyroid storm and hypothyroidism
- c. Vital signs and results of ECG, arterial blood gases, and pulse oximetry within normal limits
- d. States importance of regular follow-up and lifelong maintenance of prescribed therapy

Thyroid Tumors

Tumors of the thyroid gland are classified on the basis of being benign or malignant, the presence or absence of associated thyrotoxicosis, and the diffuse or irregular quality of the glandular enlargement. If the enlargement is sufficient to cause a visible swelling in the neck, the tumor is referred to as a goiter.

All grades of goiter are encountered, from those that are barely visible to those producing disfigurement. Some are symmetric and diffuse; others are nodular. Some are accompanied by hyperthyroidism, in which case they are described as toxic; others are associated with a euthyroid state and are referred to as nontoxic goiters.

Endemic (Iodine-Deficient) Goiter

The most common type of goiter that occurs when iodine intake is deficient is the simple or colloid goiter. In addition to being caused by an iodine deficiency, simple goiter may be caused by an intake of large quantities of goitrogenic substances in patients with unusually susceptible glands. These substances include excessive amounts of iodine. Lithium prescribed for the treatment of bipolar disorder has also been found to also have antithyroid actions (Singh & Clutter, 2019).

Simple goiter is a compensatory hypertrophy of the thyroid gland, caused by stimulation by the pituitary gland. The pituitary gland produces thyrotropin or TSH, a hormone that controls the release of thyroid hormone from the thyroid gland. Its production increases if there is subnormal thyroid activity, as when insufficient iodine is available for production of the thyroid hormone. Such goiters usually cause no symptoms, except for the swelling in the neck, which may result in tracheal compression when excessive swelling is present.

Many goiters of this type recede after the iodine imbalance is corrected. Supplementary iodine, such as SSKI, is prescribed to suppress the pituitary's thyroid-stimulating activity. When surgery is indicated, the risk of postoperative complications is minimized by ensuring a preoperative euthyroid state through treatment with antithyroid medications and iodide to reduce the

size and vascularity of the goiter. The introduction of iodized salt has been the single most effective means of preventing goiter in at-risk populations.

Nodular Goiter

Some thyroid glands are nodular because of areas of hyperplasia (overgrowth). No symptoms may arise as a result of this condition, but not uncommonly these nodules slowly increase in size, with some descending into the thorax, where they cause local pressure symptoms. Some nodules become malignant, and some are associated with a hyperthyroid state. Therefore, the patient with many thyroid nodules may eventually require surgery.

Thyroid Cancer

Cancer of the thyroid is less prevalent than other forms of cancer; however, the incidence has tripled in the last 30 years (American Cancer Society [ACS], 2019) and accounts for 90% of endocrine malignancies. Although it has the fastest-growing cancer rate among both men and women, women are three times more likely to develop this cancer than men. In addition, thyroid cancer is more likely to develop in patients that are younger than 50 years (ACS, 2019; Yoo, Yu, & Choi, 2018).

Chart 45-8



NURSING RESEARCH PROFILE

Lifestyle Factors and the Risk of Thyroid Cancer

Yoo, Y. G., Yu, B. J., & Choi, E. (2018) A comparison study: The risk factors in the lifestyles of thyroid cancer patients and healthy adults of South Korea. *Cancer Nursing*, 41(1), E48–E56.

Purpose

In South Korea and in the United States, rates of thyroid cancer have increased significantly. This study investigated which risk factors might be influencing the rates of thyroid cancer in South Korea.

Design

This retrospective comparison study compared a group of patients with thyroid cancer to a group of healthy adults. The Health Belief Model framework guided the study. The Lifestyle Measurement Scale was used to assess the 6 domains of dietary habits, alcohol consumption, smoking habits, rest and physical activity, stress management, and annual physical and health screenings. Participants self-completed the surveys.

Findings

There were 217 usable surveys completed. In the patient group ($n = 102$), mean age was 50 years; in the healthy adult group ($n = 115$), 52 years. In both the patient group and the healthy adult group, females made up the majority (85% and 76%, respectively) and most of the participants were married (94% and 93%, respectively). A history of previous smoking, lower physical activity levels, higher stress, and unhealthy eating habits (consumption of more instant food products and fewer vegetables) were all identified as risk factors for developing thyroid cancer.

Nursing Implications

All health care professionals can be instrumental in helping prevent thyroid cancer. Nurses should encourage higher physical activity levels, effective stress management, avoidance of direct and indirect exposure to smoking, and a healthy diet that includes consumption of fewer instant food products and more vegetables to help in prevention efforts.

External radiation of the head, neck, or chest in infancy and childhood increases the risk of thyroid carcinoma. The incidence of thyroid cancer appears to increase 5 to 40 years after irradiation. Consequently, people who underwent radiation treatment or were otherwise exposed to radiation as children should consult their primary provider, request an isotope thyroid scan as part of the evaluation, follow recommended treatment of abnormalities of the gland, and continue with annual checkups.

Additional risk factors that have been identified include smoking, low physical activity, unhealthy eating habits and high stress levels (Yoo et al.,

2018). See the Nursing Research Profile in [Chart 45-8](#) for more information on lifestyle risk factors.

Assessment and Diagnostic Findings

Lesions that are single, hard, and fixed on palpation or associated with cervical lymphadenopathy suggest malignancy. Thyroid function tests may be helpful in evaluating thyroid nodules and masses; however, results are rarely conclusive. An ultrasound-guided fine needle biopsy of the thyroid gland is the standard diagnostic procedure for evaluating thyroid nodules. It is performed as an outpatient procedure to make a diagnosis of thyroid cancer, to differentiate cancerous thyroid nodules from noncancerous nodules, and to stage the cancer if detected (Amdur & Dagan, 2019). The procedure is safe and usually requires only a local anesthetic agent. Additional diagnostic studies include ultrasound, MRI, CT, thyroid scans, radioactive iodine uptake studies, and thyroid suppression tests.

Medical Management

The medical management depends on the classification of cell type found on biopsy. The three common groups include well-differentiated thyroid cancer (DTC), papillary thyroid carcinoma (PTC), and follicular thyroid carcinoma (FTC) (Amdur & Dagan, 2019).

The treatment of choice for localized thyroid carcinoma is surgical removal (Amdur & Dagan, 2019). Total or near-total thyroidectomy is performed if possible (ACS, 2019). Modified neck dissection or more extensive radical neck dissection is performed if there is lymph node involvement.

Efforts are made to spare parathyroid tissue to reduce the risk of postoperative hypocalcemia and tetany. After surgery, ablation procedures are carried out with radioactive iodine to eradicate residual microscopic disease (ACS, 2019). Radioactive iodine is also used for thyroid cancers with metastasis (ACS, 2019).

After surgery, thyroid hormone is given to lower the levels of TSH to a euthyroid state (Bauerle & Riek, 2019). If the remaining thyroid tissue is inadequate to produce sufficient thyroid hormone, thyroxine is required permanently.

Several routes are available for administering radiation to the thyroid or tissues of the neck, including oral administration of radioactive iodine (Bauerle & Riek, 2019) and external administration of radiation therapy. Administration of radioactive iodine for DTC is the most successful targeted therapy in oncology (Amdur & Dagan, 2019). Short-term side effects of radioactive iodine treatment may include neck soreness, nausea, and upset stomach; tender and swollen salivary glands; dry mouth; changes in taste; and, rarely, pain

(Bauerle & Riek, 2019). The patient who receives external sources of radiation therapy is at risk for mucositis, dryness of the mouth, dysphagia, redness of the skin, anorexia, and fatigue (see [Chapter 12](#)). Chemotherapy is infrequently used to treat thyroid cancer.

Patients whose thyroid cancer is detected early, who are younger than 50 years, and who are appropriately treated have a good prognosis (Amdur & Dagan, 2019). Patients who have had papillary cancer—the most common and least aggressive tumor—have the best prognosis of all thyroid cancers (ACS, 2019). Long-term survival is also common in follicular cancer, which is a more aggressive form of thyroid cancer (Bauerle & Riek, 2019). However, continued thyroid hormone therapy and periodic follow-up and diagnostic testing are important to ensure the patient's well-being.

Later follow-up includes clinical assessment for recurrence of nodules or masses in the neck and signs of hoarseness, dysphagia, or dyspnea. The recommendations for long-term follow-up of patients with differentiated thyroid cancer are based on the stage of cancer and results of the follow-up examination 1 year following the initial treatment. The first year evaluation includes clinical examination, TSH and free thyroxine, and measurement of serum thyroglobulin within 6 months following the initial treatment, and a routine neck ultrasound with the first 6 to 12 months following initial treatment. Tests used to confirm sites of metastasis if there is clinical evidence of recurrence include radioiodine imaging, CT, MRI, skeletal x-rays, and skeletal radionucleotide imaging.

Fluorodeoxyglucose (FDG) PET is useful to establish prognosis if there is evidence of distant metastases (ACS, 2019). Free T₄, TSH, and serum calcium and phosphorus levels are monitored to determine whether the thyroid hormone supplementation is adequate and to note whether calcium balance is maintained.

Patient education emphasizes the importance of taking prescribed medications and following recommendations for follow-up monitoring. The patient who is undergoing radiation therapy is also instructed in how to assess and manage side effects of treatment (see [Chapter 12](#)).

Nursing Management

Important preoperative goals are to prepare the patient for surgery and reduce anxiety. Often, the patient's home life has become tense because of their restlessness, irritability, and nervousness secondary to hyperthyroidism. Efforts are necessary to protect the patient from tension and stress to avoid precipitating thyroid storm. Suggestions are made to limit stressful situations. Quiet and relaxing activities are encouraged.

Providing Preoperative Care

The nurse educates the patient about the importance of eating a diet high in carbohydrates and proteins. A high daily caloric intake is necessary because of the increased metabolic activity and rapid depletion of glycogen reserves. Supplementary vitamins, particularly thiamine and ascorbic acid, may be prescribed. The patient is reminded to avoid tea, coffee, cola, and other stimulants.

The nurse also informs the patient about the purpose of preoperative tests, if they are to be performed, and explains what preoperative preparations to expect. This information should help to reduce the patient's anxiety about the surgery. In addition, special efforts are made to ensure a good night's rest before surgery.

Preoperative education includes demonstrating to the patient how to support the neck with the hands after surgery to prevent stress on the incision. This involves raising the elbows and placing the hands behind the neck to provide support and reduce strain and tension on the neck muscles and the surgical incision.

Providing Postoperative Care

In the postoperative period, the priorities are to observe for any difficulty in breathing due to edema of the glottis, hematoma formation, or injury to the recurrent laryngeal nerve which requires the insertion of an airway, and to monitor the pulse and blood pressure for any indication of internal bleeding. The nurse must be alert for complaints of a sensation of pressure or fullness at the incision site which may indicate subcutaneous hemorrhage and hematoma formation and should be reported. In addition, the nurse periodically assesses the surgical dressings and reinforces as necessary. When the patient is in a recumbent position, the nurse observes the sides and the back of the neck as well as the anterior dressing for bleeding. A tracheostomy set is kept at the bedside at all times, and the surgeon is summoned at the first indication of respiratory distress. If the respiratory distress is caused by hematoma, surgical evacuation is required.

The intensity of pain is assessed, and analgesic agents are given as prescribed for pain. The nurse should anticipate apprehension in the patient and should inform the patient that oxygen will assist breathing. When moving and turning the patient, the nurse carefully supports the patient's head and avoids tension on the sutures. The most comfortable position is the semi-Fowler position, with the head elevated and supported by pillows.

IV fluids are given during the immediate postoperative period. Water may be given by mouth as soon as nausea subsides and bowel sounds are present. Usually, there is a little difficulty in swallowing; initially, cold fluids and ice may be taken better than other fluids. Often, patients prefer a soft diet to a liquid diet in the immediate postoperative period.

The patient is advised to talk as little as possible to reduce edema to the vocal cords; however, when the patient does speak, any voice changes are noted, indicating possible injury to the recurrent laryngeal nerve, which lies just behind the thyroid next to the trachea. An overbed table is provided for access to frequently used items so that the patient avoids turning their head. The table can also be used to support a humidifier when vapor-mist inhalations are prescribed for the relief of excessive mucous accumulation.



Quality and Safety Nursing Alert

An essential assessment following a thyroidectomy is of voice changes. Difficulty in speaking (the act of moving air to vibrate vocal cords) may indicate increasing edema, damage to laryngeal nerve, or hemorrhage and should be reported immediately.

The patient is encouraged to be out of bed as soon as possible and to eat foods that are easily swallowed. A high-calorie diet may be prescribed to promote weight gain. The incision may be closed using absorbable sutures, nonabsorbable sutures, and adhesive strips. Absorbable sutures dissolve within the body. If nonabsorbable sutures are used, the timeline for removal may vary; however, these types of sutures are usually removed 5 to 7 days following surgery. Adhesives will peel off spontaneously. The patient is usually discharged from the hospital on the day of surgery or soon afterward if the postoperative course is uncomplicated.

Monitoring and Managing Potential Complications

Hemorrhage, hematoma formation, edema of the glottis, and injury to the laryngeal nerve are complications reviewed previously in this chapter. Occasionally in thyroid surgery, the parathyroid glands are injured or removed, producing a disturbance in calcium metabolism. As the blood calcium level falls, hyperirritability of the nerves occurs, with spasms of the hands and feet and muscle twitching (see [Chapter 10](#)). This group of symptoms is termed *tetany*, and the nurse must immediately report its appearance because laryngospasm, although rare, may occur and obstruct the airway. Tetany of this type is usually treated with IV calcium gluconate. This calcium abnormality is usually temporary after thyroidectomy unless all parathyroid tissues were removed.



Quality and Safety Nursing Alert

Following thyroid surgery, the patient should be monitored closely for signs of tetany, including hyperirritability of the nerves, with spasms of the hands and feet and muscle twitching. Laryngospasm, although rare, may occur and obstruct the airway.

Promoting Home, Community-Based, and Transitional Care

Predischarge education is essential because these patients have short hospital stays. The patient, family, and caregivers need to be knowledgeable about the signs and symptoms that should be reported. Discharge education includes strategies for managing postoperative pain at home and for increasing humidification. The nurse explains to the patient and family the need for rest, relaxation, and adequate nutrition and to avoid putting strain on the incision and sutures. The patient is permitted to resume their former activities and responsibilities completely once recovered from surgery.

Family responsibilities and factors relating to the home environment that produce emotional tension have often been implicated as precipitating causes of thyrotoxicosis. A home visit provides an opportunity to evaluate these factors and to suggest ways to improve the home and family environment. If indicated, a referral to home, community-based or transitional care is made. The nurse reviews the history; performs a physical assessment; assesses the surgical incision; develops a plan of care with the patient and family; and educates the patient, family, and caregivers about wound care, signs and symptoms to report, stress reduction, and the importance of keeping appointments with the primary provider.

THE PARATHYROID GLANDS

Anatomic and Physiologic Overview

The parathyroid glands (normally four) are situated in the neck and embedded in the posterior aspect of the thyroid gland (see Fig. 45-7). Parathormone (parathyroid hormone)—the protein hormone produced by the parathyroid glands—regulates calcium and phosphorus metabolism. Increased secretion of parathormone results in increased calcium absorption from the kidney, intestine, and bones, which raises the serum calcium level (Norris, 2019). Some actions of this hormone are increased by the presence of vitamin D. Parathormone also tends to lower the blood phosphorus level. The serum level of ionized calcium regulates the output of parathormone. Increased serum calcium results in decreased parathormone secretion, creating a negative feedback system.

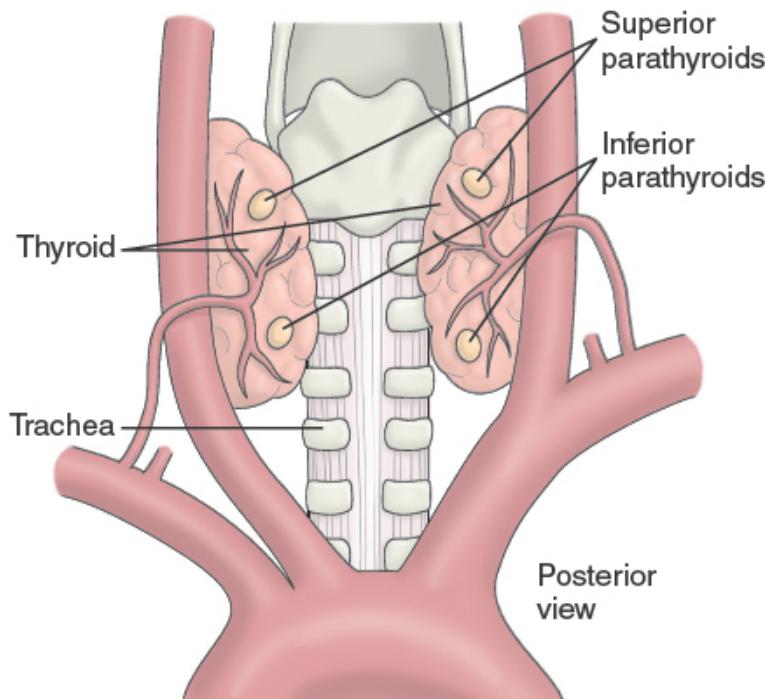


Figure 45-7 • The parathyroid glands are located behind the thyroid gland. The parathyroids may be embedded in the thyroid tissue.

Pathophysiology

Excess parathormone can result in markedly increased levels of serum calcium, which is a potentially life-threatening situation. When the product of serum calcium and serum phosphorus ($\text{calcium} \times \text{phosphorus}$) rises, calcium phosphate may precipitate in various organs of the body (e.g., the kidneys) and cause tissue calcification.

Hyperparathyroidism

Hyperparathyroidism is caused by overproduction of parathormone by the parathyroid glands and is characterized by bone decalcification and the development of renal calculi (kidney stones) containing calcium.

Primary hyperparathyroidism occurs two to four times more often in women than in men and is most common in people between 60 and 70 years of age. Its incidence is approximately 25 cases per 100,000 people. The disorder is rare in children younger than 15 years, but its incidence increases 10-fold between the ages of 15 and 65 years. Half of the people diagnosed with hyperparathyroidism do not have symptoms (Yalla & Hickey, 2019). Secondary hyperparathyroidism, with manifestations similar to those of

primary hyperparathyroidism, occurs in patients who have chronic kidney failure and the so-called renal rickets as a result of phosphorus retention, increased stimulation of the parathyroid glands, and increased parathormone secretion.

Clinical Manifestations

The patient may have no symptoms or may experience signs and symptoms resulting from involvement of several body systems. Apathy, fatigue, muscle weakness, nausea, vomiting, constipation, hypertension, and cardiac arrhythmias may occur. All of these signs and symptoms are attributable to the increased concentration of calcium in the blood. Psychological effects may vary from irritability and neurosis to psychoses caused by the direct action of calcium on the brain and nervous system. An increase in calcium produces a decrease in the excitation potential of nerve and muscle tissue.

Nephrolithiasis (formation of stones in one or both kidneys), related to the increased urinary excretion of calcium and phosphorus, is one of the major complications of hyperparathyroidism. Although the incidence is on the decline in the United States, nephrolithiasis occurs in 15% to 20% of newly diagnosed patients (Yalla & Hickey, 2019). Kidney damage can result from the precipitation of calcium phosphate in the renal pelvis and parenchyma, which causes renal calculi, obstruction, pyelonephritis, and kidney injury.

Musculoskeletal symptoms accompanying hyperparathyroidism may be caused by demineralization of the bones or by bone tumors composed of benign giant cells resulting from overgrowth of osteoclasts. The patient may develop skeletal pain and tenderness, especially of the back and joints; pain on weight bearing; pathologic fractures; deformities; and shortening of body stature. Bone loss attributable to hyperparathyroidism increases the risk of fracture.

The incidence of peptic ulcer and pancreatitis is increased with hyperparathyroidism and may be responsible for many of the GI symptoms that occur.

Assessment and Diagnostic Findings

Primary hyperparathyroidism is diagnosed by persistent elevation of serum calcium levels and an elevated concentration of parathormone. Radioimmunoassays for parathormone are sensitive and differentiate primary hyperparathyroidism from other causes of hypercalcemia in more than 80% of patients with elevated serum calcium levels (Silverberg & Fuleihan, 2019). An elevated serum calcium level alone is a nonspecific finding, because serum levels may be altered by diet, medications, and kidney and bone changes. Bone changes may be detected on x-ray or bone scans in advanced disease. The

double-antibody parathyroid hormone test is used to distinguish between primary hyperparathyroidism and malignancy as a cause of hypercalcemia. Ultrasound, MRI, thallium scan, and fine-needle biopsy have been used to evaluate the function of the parathyroid glands and to localize parathyroid cysts, adenomas, or hyperplasia.

Medical Management

Surgical Management

The recommended treatment for primary hyperparathyroidism is parathyroidectomy, the surgical removal of abnormal parathyroid tissue (Silverberg & Fuleihan, 2019). In the past, the standard parathyroidectomy involved a bilateral neck exploration under general anesthesia. Today, minimally invasive parathyroidectomy techniques allow for unilateral neck exploration using local anesthesia; these are performed on an outpatient basis. In some cases, only the removal of a single diseased gland is necessary, reducing morbidity rates associated with surgery. For asymptomatic patients who have only mildly elevated serum calcium concentrations and normal kidney function, surgery may be delayed and the patient monitored closely for worsening of hypercalcemia, bone deterioration, renal impairment, or the development of kidney stones.

Surgery is recommended for patients who are asymptomatic and who meet one or more of the following criteria: younger than 50 years; unable or unlikely to participate in follow-up care; serum calcium level more than 1 mg/dL (0.25 mmol/L) above normal reference range; GFR less than 60 mL/min; urinary calcium level greater than 400 mg per day (10 mmol per day); bone density at hip, lumbar spine, or distal radius with T score less than -2.5 or previous fracture at any site; or nephrolithiasis or nephrocalcinosis (Yalla & Hickey, 2019).

Hydration Therapy

Patients with hyperparathyroidism are at risk for renal calculi. Therefore, a daily fluid intake of 2000 mL or more is encouraged to help prevent calculus formation. The patient is instructed to report other manifestations of renal calculi, such as abdominal pain and hematuria. Thiazide diuretics are avoided, because they decrease the renal excretion of calcium and further elevate serum calcium levels. Because of the risk of hypercalcemic crisis (see later discussion), the patient is instructed to avoid dehydration and to seek immediate health care if conditions that commonly produce dehydration (e.g., vomiting, diarrhea) occur.

Mobility

The nurse encourages the patient to be mobile. The patient with limited mobility is encouraged to walk. Bones subjected to the normal stress of walking give up less calcium. Bed rest increases calcium excretion and the risk of renal calculi. Oral phosphates lower the serum calcium level in some patients; long-term use is not recommended because of the risk of ectopic calcium phosphate deposition in soft tissues.

Diet and Medications

Nutritional needs are met, but the patient is advised to avoid a diet with restricted or excess calcium. If the patient has a coexisting peptic ulcer, prescribed antacids and protein feedings are necessary. Because anorexia is common, efforts are made to improve the appetite. Prune juice, stool softeners, and physical activity, along with increased fluid intake, help offset constipation, which is common postoperatively.

Nursing Management

The insidious onset and chronic nature of hyperparathyroidism along with its diverse and commonly vague symptoms may result in depression and frustration. The family may have considered the patient's illness to be psychosomatic. An awareness of the course of the disorder and an understanding approach by the nurse may help the patient and family deal with their reactions and feelings.

The nursing management of the patient undergoing parathyroidectomy is essentially the same as that of a patient undergoing thyroidectomy. However, the previously described precautions about airway patency, dehydration, immobility, and diet are particularly important in the patient who is awaiting or recovering from parathyroidectomy. Although not all parathyroid tissue is removed during surgery in an effort to control the calcium–phosphorus balance, the nurse closely monitors the patient to detect symptoms of tetany (which may be an early postoperative complication). Most patients quickly regain function of the remaining parathyroid tissue and experience only mild, transient postoperative hypocalcemia. In patients with significant bone disease or bone changes, a more prolonged period of hypocalcemia should be anticipated. The nurse educates the patient and family about the importance of follow-up laboratory testing to ensure return of serum calcium levels to normal (see [Chart 45-9](#)).

Chart 45-9



HOME CARE CHECKLIST

The Patient with Hyperparathyroidism

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

- State the impact of hyperparathyroidism and treatment on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- Describe potential benefits and risks of parathyroidectomy.
- State the name, dose, side effects, frequency, and schedule for all medications.
- Explain the purpose, dose, route, schedule, side effects, and precautions of pharmacologic treatment (loop diuretics, phosphates, and calcitonin) of hyperparathyroidism.
- State the need to contact primary provider before taking over-the-counter medication containing calcium.
- State changes in lifestyle (e.g., diet, activity) necessary to maintain health, including:
 - Recommended intake of dietary calcium.
 - Maintenance of regular bowel habits and management of constipation (e.g., prune juice, stool softeners, increased physical activity and fluids).
 - Monitoring of fluid intake as indicated, generally 2000 mL daily.
 - Managing pain (medication and nonpharmacologic interventions).
 - Increasing mobility as indicated.
 - Relate how to reach primary provider with questions or complications.
 - State time and date of follow-up medical appointments, therapy, and testing.
 - Identify sources of support (e.g., friends, relatives, faith community).
 - Identify the contact details for support services for patients and their caregivers/families.
 - Identify the need for health promotion, disease prevention, and screening activities.

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



Complications: Hypercalcemic Crisis

Acute hypercalcemic crisis can occur with extreme elevation of serum calcium levels. Serum calcium levels greater than 13 mg/dL (3.25 mmol/L) result in neurologic, cardiovascular, and kidney symptoms that can be life-threatening (Fischbach & Fischbach, 2018). Rapid rehydration with large volumes of IV isotonic saline fluids to maintain urine output of 100 to 150 mL per hour is combined with administration of calcitonin (Shane & Berenson, 2019).

Calcitonin promotes renal excretion of excess calcium and reduces bone resorption. The saline infusion should be stopped and a loop diuretic may be needed if the patient develops edema. Dosage and rates of infusion depend on the patient profile. The patient should be monitored carefully for fluid overload. Loop diuretics are not recommended as initial therapy in the absence of heart failure and kidney insufficiency. Bisphosphonates are added to promote a sustained decrease in serum calcium levels by promoting calcium deposition in bone and reducing the GI absorption of calcium. Cytotoxic agents (e.g., mithramycin), calcitonin, and dialysis may be used in emergency situations to decrease serum calcium levels quickly.



Quality and Safety Nursing Alert

The patient in acute hypercalcemic crisis requires close monitoring for life-threatening complications (e.g., airway obstruction) and prompt treatment to reduce serum calcium levels.

A combination of calcitonin and corticosteroids is given in emergencies to reduce the serum calcium level by increasing calcium deposition in bone. Other agents that may be given to decrease serum calcium levels include bisphosphonates (e.g., etidronate, pamidronate) (Shane & Berenson, 2019).

Expert assessment and care are required to minimize complications and reverse the life-threatening hypercalcemia. Medications are given with care, and attention is given to fluid balance to promote return of normal fluid and electrolyte balance. Supportive measures are necessary for the patient and family. See [Chapter 12, Table 12-13](#), for further discussion of hypercalcemic crisis.

Hypoparathyroidism

Hypoparathyroidism is caused by abnormal parathyroid development, destruction of the parathyroid glands (surgical removal or autoimmune response), and vitamin D deficiency. The most common cause is the near-total removal of the thyroid gland. The result is inadequate secretion of parathormone (Goltzman, 2019).

Deficiency of parathormone results in hyperphosphatemia (increased blood phosphate levels) and hypocalcemia (decreased blood calcium levels). In the absence of parathormone, there is decreased intestinal absorption of dietary calcium and decreased resorption of calcium from bone and through the renal tubules. Decreased renal excretion of phosphate causes hypophosphaturia, and low serum calcium levels result in hypocalciumuria.

Clinical Manifestations

Hypocalcemia causes irritability of the neuromuscular system and contributes to the chief symptom of hypoparathyroidism—tetany. Tetany is general muscle hypertonia, with tremor and spasmodic or uncoordinated contractions occurring with or without efforts to make voluntary movements. Symptoms of latent tetany are numbness, tingling, and cramps in the extremities, and the patient complains of stiffness in the hands and feet. In overt tetany, the signs include bronchospasm, laryngeal spasm, carpopedal spasm (flexion of the elbows and wrists and extension of the carpophalangeal joints and dorsiflexion of the feet), dysphagia, photophobia, cardiac arrhythmias, and seizures. Other symptoms include anxiety, irritability, depression, and even delirium. ECG changes and hypotension also may occur.

Assessment and Diagnostic Findings

A positive Chvostek sign or a positive Troussseau sign suggests latent tetany. **Chvostek sign** is positive when a sharp tapping over the facial nerve just in front of the parotid gland and anterior to the ear causes spasm or twitching of the mouth, nose, and eye (see Fig. 10-8A). **Troussseau sign** is positive when carpopedal spasm is induced by occluding the blood flow to the arm for 3 minutes with a blood pressure cuff (see Fig. 10-8B). The diagnosis of hypoparathyroidism often is difficult because of the vague symptoms, such as aches and pains. Therefore, laboratory studies are especially helpful. Tetany develops at very low serum calcium levels. Serum phosphate levels are increased, and x-rays of bone show increased density. Calcification is detected on x-rays of the subcutaneous or paraspinal basal ganglia of the brain.

Medical Management

The goal of therapy is to increase the serum calcium level to 9 to 10 mg/dL (2.2 to 2.5 mmol/L) and to eliminate the symptoms of hypoparathyroidism and hypocalcemia. Management is determined by the underlying cause and patient profile. Treatment may include combinations of calcium, magnesium, and ergocalciferol or calcitriol, the latter being preferred. A thiazide diuretic (e.g., hydrochlorothiazide) may be given to help decrease urinary calcium excretion (Goltzman, 2019). Recombinant parathyroid hormone has been approved for the treatment of osteoporosis but not for hypoparathyroidism at this time (Goltzman, 2019).

When hypocalcemia and tetany occur after a thyroidectomy, the immediate treatment is administration of IV calcium gluconate. If this does not decrease neuromuscular irritability and seizure activity immediately, sedative agents such as pentobarbital may be given.

Because of neuromuscular irritability, the patient with hypocalcemia and tetany requires an environment that is free of noise, drafts, bright lights, or sudden movement. If the patient develops respiratory distress, a tracheostomy or mechanical ventilation may become necessary, along with medications that cause bronchodilation in order to provide respiratory support.

Therapy for chronic hypoparathyroidism is determined after serum calcium levels are obtained. A diet high in calcium and low in phosphorus is prescribed. Although milk, milk products, and egg yolk are high in calcium, they are restricted because they also contain high levels of phosphorus. Spinach also is avoided because it contains oxalate, which would form insoluble calcium substances. Oral tablets of calcium salts, such as calcium gluconate, may be used to supplement the diet. Aluminum hydroxide gel or aluminum carbonate also is given after meals to bind phosphate and promote its excretion through the GI tract.

Nursing Management

Nursing management of the patient with possible acute hypoparathyroidism includes the following:

- Care of postoperative patients who have undergone thyroidectomy, parathyroidectomy, or radical neck dissection is directed toward detecting early signs of hypocalcemia and anticipating signs of tetany, seizures, and respiratory difficulties.
- Calcium gluconate should be available for emergency IV administration. If the patient requiring administration of calcium gluconate has a cardiac disorder, is subject to arrhythmias, or is receiving digitalis, the calcium gluconate is given slowly and cautiously.
- Calcium and digitalis increase systolic contraction and also potentiate each other; this can produce potentially fatal arrhythmias. Consequently, the cardiac patient requires continuous cardiac monitoring and careful assessment.

An important aspect of nursing care is patient education about medications and diet therapy. The patient needs to know the reason for high calcium and low phosphate intake and the symptoms of hypocalcemia and hypercalcemia; they should know to contact the primary provider immediately if these symptoms occur (see [Chart 45-10](#)).

Chart 45-10



HOME CARE CHECKLIST

The Patient with Hypoparathyroidism

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

- State the impact of hypoparathyroidism and treatment on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- State purpose, dose, route, schedule, side effects, and precautions of prescribed medications (calcium, phosphate binders).
- State changes in lifestyle (e.g., diet, activity) necessary to maintain health, including:
 - Ensuring a diet high in calcium and vitamin D, low in phosphorous.
 - Alternating activity and rest periods.
- State precipitating factors and interventions for complications (seizure, cardiac arrhythmias, cardiac arrest).
- State necessary actions for seizure activity.
- Relate how to reach primary provider with questions or complications.
- State time and date of follow-up medical appointments, therapy, and testing.
- Identify sources of support (e.g., friends, relatives, faith community).
- Identify the contact details for support services for patients and their caregivers/families.
- Identify the need for health promotion, disease prevention, and screening activities.

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

THE ADRENAL GLANDS

Anatomic and Physiologic Overview

Each person has two adrenal glands, one attached to the upper portion of each kidney. Each adrenal gland is, in reality, two endocrine glands with separate, independent functions. The adrenal medulla at the center of the gland secretes catecholamines, and the outer portion of the gland, the adrenal cortex, secretes steroid hormones (Norris, 2019). The secretion of hormones from the adrenal cortex is regulated by the hypothalamic–pituitary–adrenal axis. The hypothalamus secretes corticotropin-releasing hormone (CRH), which stimulates the pituitary gland to secrete ACTH, which in turn stimulates the adrenal cortex to secrete glucocorticoid hormone (cortisol). Increased levels of the adrenal hormone then inhibit the production or secretion of CRH and ACTH. This system is an example of a negative feedback mechanism.

Adrenal Medulla

The adrenal medulla functions as part of the autonomic nervous system. Stimulation of preganglionic sympathetic nerve fibers, which travel directly to the cells of the adrenal medulla, causes release of the catecholamine hormones epinephrine and norepinephrine. About 90% of the secretion of the human adrenal medulla is epinephrine (also called *adrenaline*). Catecholamines regulate metabolic pathways to promote catabolism of stored fuels to meet caloric needs from endogenous sources. The major effects of epinephrine release are to prepare to meet a challenge (fight-or-flight response). Secretion of epinephrine causes decreased blood flow to tissues that are not needed in emergency situations, such as the GI tract, and increased blood flow to tissues that are important for effective fight or flight, such as cardiac and skeletal muscle. Catecholamines also induce the release of free fatty acids, increase the basal metabolic rate, and elevate the blood glucose level.

Adrenal Cortex

A functioning adrenal cortex is necessary for life; adrenocortical secretions make it possible for the body to adapt to stress of all kinds. The three types of steroid hormones produced by the adrenal cortex are glucocorticoids, mainly cortisol; mineralocorticoids, mainly aldosterone; and sex hormones, mainly androgens (Norris, 2019). Without the adrenal cortex, severe stress would cause peripheral circulatory failure, circulatory shock, and prostration. Survival in the absence of a functioning adrenal cortex is possible only with nutritional, electrolyte, and fluid replacement and appropriate replacement with exogenous adrenocortical hormones.

Glucocorticoids

The **glucocorticoids** are so named because they have an important influence on glucose metabolism: Increased cortisol secretion results in elevated blood glucose levels. However, the glucocorticoids have major effects on the metabolism of almost all organs of the body. Glucocorticoids are secreted from the adrenal cortex in response to the release of ACTH from the anterior lobe of the pituitary gland. This system represents an example of negative feedback. The presence of glucocorticoids in the blood inhibits the release of CRH from the hypothalamus and also inhibits ACTH secretion from the pituitary. The resultant decrease in ACTH secretion causes diminished release of glucocorticoids from the adrenal cortex.

Corticosteroids are the classification of drugs that include glucocorticoids. These drugs are given to inhibit the inflammatory response to tissue injury and to suppress allergic manifestations. Their side effects include the development of diabetes, osteoporosis, and peptic ulcer; increased protein breakdown

resulting in muscle wasting and poor wound healing; and redistribution of body fat. When large doses of exogenous glucocorticoids are given, the release of ACTH and endogenous glucocorticoids are inhibited. This can cause the adrenal cortex to atrophy. If exogenous glucocorticoid administration is discontinued suddenly, adrenal insufficiency results because of the inability of the atrophied cortex to respond adequately.

Mineralocorticoids

Mineralocorticoids exert their major effects on electrolyte metabolism. They act principally on the renal tubular and GI epithelium to cause increased sodium ion absorption in exchange for excretion of potassium or hydrogen ions. ACTH only minimally influences aldosterone secretion. It is primarily secreted in response to the presence of angiotensin II in the bloodstream. Angiotensin II is a substance that elevates the blood pressure by constricting arterioles. Its concentration is increased when renin is released from the kidney in response to decreased perfusion pressure. The resultant increased aldosterone levels promote sodium reabsorption by the kidney and the GI tract, which tends to restore blood pressure to normal. The release of aldosterone is also increased by hyperkalemia. Aldosterone is the main hormone for the long-term regulation of sodium balance.

Adrenal Sex Hormones (Androgens)

Androgens, the third major type of steroid hormones produced by the adrenal cortex, exert effects similar to those of male sex hormones. The adrenal gland may also secrete small amounts of some estrogens, or female sex hormones. ACTH controls the secretion of adrenal androgens. When secreted in normal amounts, the adrenal androgens have little effect, but when secreted in excess, they produce masculinization in women, feminization in men, or premature sexual development in children. This is called the adrenogenital syndrome.

Pheochromocytoma

Pheochromocytoma is a rare tumor that is usually benign and originates from the chromaffin cells of the adrenal medulla. This tumor is the cause of high blood pressure in 0.1% of patients with hypertension and is usually fatal if undetected and untreated; however, it is usually cured by surgery. In 90% of patients the tumor arises in the medulla; in the remaining patients, it occurs in the extra-adrenal chromaffin tissue located in or near the aorta, ovaries, spleen, or other organs. Pheochromocytoma may occur at any age, but its peak incidence is between 40 and 50 years of age and affects men and women equally (U.S. National Library of Medicine, 2019). Ten percent of the tumors are bilateral, and 10% are malignant. Because of the high incidence of

pheochromocytoma in family members of affected people, the patient's family members should be alerted and screened for this tumor. Pheochromocytoma may occur in the familial form as part of multiple endocrine neoplasia type 2; therefore, it should be considered a possibility in patients who have medullary thyroid carcinoma and parathyroid hyperplasia or tumor.

Clinical Manifestations

The nature and severity of symptoms of functioning tumors of the adrenal medulla depend on the relative proportions of epinephrine and norepinephrine secretion. The typical triad of symptoms is headache, diaphoresis, and palpitations in the patient with hypertension. Hypertension and other cardiovascular disturbances are common. The hypertension may be intermittent or persistent. If the hypertension is sustained, it may be difficult to distinguish from other causes of hypertension. Other symptoms may include tremor, headache, flushing, and anxiety. Hyperglycemia may result from conversion of liver and muscle glycogen to glucose due to epinephrine secretion; insulin may be required to maintain normal blood glucose levels.

The clinical picture in the paroxysmal form of pheochromocytoma is usually characterized by acute, unpredictable attacks lasting seconds or several hours. Symptoms usually begin abruptly and subside slowly. During these attacks, the patient is extremely anxious, tremulous, and weak. The patient may experience headache, vertigo, blurring of vision, tinnitus, air hunger, and dyspnea. Other symptoms include polyuria, nausea, vomiting, diarrhea, abdominal pain, and a feeling of impending doom. Palpitations and tachycardia are common (Singh & Herrick, 2019). Blood pressures exceeding 250/150 mm Hg have been recorded. Such blood pressure elevations are life-threatening and can cause severe complications, such as cardiac arrhythmias, dissecting aneurysm, stroke, and acute kidney failure. Orthostatic hypotension (decrease in systolic blood pressure, lightheadedness, dizziness on standing) occurs in 70% of patients with untreated pheochromocytoma.

Assessment and Diagnostic Findings

Pheochromocytoma is suspected if signs of sympathetic nervous system overactivity occur in association with marked elevation of blood pressure. These signs can be associated with the “five Hs”: **h**ypertension, **h**eadache, **h**yperhidrosis (excessive sweating), **h**ypermetabolism, and **h**yperglycemia. The presence of these signs is highly predictive of pheochromocytoma. Paroxysmal symptoms of pheochromocytoma commonly develop in the fifth decade of life.

Measurements of urine and plasma levels of catecholamines and metanephrine (MN), a catecholamine metabolite, are the most direct and

conclusive tests for overactivity of the adrenal medulla. A test for detecting pheochromocytoma measures free MN in plasma by high-pressure liquid chromatography and electrochemical detection. A negative test result virtually excludes pheochromocytoma. Measurements of catecholamine metabolites (MN and vanillylmandelic acid [VMA]) or free catecholamines have been extensively used in the clinical setting. In most cases, pheochromocytoma can be diagnosed or confirmed based on a properly collected 24-hour urine sample. Levels can be as high as two times the normal limit. A 24-hour urine specimen is collected to detect free catecholamines, MN, and VMA; the use of combined tests increases the diagnostic accuracy of testing. A number of medications and foods, such as coffee and tea (including decaffeinated varieties), bananas, chocolate, vanilla, and aspirin, may alter the results of these tests; therefore, careful instructions to avoid restricted items must be given to the patient. Urine collected over a 2- or 3-hour period after an attack of hypertension can be assayed for catecholamine content (Singh & Herrick, 2019).

The total plasma catecholamine (epinephrine and norepinephrine) concentration is measured with the patient supine and at rest for 30 minutes. To prevent elevation of catecholamine levels resulting from the stress of venipuncture, a butterfly needle, scalp vein needle, or venous catheter may be inserted 30 minutes before the blood specimen is obtained.

Factors that may elevate catecholamine concentrations must be controlled to obtain valid results; these factors include consumption of coffee or tea (including decaffeinated varieties), the use of tobacco, emotional and physical stress, and the use of many prescription and over-the-counter medications (e.g., amphetamines, nose drops or sprays, decongestant agents, bronchodilators).

Normal plasma values of epinephrine are 100 pg/mL (590 pmol/L); normal values of norepinephrine are generally less than 100 to 550 pg/mL (590 to 3240 pmol/L). Values of epinephrine greater than 400 pg/mL (2180 pmol/L) or norepinephrine values greater than 2000 pg/mL (11,800 pmol/L) are considered diagnostic of pheochromocytoma. Values that fall between normal levels and those diagnostic of pheochromocytoma indicate the need for further testing.

A clonidine suppression test may be performed if the results of plasma and urine tests of catecholamines are inconclusive. Clonidine is a centrally acting antiadrenergic medication that suppresses the release of neurogenically mediated catecholamines. The suppression test is based on the principle that catecholamine levels are normally increased through the activity of the sympathetic nervous system. In pheochromocytoma, increased catecholamine levels result from the diffusion of excess catecholamines into the circulation, bypassing normal storage and release mechanisms. Therefore, in patients with pheochromocytoma, clonidine does not suppress the release of catecholamines (Singh & Herrick, 2019).

Imaging studies, such as CT, MRI, and ultrasonography, may also be carried out to localize the pheochromocytoma and to determine whether more than one tumor is present. The use of ^{131}I -metaiodobenzylguanidine (MIBG) scintigraphy may be required to determine the location of the pheochromocytoma and to detect metastatic sites outside the adrenal gland. MIBG is a specific isotope for catecholamine-producing tissue. It has been helpful in identifying tumors not detected by other tests or procedures. MIBG scintigraphy is a noninvasive, safe procedure that has increased the accuracy of diagnosis of adrenal tumors (Fischbach & Fischbach, 2018).

Other diagnostic studies may focus on evaluating the function of other endocrine glands because of the association of pheochromocytoma in some patients with other endocrine tumors.

Medical Management

During an episode or attack of hypertension, tachycardia, anxiety, and the other symptoms of pheochromocytoma, bed rest with the head of the bed elevated is prescribed to promote an orthostatic decrease in blood pressure.

Pharmacologic Therapy

The patient may be treated preoperatively on an inpatient or outpatient basis. Regardless of the setting, monitoring of blood pressure and cardiac function is essential. The goals are to control hypertension before and during surgery and volume expansion, and to prevent a catecholamine storm as a result of surgery (Singh & Herrick, 2019).

Preoperatively, the patient may begin treatment with a low dose of an alpha-adrenergic blocker, either phenoxybenzamine or doxazosin, 10 to 14 days or longer prior to surgery (Singh & Herrick, 2019). The patient should be informed about the potential for adverse effects of these medications, which include orthostasis, nasal stuffiness, increased fatigue, and retrograde ejaculation in men. The medication dosages are started at a low dose and increased every 2 to 3 days as needed to control blood pressure. Patients may be required to consume a high-sodium diet or take salt supplements.

After administration of the alpha-blockers, the blood pressure should be monitored closely. In an outpatient setting, the blood pressure should be taken twice daily in a sitting and standing position. The targets are less than 130/80 mm Hg (seated) with a standing systolic pressure greater than 90 mm Hg and target heart rate of 60 to 70 beats per minute sitting and 70 to 80 beats per minute standing (Singh & Herrick, 2019). Age and comorbid disease should be taken into consideration when establishing and evaluating targets. Propranolol and metoprolol may be administered with caution to achieve the target heart rate (Singh & Herrick, 2019).

Calcium channel blockers such as nifedipine are sometimes used as an alternative or supplement to preoperative alpha- and beta-blockers, when blood pressure control is inadequate or the patient is unable to tolerate the side effects. Nifedipine and nicardipine may be used safely without causing undue hypotension. For episodes of severe hypertension, nifedipine is a fast and effective treatment, because the capsules can be pierced and chewed. The patient needs to be well hydrated before, during, and after surgery to prevent hypotension. Additional medications that may be used preoperatively include catecholamine synthesis inhibitors, such as alpha-methyl-*p*-tyrosine (metyrosine). These are occasionally used if adrenergic blocking agents (i.e., alpha- and beta-blockers) are not effective. Long-term use of metyrosine may result in many adverse effects, including sedation, depression, diarrhea, anxiety, nightmares, dysuria, impotence, elevated aspartate aminotransferase, anemia, thrombocytopenia, crystalluria, galactorrhea (breast discharge), and extrapyramidal signs (e.g., drooling, speech impairment, tremors).

Surgical Management

The definitive treatment of pheochromocytoma is surgical removal of the tumor, usually with **adrenalectomy** (removal of one or both adrenal glands); surgical treatment is considered high risk in this patient population. Surgery may be performed using a laparoscopic approach or an open operation. The laparoscopic approach is the preferred method for patients with pheochromocytomas including large tumors because of the decreased blood loss, decreased hospitalization time, and decreased morbidity. Bilateral adrenalectomy may be necessary if tumors are present in both adrenal glands. Patient preparation includes control of blood pressure and blood volumes; usually, this is carried out over 10 to 14 days, as described previously. A calcium channel blocker (nicardipine) can be given intraoperatively exclusively or in combination with the alpha- and beta-blockers to control blood pressure.

A hypertensive crisis, however, can still arise as a result of manipulation of the tumor during surgical excision, causing a release of stored epinephrine and norepinephrine, with marked increases in blood pressure and changes in heart rate. Exploration of other possible tumor sites is frequently undertaken to ensure removal of all tumor tissue. As a result, the patient is subject to the stress and effects of a long surgical procedure, which may increase the risk of hypertension postoperatively.

Corticosteroid replacement is required if bilateral adrenalectomy has been necessary. Corticosteroids may also be required for the first few days or weeks after removal of a single adrenal gland. IV administration of corticosteroids (methylprednisolone) may begin on the evening before surgery and continue during the early postoperative period to prevent adrenal insufficiency. Oral

preparations of corticosteroids (prednisone) are prescribed after the acute stress of surgery diminishes.

Hypotension and hypoglycemia may occur in the postoperative period because of the sudden withdrawal of excessive amounts of catecholamines. Therefore, careful attention is directed toward monitoring and treating these changes. Hypertension may continue if not all pheochromocytoma tissue was removed, if pheochromocytoma recurs, or if the blood vessels were damaged by severe and prolonged hypertension. Several days after surgery, urine and plasma levels of catecholamines and their metabolites are measured to determine whether the surgery was successful.

Nursing Management

The patient who has undergone surgery to treat pheochromocytoma has experienced a stressful preoperative and postoperative course and may remain fearful of repeated attacks. Although it is usually expected that all pheochromocytoma tissue has been removed, there is a possibility that other sites were undetected and that attacks may recur. The patient is monitored until stable with special attention given to ECG changes, arterial pressures, fluid and electrolyte balance, and blood glucose levels. IV access will be required for administration of fluids and medications.

Promoting Home, Community-Based, and Transitional Care



Educating Patients About Self-Care

During the pre- and postoperative phases of care, the nurse educates the patient about the importance of follow-up monitoring to ensure that pheochromocytoma does not recur undetected. After adrenalectomy, the use of corticosteroids may be needed. Therefore, the nurse educates the patient about their purpose, the medication schedule, and the risks of skipping doses or stopping their administration abruptly.

The patient and family are educated about how to measure the patient's blood pressure and when to notify the primary provider about changes in blood pressure. In addition, the nurse provides verbal and written instructions about the procedure for collecting 24-hour urine specimens to monitor urine catecholamine levels.

Continuing and Transitional Care

A follow-up visit from a home, community-based or transitional care nurse may be indicated to assess the patient's postoperative recovery, surgical incision, knowledge regarding medication, and adherence to the medication

schedule. This may help reinforce previous education about management and monitoring. The nurse also obtains blood pressure measurements and assists the patient in preventing or dealing with problems that may result from long-term use of corticosteroids.

Because of the risk of recurrence of hypertension, periodic checkups are required, especially in young patients and in those whose families have a history of pheochromocytoma. The patient is scheduled for periodic follow-up appointments to observe for return of normal blood pressure and plasma and urine levels of catecholamines.

Adrenocortical Insufficiency (Addison's Disease)

Primary adrenal insufficiency, also called **Addison's disease**, is the result of dysfunction of the hypothalamus–pituitary gland–adrenal gland feedback loop which results in insufficient production of steroids by the adrenal glands (Norris, 2019). Addison's disease is considered rare. In 70% to 90% of cases, the cause is an autoimmune disorder but tuberculosis and histoplasmosis are also associated with destruction of adrenal tissue; therefore, both should be considered in the diagnostic workup. Other causes include surgical removal of both adrenal glands; medications such as rifampin, barbiturates, ketoconazole, and tyrosine kinase inhibitors; and metastatic cancers such as lung, breast, colon, and melanoma (Zhang & Carmichael, 2019). Secondary adrenal insufficiency may result from the sudden cessation of exogenous adrenocortical hormonal therapy, which suppresses the body's normal response to stress and interferes with normal feedback mechanisms. Treatment with daily administration of corticosteroids for 2 to 4 weeks may suppress function of the adrenal cortex; therefore, adrenal insufficiency should be considered in any patient who has been treated with corticosteroids (Zhang & Carmichael, 2019).

Clinical Manifestations

The loss of mineralocorticoids leads to increased excretion of sodium, chloride, and water with increased retention of potassium. This may lead to a deficiency in extracellular fluid causing decreased cardiac output. The loss of glucocorticoids results in hypoglycemia with complaints of muscle weakness, lethargy, and GI symptoms including anorexia, weight loss, nausea and vomiting. In addition, the increase in levels of ACTH results in hyperpigmentation of the skin and mucous membranes, especially of the knuckles, knees, and skin folds (Norris, 2019).

Patients with Addison's disease are at risk to develop an **Addisonian crisis**, a life-threatening complication in which severe hypotension, cyanosis, fever,

nausea, vomiting, and signs of shock develop. In addition, the patient may have pallor; complain of headache, abdominal pain, and diarrhea; and may show signs of confusion and restlessness. Even slight overexertion, exposure to cold, acute infection, or a decrease in salt intake may lead to circulatory collapse, shock, and death, if untreated. The stress of surgery or dehydration resulting from preparation for diagnostic tests or surgery may precipitate an Addisonian or hypotensive crisis because of the inhibited feedback loop.

Assessment and Diagnostic Findings

Although the clinical manifestations presented appear specific, the onset of Addison's disease usually occurs with nonspecific symptoms. The diagnosis is confirmed by laboratory test results. Combined measurements of early-morning serum cortisol and plasma ACTH are performed to differentiate primary adrenal insufficiency from secondary adrenal insufficiency and from normal adrenal function. Patients with primary insufficiency have a greatly increased plasma ACTH level and a serum cortisol concentration lower than the normal range or in the low-normal range (Zhang & Carmichael, 2019). Other laboratory findings include hypoglycemia (decreased levels of blood glucose), hyponatremia (decreased levels of sodium), hyperkalemia (increased serum potassium concentration), and leukocytosis (increased white blood cell count).

Medical Management

Immediate treatment is directed toward combating circulatory shock: restoring blood circulation, administering fluids and corticosteroids, monitoring vital signs, and placing the patient in a recumbent position with the legs elevated. Hydrocortisone is administered by IV, followed by 3 to 4 L of normal saline or 5% dextrose solution. Vasopressors may be required if hypotension persists.

Antibiotics may be given if infection has precipitated adrenal crisis in a patient with chronic adrenal insufficiency. In addition, the patient is assessed closely to identify other factors, stressors, or illnesses that led to the acute episode.

Oral intake may be initiated as soon as tolerated. IV fluids are gradually decreased after oral fluid intake is adequate to prevent hypovolemia. If the adrenal gland does not regain function, the patient needs lifelong replacement of corticosteroids and mineralocorticoids to prevent recurrence of adrenal insufficiency. Patients who are undergoing stressful procedures, surgery, significant illnesses, or are in the third trimester of pregnancy require additional supplementary therapy with corticosteroid medications to prevent Addisonian crisis. In addition, dietary intake may need to be supplemented

with additional salt to manage GI losses of fluids through vomiting and diarrhea (Zhang & Carmichael, 2019).

Nursing Management

Assessing the Patient

The health history and examination focus on the presence of symptoms of fluid imbalance and the patient's level of stress. The nurse should monitor the blood pressure and pulse rate as the patient moves from a lying, sitting, and standing position to assess for inadequate fluid volume. A decrease in systolic pressure (20 mm Hg or more) may indicate depletion of fluid volume, especially if accompanied by symptoms. The skin should be assessed for changes in color and turgor, which could indicate chronic adrenal insufficiency and hypovolemia. The patient is assessed for change in weight, muscle weakness, fatigue, and any illness or stress that may have precipitated the acute crisis.

Monitoring and Managing Addisonian Crisis

The patient at risk is monitored for signs and symptoms indicative of Addisonian crisis, which can include shock; hypotension; rapid, weak pulse; rapid respiratory rate; pallor; and extreme weakness (see [Chapter 11](#)). Physical and psychological stressors such as cold exposure, overexertion, infection, and emotional distress should be avoided.

The patient with Addisonian crisis requires immediate treatment with IV administration of fluid, glucose, and electrolytes, especially sodium; replacement of missing steroid hormones; and vasopressors. The nurse anticipates and meets the patient's needs to promote return to a precrisis state.

Restoring Fluid Balance

The nurse encourages the patient to consume foods and fluids that assist in restoring and maintaining fluid and electrolyte balance which in turn maintains adequate cardiac output. Along with the dietitian, the nurse helps the patient select foods high in sodium during GI disturbances and in very hot weather.

The nurse educates the patient and family to administer hormone replacement as prescribed and to modify the dosage during illness and other stressful situations. Written and verbal instructions are provided about the administration of exogenous glucocorticoids (i.e., corticosteroid medications such as hydrocortisone, cortisone, and prednisone) and mineralocorticoids (fludrocortisone) as prescribed. The patient should be instructed to take prescribed corticosteroids with antacids or meals. The patient should be informed that the steroid therapy usually corrects the mood swings and mental status changes which adrenal insufficiency frequently causes (Quintanar, 2019).

Improving Activity Tolerance

Until the patient's condition is stabilized, the nurse takes precautions to avoid unnecessary activity and stress that could precipitate another hypotensive episode. Efforts are made to detect signs of infection or the presence of other stressors. Explaining the rationale for minimizing stress during the acute crisis assists the patient to increase activity gradually.

Promoting Home, Community-Based, and Transitional Care



Educating Patients About Self-Care

Because of the need for lifelong replacement of adrenal cortex hormones to prevent Addisonian crises, the patient and family members receive explicit education about the rationale for replacement therapy and proper dosage. In addition, the patient, family, and caregivers are educated about the signs of excessive or insufficient hormone replacement. Stress can precipitate an Addisonian crisis, and in times of stress the usual dose may need to be adjusted. The patient should have an emergency kit available with syringe and either hydrocortisone or dexamethasone as prescribed by the primary provider (Zhang & Carmichael, 2019). Specific verbal and written instructions about how and when to use the injection are also provided to the patient and family or caregivers. Chart 45-11 summarizes education for patients with Addison's disease and their caregivers.

Continuing and Transitional Care

Although most patients can return to their job and family responsibilities soon after hospital discharge, others cannot do so because of concurrent illnesses or incomplete recovery from the episode of adrenal insufficiency. In these circumstances, a referral for home, community-based or transitional care enables the nurse to assess the patient's recovery, monitor hormone replacement, and evaluate stress in the home. The nurse assesses the patient's and family's knowledge about medication therapy and dietary modifications and provides education as needed. The home health nurse educates the patient and family on the importance of keeping follow-up visits with the primary provider and participating in health promotion activities and health screening.

Chart 45-11



HOME CARE CHECKLIST

The Patient with Adrenal Insufficiency (Addison's Disease)

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

- State the impact of adrenal insufficiency and treatment on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- State the purpose, dose, route, schedule, side effects, and precautions of prescribed medications (corticosteroid replacement).
- State that compliance with medical regimen is lifelong.
- Recognize the need for dosage adjustment during times of stress.
- State changes in lifestyle (e.g., diet, activity) necessary to maintain health, including:
 - Wearing medical alert identification, and carrying medical information card.
 - Avoiding strenuous activity in hot, humid weather.
 - Identifying strategies for dealing with stress and avoiding adrenal crisis.
 - Notifying primary providers about disease before treatment or procedure.
 - Increasing fluid intake and salt with excessive perspiration.
 - Ensuring high-carbohydrate, high-protein diet with adequate sodium intake.
 - State warning signs of adrenal crisis and the need for emergency care.
 - Explain components of an emergency kit and indications for their use; demonstrate how to use them.
 - Relate how to reach primary provider with questions or complications.
 - State time and date of follow-up medical appointments, therapy, and testing.
 - Identify sources of support (e.g., friends, relatives, faith community).
 - Identify the contact details for support services for patients and their caregivers/families.
 - Identify the need for health promotion, disease prevention, and screening activities.

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

Cushing's Syndrome

The most common cause of Cushing's syndrome (also known as Cushing's disease) is the use of corticosteroid medications, but the syndrome can also be due to excessive glucocorticoid production secondary to hyperplasia of the

adrenal cortex (Sadiq & Silverstein, 2019a). However, overproduction of endogenous glucocorticoids may be caused by several mechanisms, including a tumor of the pituitary gland that produces ACTH and stimulates the adrenal cortex to increase its hormone secretion despite production of adequate amounts. Primary hyperplasia of the adrenal glands in the absence of a pituitary tumor is less common. Another less common cause of Cushing's syndrome is the ectopic production of ACTH by malignancies; bronchogenic carcinoma is the most common type. Regardless of the cause, the normal feedback mechanisms that control the function of the adrenal cortex become ineffective, and the usual diurnal pattern of cortisol is lost. The signs and symptoms of Cushing's syndrome are primarily a result of oversecretion of glucocorticoids and androgens, although mineralocorticoid secretion may be affected as well (Norris, 2019).

Clinical Manifestations

When overproduction of the adrenocortical hormone occurs, arrest of growth, obesity, and musculoskeletal changes occur along with glucose intolerance. The classic picture of Cushing's syndrome in the adult is that of central-type obesity, with a fatty "buffalo hump" in the neck and supraclavicular areas, a heavy trunk, and relatively thin extremities. The skin is thin, fragile, and easily traumatized; ecchymoses (bruises) and striae develop. The patient complains of weakness and lassitude. Sleep is disturbed because of altered diurnal secretion of cortisol.

Excessive protein catabolism occurs, producing muscle wasting and osteoporosis. Kyphosis, backache, and compression fractures of the vertebrae may result. Retention of sodium and water occurs as a result of increased mineralocorticoid activity, producing hypertension and heart failure.

The patient develops a "moon-faced" appearance and may experience increased oiliness of the skin and acne. Hyperglycemia or overt diabetes may develop. The patient may also report weight gain, slow healing of minor cuts, and bruises.

Women between the ages of 20 and 40 years are five times more likely than men to develop Cushing's syndrome. In females of all ages, virilization may occur as a result of excess androgens. Virilization is characterized by the appearance of masculine traits and the recession of feminine traits. Hirsutism (excessive growth of hair on the face) occurs, the breasts atrophy, menses cease, the clitoris enlarges, and the voice deepens. Libido is lost in men and women. Distress and depression are common and are increased by the severity of the physical changes that occur with this syndrome. If Cushing's disease is a consequence of pituitary tumor, visual disturbances may occur because of pressure of the growing tumor on the optic chiasm. [Chart 45-12](#) summarizes the clinical manifestations of Cushing's syndrome.

Chart 45-12

Clinical Manifestations of Cushing's Syndrome

Cardiovascular

Heart failure
Hypertension

Dermatologic

Acne
Ecchymoses
Petechiae
Striae
Thinning of skin

Endocrine/Metabolic

Adrenal suppression
Altered calcium metabolism
Buffalo hump
Hyperglycemia
Hypokalemia
Impotence
Menstrual irregularities
Metabolic alkalosis
Moon face
Negative nitrogen balance
Sodium retention
Truncal obesity

Gastrointestinal

Pancreatitis
Peptic ulcer

Immune Function

Decreased inflammatory responses
Impaired wound healing
Increased susceptibility to infections

Muscular

Muscle weakness
Myopathy

Ophthalmic

Cataracts
Glaucoma

Psychiatric

Mood alterations
Psychoses

Skeletal

Aseptic necrosis of femur
Osteoporosis
Spontaneous fractures
Vertebral compression fractures



This woman with Cushing syndrome has several classic signs, including facial hair, buffalo hump, and moon face. Reprinted with permission from Rubin, R., Strayer, D. S., & Rubin, E. (2012). *Rubin's pathology* (6th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

Assessment and Diagnostic Findings

The three tests used to diagnose Cushing's syndrome are serum cortisol, urinary cortisol, and low-dose dexamethasone suppression tests. Two of these three tests need to be unequivocally abnormal to diagnose Cushing's syndrome. If the results of all three tests are normal, the patient likely does not have Cushing's syndrome (but may have a mild case, or the manifestations may be cyclic). For these patients, further testing is not recommended unless symptoms progress. If test results are either slightly abnormal or discordant, further testing is recommended.

Serum cortisol levels are usually higher in the early morning (6 to 8 am) and lower in the evening (4 to 6 pm). This variation is lost in patients with Cushing's syndrome (Fischbach & Fischbach, 2018).

A urinary cortisol test requires a 24-hour urine collection. The nurse instructs the patient how to collect and store the specimen. If the results of the urinary cortisol test are three times the upper limit of the normal range and one other test is abnormal, Cushing's syndrome can be assumed.

An overnight dexamethasone suppression test is used to diagnosis pituitary and adrenal causes of Cushing's syndrome. It can be performed on an outpatient basis. Dexamethasone (1 or 8 mg) is given orally late in the evening or at bedtime, and a plasma cortisol level is obtained at 8 am the next morning. Suppression of cortisol to less than 5 mg/dL indicates that the hypothalamic-pituitary-adrenal axis is functioning properly (Fischbach & Fischbach, 2018). Stress, obesity, depression, and medications such as anticonvulsant agents, estrogen (during pregnancy or as oral medications), and rifampin can falsely elevate cortisol levels.

Indicators of Cushing's syndrome include an increase in serum sodium and blood glucose levels and a decrease in serum potassium, a reduction in the number of blood eosinophils, and disappearance of lymphoid tissue. Measurements of plasma and urinary cortisol levels are obtained. Several blood samples may be collected to determine whether the normal diurnal variation in plasma levels is present; this variation is frequently absent in adrenal dysfunction. If several blood samples are required, they must be collected at the times specified, and the time of collection must be noted on the requisition slip.

Medical Management

If Cushing's syndrome is caused by pituitary tumors rather than tumors of the adrenal cortex, treatment is directed at the pituitary gland. Surgical removal of the tumor by transsphenoidal hypophysectomy (see [Chapter 61](#)) is the treatment of choice. Radiation of the pituitary gland has also been successful, although it may take several months for control of symptoms. Adrenalectomy is the treatment of choice in patients with unilateral primary adrenal hypertrophy. Medical management is recommended for bilateral adrenal dysplasia.

Postoperatively, symptoms of adrenal insufficiency may begin to appear 12 to 48 hours after surgery because of reduction of the high levels of circulating adrenal hormones. Temporary replacement therapy with hydrocortisone may be necessary for several months, until the adrenal glands begin to respond normally to the body's needs.

Adrenal enzyme inhibitors (e.g., metyrapone, aminoglutethimide, mitotane, and ketoconazole) may be used to reduce hyperadrenalinism if the syndrome is caused by ectopic ACTH secretion by a tumor that cannot be eradicated. Close monitoring is necessary, because symptoms of inadequate adrenal function may result, and side effects of the medications may occur.

If Cushing's syndrome is a result of the administration of corticosteroids, an attempt is made to reduce or taper the medication to the minimum dosage needed to treat the underlying disease process (e.g., autoimmune or allergic disease, rejection of a transplanted organ). Frequently, alternate-day therapy decreases the symptoms of Cushing's syndrome and allows recovery of the adrenal glands' responsiveness to ACTH.

Diabetes and peptic ulcer are common in patients with Cushing's syndrome. Therefore, insulin therapy and medication to prevent or treat peptic ulcer are initiated if needed. Before, during, and after surgery, blood glucose monitoring and assessment of stools for blood are carried out to monitor for these complications. If the patient has other symptoms of Cushing's syndrome, these are considered in the preoperative preparation. For example, if the patient has experienced weight gain, special instruction is given about postoperative breathing exercises.

NURSING PROCESS

The Patient with Cushing's Syndrome

Assessment

The health history and examination focus on the effects on the body of high concentrations of adrenal cortex hormones and on the inability of the adrenal cortex to respond to changes in cortisol and aldosterone levels. The history includes information about the patient's level of activity and ability to carry out routine and self-care activities. The skin is observed and assessed for trauma, infection, breakdown, bruising, and edema. Changes in physical appearance are noted, and the patient's responses to these changes are elicited. The nurse assesses the patient's mental function, including mood, responses to questions, awareness of environment, and level of depression. The family is often a good source of information about gradual changes in the patient's physical appearance as well as emotional status.

Diagnosis

NURSING DIAGNOSES

Based on the assessment data, major priority nursing diagnoses include the following:

- Risk for impaired cardiac function associated with changes in cardiac function
- Risk for injury associated with weakness
- Risk for infection associated with altered immune system function
- Impaired skin integrity associated with edema, impaired healing, and thin and fragile skin
- Disturbed body image associated with altered physical appearance, impaired sexual functioning, and decreased activity level
- Difficulty coping associated with mood swings, irritability, and depression

COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS

Potential complications may include the following:

- Addisonian crisis
- Adverse effects of adrenocortical activity

Planning and Goals

The major goals for the patient include maintenance of adequate cardiac function, decreased risk of injury, decreased risk of infection, improved skin integrity, improved body image, improved mental function, and absence of complications.

Nursing Interventions

MAINTAINING ADEQUATE CARDIAC FUNCTION

The patient who is taking corticosteroid medications should be monitored for the presence of hypertension and hypokalemia. The nurse assesses fluid and electrolyte status by monitoring laboratory values and daily weights. The patient should be educated about foods low in sodium to decrease fluid retention and foods high in potassium; referral to a dietitian may be useful. The patient should also be instructed to report and pedal edema or changes in activity tolerance.

DECREASING RISK OF INJURY

Establishing a protective environment helps prevent falls, fractures, and other injuries to bones and soft tissues. The patient who is very weak may require assistance from the nurse in ambulating to avoid falling or bumping into sharp corners of furniture. Foods high in protein, calcium, and vitamin D are recommended to minimize muscle wasting and osteoporosis. Referral to a dietitian may assist the patient in selecting appropriate foods that are also low in sodium and calories.

DECREASING RISK OF INFECTION

The patient should avoid unnecessary exposure to others with infections. The nurse frequently assesses the patient for subtle signs of infection, because the anti-inflammatory effects of corticosteroids may mask the common signs of inflammation and infection.

PROMOTING SKIN INTEGRITY

Meticulous skin care is necessary to avoid traumatizing the patient's fragile skin. The use of adhesive tape is avoided, because it can irritate the skin and tear the fragile tissue when the tape is removed. The nurse frequently assesses the skin and bony prominences and encourages and assists the patient to change positions frequently to prevent skin breakdown.

IMPROVING BODY IMAGE

The patient may benefit from discussion of the effect the changes have had on their self-concept and relationships with others. Weight gain and edema may be modified by a low-carbohydrate, low-sodium diet, and a high-protein intake may reduce some of the other bothersome symptoms. The patient may also benefit from discussion of the changes being temporary if the treatment with corticosteroids is temporary.

IMPROVING COPING

Explanations to the patient and family members about the cause of emotional instability are important in helping them cope with the mood swings, irritability, and depression that may occur. Psychotic behavior may occur in a few patients and should be reported. The nurse encourages the patient and family members to verbalize their feelings and concerns.

MONITORING AND MANAGING POTENTIAL COMPLICATIONS

Addisonian Crisis. The patient with Cushing's syndrome whose symptoms are treated by withdrawal of corticosteroids, by adrenalectomy, or by removal of a pituitary tumor is at risk for adrenal hypofunction and Addisonian crisis. If high levels of circulating adrenal hormones have suppressed the function of the adrenal cortex, atrophy of the adrenal cortex is likely. If the circulating hormone level is decreased rapidly because of surgery or abrupt cessation of corticosteroid agents, manifestations of adrenal hypofunction and Addisonian crisis may develop. Therefore, the patient with Cushing's syndrome should be assessed for signs and symptoms of Addisonian crisis as discussed previously. If Addisonian crisis occurs, the patient is treated for circulatory collapse and shock (see [Chapter 11](#)).

Adverse Effects of Adrenocortical Activity. The nurse assesses fluid and electrolyte status by monitoring laboratory values and daily weights. Because of the increased risk of glucose intolerance and hyperglycemia, blood glucose monitoring is initiated. The nurse reports elevated blood glucose levels to the primary provider so that treatment can be prescribed if needed. If indicated, the patient may need to be educated in self-monitoring of blood glucose and insulin injections.

PROMOTING HOME, COMMUNITY-BASED, AND TRANSITIONAL CARE



Educating Patients About Self-Care. The patient, family, and caregivers should be educated that acute adrenal insufficiency and underlying symptoms will recur if medication is stopped abruptly without medical supervision. The nurse stresses the need for dietary modifications to ensure adequate calcium intake without increasing the risks for hypertension, hyperglycemia, and weight gain. The nurse educates the patient and family about how to monitor blood pressure, blood glucose levels, and weight. Patients should be advised to wear a medical alert bracelet and to notify other health care providers (e.g., dentist) about their condition (see [Chart 45-13](#)).

Continuing and Transitional Care. The need for follow-up depends on the origin and duration of the disease and its management. The patient who has been treated by adrenalectomy or removal of a pituitary tumor requires close monitoring to ensure that adrenal function has returned to normal and adequacy of circulating adrenal hormones. Home care referral may be indicated to ensure a safe environment that minimizes stress and risk of falls and other side effects. The home health nurse assesses the patient's physical and psychological status and reports changes to the primary provider. The nurse also assesses the patient's understanding of and ability to manage the medication regimen and reinforces previous education about the medications and the importance of taking them as prescribed. The nurse

emphasizes the importance of regular medical follow-up, the side effects and toxic effects of medications, and the need to wear medical identification with Addison's and Cushing's diseases. In addition, the patient and family are reminded about the importance of health promotion activities and recommended health screening, including bone mineral density testing.

Chart 45-13



HOME CARE CHECKLIST

The Patient with Cushing's Syndrome

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

- State the impact of Cushing's syndrome and treatment on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- State the relationship between adrenal hormones, emotional state, and stress.
- State the purpose, dose, route, schedule, side effects, and precautions for prescribed medications (adrenocortical inhibitors).
- State importance of compliance with medical regimen.
- State the need to contact primary provider before taking over-the-counter medications.
- State changes in lifestyle (e.g., diet, activity) necessary to maintain health, including:
 - Wearing medical alert identification, and carrying medical information card.
 - Identifying methods for managing labile emotions.
 - Describing protective skin care measures and the use of protective devices and practices to decrease injury/fracture.
 - Identifying foods high in potassium and low in sodium, calories, and carbohydrates.
 - Describing measures to decrease risk of infection.
 - Balancing rest and activity.
 - Monitoring blood pressure, blood glucose levels, and weight.
 - Identify signs and symptoms of excessive and insufficient adrenal hormone.
 - Relate how to reach primary provider with questions or complications.
 - State time and date of follow-up medical appointments, therapy, and testing.
 - Identify sources of support (e.g., friends, relatives, faith community).
 - Identify the contact details for support services for patients and their caregivers/families.
 - Identify the need for health promotion, disease prevention, and screening activities.

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

Evaluation

Expected patient outcomes may include:

1. Maintains adequate cardiac function
 - a. Blood pressure is maintained within acceptable ranges
 - b. Potassium levels are within normal ranges
 - c. Absence of pedal edema
2. Decreases risk of injury
 - a. Is free of fractures or soft tissue injuries
 - b. Is free of ecchymotic areas
3. Decreases risk of infection
 - a. Experiences no temperature elevation, redness, pain, or other signs of infection or inflammation
 - b. Avoids contact with others who have infections
4. Attains/maintains skin integrity
 - a. Has intact skin, without evidence of breakdown or infection
 - b. Exhibits decreased edema in extremities and trunk
 - c. Changes position frequently and inspects bony prominences daily
5. Achieves improved body image
 - a. Verbalizes feelings about changes in appearance, sexual function, and activity level
 - b. States that physical changes are a result of excessive glucocorticoids
6. Exhibits improved thought processes
7. Exhibits absence of complications
 - a. Exhibits normal vital signs and weight and is free of symptoms of Addisonian crisis
 - b. Identifies signs and symptoms of adrenocortical hypofunction that should be reported and measures to take in case of severe illness and stress
 - c. Identifies strategies to minimize complications of Cushing's syndrome
 - d. Adheres to recommendations for follow-up appointments and health screening

Primary Aldosteronism

The principal action of aldosterone is to conserve body sodium. Under the influence of this hormone, the kidneys excrete less sodium and more potassium and hydrogen. Primary aldosteronism is also known as Conn syndrome. Etiologic factors for primary aldosteronism include tumors of the adrenal gland, ovarian tumors that secrete aldosterone, and a family history. The true incidence of primary aldosteronism is unknown (Ma & Baranski,

2019) but studies have indicated that it is underestimated (Monticone, Burrello, Tizzani, et al., 2017). Excessive production of aldosterone causes a distinctive pattern of biochemical changes and a corresponding set of clinical manifestations that are diagnostic of this condition.

Clinical Manifestations

Hypertension is the most prominent and almost universal sign of primary aldosteronism. Patients with uncomplicated, complicated, and treatment-resistant hypertension and hypertension with hypokalemia should be considered at risk for this disorder. However, hypokalemia should no longer be considered a requisite for this diagnosis. It is now recognized that hypokalemia occurs in less than half of the patients (Ma & Baranski, 2019). If hypokalemia is present, it may be responsible for the variable muscle weakness or paralysis, cramping, and fatigue in patients with aldosteronism, as well as the kidneys' inability to acidify or concentrate the urine. Accordingly, the urine volume is excessive, leading to polyuria. Serum, by contrast, becomes abnormally concentrated, contributing to polydipsia (excessive thirst) and arterial hypertension. A secondary increase in blood volume and possible direct effects of aldosterone on nerve receptors, such as the carotid sinus, are other factors that result in hypertension.

Hypokalemic alkalosis may decrease the ionized serum calcium level and predispose the patient to tetany and paresthesias. Chvostek and Troussseau signs may be used to assess neuromuscular irritability before overt paresthesia and tetany occur. Glucose intolerance may occur, because hypokalemia interferes with insulin secretion from the pancreas.

Assessment and Diagnostic Findings

Withholding antihypertensive medication before laboratory testing is not required. If the patient is taking an antihypertensive agent, this information should be considered when interpreting the results of the PAC/PRA (plasma aldosterone concentration/plasma renin activity) study as well as the ARR (aldosterone-renin ratio) (Ma & Baranski, 2019).

Medical Management

The recommended treatment of unilateral primary aldosteronism is total surgical removal of the adrenal tumor through laparoscopic adrenalectomy rather than open surgery. Laparoscopic surgery is associated with shorter hospital stays and generally fewer complications following surgery (Ma & Baranski, 2019). During the preoperative period, blood pressure and potassium levels are closely monitored. During the immediate postoperative period, the

patient is susceptible to fluctuations in adrenocortical hormones and requires administration of corticosteroids, fluids, and other agents to maintain blood pressure and prevent acute complications. If the adrenalectomy is bilateral, replacement of corticosteroids will be lifelong. A normal serum glucose level is maintained with insulin, appropriate IV fluids, and dietary modifications. Hypokalemia typically resolves for all patients after surgery and hypertension is resolved in 35% to 60%. Postoperatively, spironolactone and potassium supplements should be discontinued and antihypertensives should be decreased. Potassium levels should be checked once a week for 4 weeks postoperatively (Ma & Baranski, 2019). Medical treatment rather than surgery is recommended for patients with bilateral adrenal involvement due to poor blood pressure control and other risks.

Pharmacologic Therapy

Pharmacologic management is required for patients with bilateral adrenal hyperplasia or unilateral aldosterone hypersecretion who do not undergo surgery. Spironolactone is recommended as the first-line drug to control hypertension. Eplerenone, a more expensive drug, is recommended as a second-line drug if the patient cannot tolerate side effects of spironolactone (Ma & Baranski, 2019). Serum potassium and creatinine should be monitored frequently during the first 4 to 6 weeks of taking spironolactone. Ongoing monitoring will be determined by the clinical course. The half-life of digoxin may be increased when taken with spironolactone, and its dosage may need to be adjusted.

Nursing Management

Nursing management in the postoperative period includes frequent assessment of vital signs to detect early signs and symptoms of adrenal insufficiency and crisis or hemorrhage. Explaining all treatments and procedures, providing comfort measures, and providing rest periods can reduce the patient's stress and anxiety level.

TABLE 45-2

Commonly Used Corticosteroid Preparations

Generic Names	Select Trade Names
beclomethasone	Beconase AQ, Qnasl
betamethasone	Beta-Val, Dermabet, Luxiq, Valnac
dexamethasone	Dexamethasone Intensol
hydrocortisone	Colocort, Cortef, Cortenema, Solu-Cortef
methylprednisolone	Depo-Medrol, Solu-Medrol
prednisone	Prednisone Intensol, Rayos
prednisolone	Prelone
triamcinolone	Kenalog, Triderm

Adapted from Comerford, K. C., & Durkin, M. T. (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer.

Corticosteroid Therapy

Corticosteroids are used extensively for adrenal insufficiency and are also widely used in suppressing inflammation and autoimmune reactions, controlling allergic reactions, and reducing the rejection process in transplantation. Commonly used corticosteroid preparations are listed in Table 45-2. Their anti-inflammatory and antiallergy actions make corticosteroids effective in treating rheumatic or connective tissue diseases, such as rheumatoid arthritis and SLE. They are also frequently used in the treatment of asthma, multiple sclerosis, and other autoimmune disorders.

High doses appear to allow patients to tolerate high degrees of stress. Such antistress action may be caused by the ability of corticosteroids to aid circulating vasopressor substances in keeping the blood pressure elevated; other effects, such as maintenance of the serum glucose level, also may keep blood pressure elevated.

Side Effects

Although the synthetic corticosteroids are safer for some patients because of relative freedom from mineralocorticoid activity, most natural and synthetic corticosteroids produce similar kinds of side effects. The dose required for anti-inflammatory and antiallergy effects also produces metabolic effects, pituitary and adrenal gland suppression, and changes in the function of the central nervous system. Therefore, although corticosteroids are highly effective therapeutically, they may also be very dangerous. Dosages of these medications are frequently altered to allow high concentrations when necessary and then tapered in an attempt to avoid undesirable effects. This

requires that patients be observed closely for side effects and that the dose be reduced when high doses are no longer required. Suppression of the adrenal cortex may persist up to 1 year after a course of corticosteroids.

Therapeutic Uses of Corticosteroids

The dosage of corticosteroids is determined by the nature and chronicity of the illness as well as the patient's other medical conditions. Rheumatoid arthritis, bronchial asthma, and multiple sclerosis are chronic disorders that corticosteroids do not cure; however, these medications may be useful when other measures do not provide adequate control of symptoms. In addition, corticosteroids may be used to treat acute exacerbations of these disorders.

In such situations, the adverse effects of corticosteroids are weighed against the patient's current condition. These medications may be used for a period but then are gradually reduced or tapered as the symptoms subside. The nurse plays an important role in providing encouragement and understanding during times when the patient is experiencing (or is apprehensive about experiencing) recurrence of symptoms while taking smaller doses.

Treatment of Acute Conditions

Acute flare-ups and crises are treated with large doses of corticosteroids. Examples include emergency treatment for bronchial obstruction in status asthmaticus and for septic shock from septicemia caused by gram-negative bacteria. Other measures, such as anti-infective agents or medications, are also used with corticosteroids to treat shock and other major symptoms. At times, corticosteroids are continued past the acute flare-up stage to prevent serious complications.

Ophthalmologic Treatment

Outer eye infections can be treated by topical application of corticosteroid eye drops, because the agents do not cause systemic toxicity. However, long-term application can cause an increase in intraocular pressure, which leads to glaucoma in some patients. In addition, prolonged use of corticosteroids can sometimes lead to cataract formation.

Dermatologic Disorders

Topical administration of corticosteroids in the form of creams, ointments, lotions, and aerosols is especially effective in many dermatologic disorders. It may be more effective in some conditions to use occlusive dressings around the affected part to achieve maximum absorption of the medication. Penetration and absorption are also increased if the medication is applied when the skin is hydrated or moist (e.g., immediately after bathing).

Absorption of topical agents varies with body location. For example, absorption is greater through the layers of skin on the scalp, face, and genital area than on the forearm; as a result, the use of topical agents on these sites increases the risk of side effects. The availability of over-the-counter topical corticosteroids increases the risk of side effects in patients who are unaware of their potential risks. Excessive use of these agents, especially on large surface areas of inflamed skin, can lead to decreased therapeutic effects and increased side effects.

Dosage

Attempts have been made to determine the best time to administer pharmacologic doses of steroids. If symptoms have been controlled on a 6- or 8-hour program, a once-daily or every-other-day schedule may be implemented. In keeping with the natural secretion of cortisol, the best time of day for the total corticosteroid dose is in the early morning, between 7 and 8 am. Large-dose therapy at 8 am, when the adrenal gland is most active, produces maximal suppression of the gland. A large 8 am dose is more physiologic because it allows the body to escape effects of the steroids from 4 pm to 6 am, when serum levels are normally low, hence minimizing cushingoid effects. If symptoms of the disorder being treated are suppressed, alternate-day therapy is helpful in reducing pituitary–adrenal suppression in patients requiring prolonged therapy. Some patients report discomfort associated with symptoms of their primary illness on the second day; therefore, the nurse must explain to patients that this regimen is necessary to minimize side effects and suppression of adrenal function.

Tapering

Corticosteroid dosages are tapered (reduced gradually) to allow normal adrenal function to return and to prevent steroid-induced adrenal insufficiency. Up to 1 year or longer after the use of corticosteroids, the patient is still at risk for adrenal insufficiency in times of stress. For example, if surgery for any reason is necessary, the patient is likely to require IV corticosteroids during and after surgery to reduce the risk of acute adrenal crisis.

Nursing Management

Nursing management of corticosteroid therapy includes many important interventions. [Table 45-3](#) provides an overview of the effects of corticosteroid therapy and the nursing implications.

Promoting Home, Community-Based, and Transitional Care



Educating Patients About Self-Care

The nurse educates the patient, family, and caregivers that acute adrenal insufficiency and underlying symptoms will recur if corticosteroid therapy is stopped abruptly without medical supervision. The patient should be instructed to always have an adequate supply of the corticosteroid medication to avoid running out.

Continuing and Transitional Care

The patient who requires continued corticosteroid therapy is monitored to ensure understanding of the medications and the need for a dosage that treats the underlying disorder while minimizing the side effects. Home care referral may be indicated to ensure a safe environment that minimizes stress, risk of falls, and other side effects. The nurse assesses the patient's physical and psychological status and reports changes to the primary provider. The nurse also assesses the patient's understanding of and ability to manage the medication regimen and reinforces previous education about the medications and the importance of taking them as prescribed. The nurse emphasizes the importance of regular medical follow-up, the side effects of the medications, and the effects of abruptly discontinuing corticosteroids. In addition, the patient and family are reminded about the importance of health promotion activities and recommended health screening, including bone mineral density testing.

TABLE 45-3

Corticosteroid Therapy and Implications for Nursing

Practice

Side Effects	Nursing Interventions
Cardiovascular Effects	
Hypertension	Monitor for elevated blood pressure.
Thrombophlebitis	Assess for signs and symptoms of deep venous thrombosis: redness, warmth, tenderness, and edema of an extremity.
Thromboembolism	Remind patient to avoid positions and situations that restrict blood flow (e.g., crossing legs, prolonged sitting in same position).
Accelerated atherosclerosis	Encourage foot and leg exercises when recumbent. Encourage low-sodium diet. Encourage limited intake of fat.
Immunologic Effects	
Increased risk of infection and masking of signs of infection	Assess for subtle signs of infection and inflammation. Encourage patient to avoid exposure to others with upper respiratory infection. Monitor patient for fungal infections. Encourage good hand hygiene.
Ophthalmologic Changes	
Glaucoma	Encourage yearly eye examinations.
Corneal lesions	Refer patient to ophthalmologist if changes in visual acuity are detected.
Musculoskeletal Effects	
Muscle wasting	Encourage high-protein intake.
Poor wound healing	Encourage high-protein intake and vitamin C supplementation.
Osteoporosis with vertebral compression fractures, pathologic fractures of long bones, aseptic necrosis of head of the femur	Encourage diet high in calcium and vitamin D or calcium and vitamin D supplementation if indicated. Take measures to avoid falls and other trauma. Use caution in moving and turning patient. Encourage postmenopausal women on corticosteroids to consider bone mineral density testing and treatment, if indicated. Instruct patient to rise slowly from bed or chair to avoid falling due to orthostatic hypotension.
Metabolic Effects	
Alterations in glucose metabolism	Monitor blood glucose levels at periodic intervals.

Steroid withdrawal syndrome	<p>Instruct patient about medications, diet, and exercise prescribed to control blood glucose level.</p> <p>Report signs of adrenal insufficiency.</p> <p>Administer corticosteroids and mineralocorticoids as prescribed.</p> <p>Instruct patient about importance of taking corticosteroids as prescribed without abruptly stopping therapy.</p> <p>Encourage patient to obtain and wear a medical identification bracelet.</p> <p>Advise patient to notify all health care providers (e.g., dentist) about the need for corticosteroid therapy.</p>
Changes in Appearance	
Moon face	Encourage low-calorie, low-sodium diet.
Weight gain	Assure patient that most changes in appearance are temporary and will disappear if and when corticosteroid therapy is no longer necessary.
Acne	
Fluid and Electrolyte Imbalances	
	Monitor I&O and electrolytes. Administer fluids and electrolytes as prescribed.

I&O, intake and output.

CRITICAL THINKING EXERCISES

1  ebp A 62-year-old man has been recently diagnosed with hypothyroidismn and requires education to manage his condition. What evidence-based practices will you include when educating this patient? Identify at least three essential pieces of information you would include.

2  pq You are caring for a patient who requires long-term administration of prednisone. Identify three priority nursing diagnoses for this patient related to the use of the prednisone. State the expected outcomes and at least three priority interventions with scientific rationales for each nursing diagnosis.

3  ipc A patient with a 6-month history of Addison's disease is hospitalized for Addisonian crisis and is transferred to a medical unit after a 2-day stay in the intensive care unit. During interprofessional collaborative rounds, the team discusses this patient. What information will the primary provider, the nurse, the physical therapist, and the discharge planner contribute to the discussion? What patient-centered goals will each discipline set for the patient?

4  pq Identify the priorities, approach, and techniques you would use to perform a comprehensive assessment on a 60-year-old patient newly diagnosed with Cushing's syndrome. How will your priorities, approach, and techniques differ if the patient has a visual impairment or is hard of hearing? If the patient is from a culture with very different values from your own?

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*Asterisk indicates nursing research.

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Resources

- American Association of Clinical Endocrinologists (AACE), www.aace.com
- American Cancer Society, www.cancer.org
- American Thyroid Association, www.thyroid.org
- Cushing's Support and Research Foundation (CSRF), www.csrf.net
- Endocrine Society, www.endocrine.org
- Hormone Health Network, www.hormone.org
- National Adrenal Diseases Foundation (NADF), www.nadf.us
- National Cancer Institute, Cancer Net for Health Professionals, www.cancer.gov
- National Institute of Diabetes and Digestive and Kidney Disease, www.niddk.nih.gov

46 Management of Patients with Diabetes

LEARNING OUTCOMES

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

1. Differentiate between the types of diabetes, associated etiologic factors, and pathophysiologic alterations.
2. Identify the diagnostic and clinical significance of blood glucose test results.
3. Describe the relationships among diet and dietary modifications, exercise, and medication (i.e., insulin or oral antidiabetic agents) for people with diabetes.
4. Use the nursing process as a framework for care of the patient who has hyperglycemia with diabetic ketoacidosis or hyperglycemic hyperosmolar syndrome.
5. Describe management strategies for a person with diabetes to use during “sick days.”
6. Outline the major complications of diabetes and the self-care behaviors that are important in their prevention.

NURSING CONCEPTS

- Acid–Base Balance
- Family
- Fluids and Electrolytes
- Infection
- Metabolism
- Patient Education

GLOSSARY

diabetes: a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both

diabetic ketoacidosis (DKA): a metabolic derangement, most commonly occurring in type 1 diabetes, that results from a deficiency of insulin; highly acidic ketone bodies are formed, resulting in acidosis

fasting plasma glucose (FPG): blood glucose determination obtained in the laboratory after fasting for at least 8 hours

gestational diabetes: any degree of glucose intolerance with its onset during pregnancy

glycated hemoglobin: a measure of glucose control that is a result of glucose molecule attaching to hemoglobin for the life of the red blood cell (120 days) (*synonyms:* glycosylated hemoglobin, HgbA_{1C}, or A1C)

glycemic index: the amount a given food increases the blood glucose level compared with an equivalent amount of glucose

hyperglycemia: elevated blood glucose level

hyperglycemic hyperosmolar syndrome (HHS): a metabolic disorder, most commonly of type 2 diabetes resulting from a relative insulin deficiency initiated by an illness that raises the demand for insulin

hypoglycemia: low blood glucose level

impaired fasting glucose (IFG) or impaired glucose tolerance (IGT): a metabolic stage intermediate between normal glucose homeostasis and diabetes; referred to as prediabetes

insulin: a hormone secreted by the beta cells of the islets of Langerhans of the pancreas that is necessary for the metabolism of carbohydrates, proteins, and fats; a deficiency of insulin results in diabetes

ketone: a highly acidic substance formed when the liver breaks down free fatty acids in the absence of insulin

latent autoimmune diabetes of adults (LADA): a subtype of diabetes

medical nutrition therapy (MNT): nutritional therapy prescribed for management of diabetes that usually is given by a registered dietitian

nephropathy: a long-term complication of diabetes in which the kidney cells are damaged; characterized by microalbuminuria in early stages and progressing to end-stage kidney disease

neuropathy: a long-term complication of diabetes resulting from damage to the nerve cell

prediabetes: impaired glucose metabolism in which blood glucose concentrations fall between normal levels and those considered diagnostic for diabetes; includes impaired fasting glucose and impaired glucose tolerance, not clinical entities in their own right but risk factors for future diabetes and cardiovascular disease

retinopathy: a condition that occurs when the small blood vessels that nourish the retina in the eye are damaged

self-monitoring of blood glucose (SMBG): a method of capillary blood glucose testing

type 1 diabetes: a metabolic disorder characterized by an absence of insulin production and secretion from autoimmune destruction of the beta cells of the islets of Langerhans in the pancreas (*formerly:* insulin-dependent diabetes, or juvenile diabetes)

type 2 diabetes: a metabolic disorder characterized by the relative deficiency of insulin production and a decreased insulin action and increased insulin resistance (*formerly:* non-insulin-dependent diabetes, or adult-onset diabetes)

Diabetes is a group of metabolic diseases characterized by **hyperglycemia** (an elevated level of glucose in the blood) resulting from defects in insulin secretion, insulin action, or both (Centers for Disease Control and Prevention [CDC], 2020). Care of the patient with diabetes, formerly known as diabetes mellitus but now more commonly referred to as diabetes, requires an understanding of the epidemiology, pathophysiology, diagnostic testing, medical and nursing care, and rehabilitation of patients with diabetes. The field of diabetes is dynamic with constant advances in technology, research, and medications that can improve the life and well-being of people with diabetes. Nurses care for patients with diabetes in all settings. This chapter focuses on the nursing management of patients with diabetes.

DIABETES

Epidemiology

It is estimated that more than 34.1 million adults in the United States have diabetes, although almost one third of these cases are undiagnosed (CDC, 2020). The number of people older than 20 years newly diagnosed with diabetes increases by 1.7 million per year. If this trend continues, one in every three adults in the United States could have diabetes by 2050. In 2018, the percentage of adults with diabetes increased with age, reaching 28.3% of those age 65 years or older (CDC, 2020).

The rate of prediabetes is also steadily increasing. It is estimated that 35.5% of U.S. adults aged 18 years or older (88 million people) had prediabetes in 2018, based on laboratory findings. Nearly half (48.3%) of adults aged 65 years or older had prediabetes (CDC, 2020). Over \$237 billion a year is spent in medical costs and \$90 billion a year is lost in productivity related to diabetes (CDC, 2020).

Ethnic and racial minority populations are disproportionately affected by diabetes. The age-adjusted prevalence of diabetes is increasing among all gender and racial groups, but compared with Caucasians, African Americans, and members of other racial and ethnic groups (Native Americans and persons of Hispanic origin) are more likely to develop diabetes, are at greater risk for many of the complications, and have higher death rates due to diabetes (CDC, 2020). [Chart 46-1](#) summarizes risk factors for diabetes.

Diabetes can have far-reaching and devastating physical, social, and economic consequences, including the following (CDC, 2020; Virani, Alonso, Benjamin, et al., 2020):

- In the United States, diabetes is the leading cause of nontraumatic amputations and end-stage kidney disease (ESKD).
- Diabetes is the seventh leading cause of death in the United States and the leading cause of new blindness in adults aged 18 to 64 years.
- Emergency department visits and hospitalization rates for adults and children with diabetes are greater than for the general population.

The economic cost of diabetes continues to increase because of increasing health care costs and an aging population.

Classification

The major classifications of diabetes are type 1 diabetes, type 2 diabetes, gestational diabetes, latent autoimmune diabetes of adults (LADA), and diabetes associated with other conditions or syndromes (American Diabetes Association [ADA], 2020). The different types of diabetes vary in cause, clinical course, and treatment (see [Table 46-1](#)). The classification system is dynamic in two ways. First, research findings suggest many differences among individuals within each category. Second, except for people with type 1 diabetes, patients may move from one category to another. For example, a woman with gestational diabetes may, after delivery, move into type 2 diabetes. **Prediabetes** is classified as **impaired glucose tolerance (IGT) or impaired fasting glucose (IFG)** and refers to a condition in which blood glucose concentrations fall between normal levels and those considered diagnostic for diabetes (ADA, 2020; CDC, 2020).

Chart 46-1  RISK FACTORS	
Diabetes	
	<ul style="list-style-type: none">• Age >30 years for type 2 and <30 years for type 1• High-density lipoprotein (HDL) cholesterol level ≤35 mg/dL (0.90 mmol/L) and/or triglyceride level ≥250 mg/dL (2.8 mmol/L)• History of gestational diabetes or delivery of a baby over 9 lb• Hypertension• Family history of diabetes (e.g., parents or siblings with diabetes)• Obesity (i.e., ≥20% over desired body weight or body mass index ≥30 kg/m²)• Previously identified impaired fasting glucose or impaired glucose tolerance• Race/ethnicity (e.g., African Americans, Hispanic Americans, Native Americans, Asian Americans, Pacific Islanders)

Adapted from American Diabetes Association (ADA). (2020). Standards of medical care in diabetes—2020. *Diabetes Care*, 43(Suppl 1), S1–S212.

TABLE 46-1 Common Types of Diabetes and Related Glucose Intolerances

Classification	Clinical Characteristics and Implications
Type 1 (5–10% of all patients with diabetes; formerly juvenile diabetes, or insulin-dependent diabetes)	<p>Onset any age, but usually young (<30 yrs) Usually thin at diagnosis; recent weight loss Etiology includes genetic, immunologic, and environmental factors (e.g., virus) Often have islet cell antibodies Often have antibodies to insulin even before insulin treatment Little or no endogenous insulin Need exogenous insulin to preserve life Ketosis prone when insulin absent Acute complication of hyperglycemia: diabetic ketoacidosis</p>
Type 2 (90–95% of all diabetes: patients with obesity—80% of type 2, patients without obesity—20% of type 2; formerly adult-onset diabetes, or non-insulin-dependent diabetes)	<p>Onset any age, usually ≥30 yrs Usually obesity is present at diagnosis Causes include obesity, heredity, and environmental factors No islet cell antibodies Decrease in endogenous insulin, or increased with insulin resistance Most patients can control blood glucose through weight loss if they have obesity Oral antidiabetic agents may improve blood glucose levels if dietary modification and exercise are unsuccessful May need insulin on a short- or long-term basis to prevent hyperglycemia Ketosis uncommon, except in stress or infection Acute complication: hyperglycemic hyperosmolar syndrome</p>
Diabetes associated with other conditions or syndromes (previously classified as secondary diabetes)	<p>Accompanied by conditions known or suspected to cause the disease: pancreatic diseases, hormonal abnormalities, medications such as corticosteroids and estrogen-containing preparations Depending on the ability of the pancreas to produce insulin, the patient may require treatment with oral antidiabetic agents or insulin.</p>
Gestational diabetes	<p>Onset during pregnancy, usually in the second or third trimester Because of hormones secreted by the placenta, which inhibit the action of insulin Above-normal risk for perinatal complications, especially macrosomia (abnormally large babies) Treated with diet and, if needed, insulin to strictly maintain normal blood glucose levels Occurs in about 18% of pregnancies Glucose intolerance transitory but may recur: <ul style="list-style-type: none"> In subsequent pregnancies 35–60% will develop diabetes (usually type 2) within 10–20 yrs, especially if they have obesity Risk factors include obesity, age >30 yrs, family history of diabetes, previous large babies (>9 lb) Screening tests (glucose challenge test) should be performed on all pregnant women between 24 and 28 wks of gestation Should be screened for diabetes every 3 yrs </p>
Prediabetes (previously classified as abnormality of glucose tolerance)	<p>Previous history of hyperglycemia (e.g., during pregnancy or illness) Current normal glucose metabolism Impaired glucose tolerance or impaired fasting glucose screening after age 40 yrs if there is a family history of diabetes or if symptomatic Encourage ideal body weight, because loss of 10–15 lb may improve glycemic control</p>

Adapted from American Diabetes Association (ADA). (2020). Standards of medical care in diabetes—2020. *Diabetes Care*, 43(Suppl 1), S1–S212; Virani, S. S., Alonso, A., Benjamin, E. J., et al. (2020). Heart disease and stroke statistics—2020 update: A report from the American Heart Association. *Circulation*, 141(9), e139–e596.



COVID-19 Considerations

The 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 data from China suggest that having any type of diabetes could be an important risk factor for becoming infected with SARS-CoV-2 as well as for being hospitalized to manage COVID-19 (Sommerstein, Kochen, Messerli, et al., 2020). Patients with diabetes who are hospitalized with COVID-19 have higher rates of intubation and mortality. Researchers who looked at 486 patients hospitalized with COVID-19 reported those who were older than 60 years of age, male, and had a history of diabetes were at greater risk of requiring intubation (Hur, Price, Gray, et al., 2020). Another study reported that increased mortality in patients with diabetes was associated with older age, having a positive C-reactive protein laboratory finding, and being on insulin (Chen, Yang, Chen, et al., 2020).

Pathophysiology CONCEPTS IN ACTION

Insulin is a hormone secreted by beta cells, which are one of four types of cells in the islets of Langerhans in the pancreas (Norris, 2019). Insulin is an anabolic, or storage, hormone. When a person eats a meal, insulin secretion increases and moves glucose from the blood into muscle, liver, and fat cells. In those cells, insulin has the following actions:

- Transports and metabolizes glucose for energy
- Stimulates storage of glucose in the liver and muscle (in the form of glycogen)
- Signals the liver to stop the release of glucose
- Enhances storage of dietary fat in adipose tissue
- Accelerates transport of amino acids (derived from dietary protein) into cells
- Inhibits the breakdown of stored glucose, protein, and fat

During fasting periods (between meals and overnight), the pancreas continuously releases a small amount of insulin (basal insulin); another pancreatic hormone called *glucagon* (secreted by the alpha cells of the islets of Langerhans) is released when blood glucose levels decrease, which stimulates the liver to release stored glucose. The insulin and the glucagon together maintain a constant level of glucose in the blood by stimulating the release of glucose from the liver.

Initially, the liver produces glucose through glycogenolysis (the breakdown of glycogen). After 8 to 12 hours without food, the liver forms glucose from the breakdown of noncarbohydrate substances, including amino acids, through the process of gluconeogenesis.

Type 1 Diabetes

Type 1 diabetes is characterized by the destruction of the pancreatic beta cells (Norris, 2019). Combined genetic, immunologic, and possibly environmental (e.g., viral) factors are thought to contribute to beta-cell destruction. Although the events that lead to beta-cell destruction are not fully understood, it is generally accepted that a genetic susceptibility is a common underlying factor in the development of type 1 diabetes. People do not inherit type 1 diabetes itself but rather a genetic predisposition, or tendency, toward the development of type 1 diabetes. This genetic tendency has been found in people with certain human leukocyte antigen types. There is also evidence of an autoimmune response in type 1 diabetes. This is an abnormal response in which antibodies are directed against normal tissues of the body, responding to these tissues as if they were foreign. Autoantibodies against islet cells and against endogenous (internal) insulin have been detected in people at the time of diagnosis and even several years before the development of clinical signs of type 1 diabetes. In addition to genetic and immunologic components, environmental factors such as viruses or toxins that may initiate destruction of the beta cell continue to be investigated.

Regardless of the specific cause, the destruction of the beta cells results in decreased insulin production, increased glucose production by the liver, and fasting hyperglycemia. In addition, glucose derived from food cannot be stored in the liver but instead remains in the bloodstream and contributes to postprandial (after meals) hyperglycemia. If the concentration of glucose in the blood exceeds the renal threshold for glucose, usually 180 to 200 mg/dL (9.9 to 11.1 mmol/L), the kidneys may not reabsorb all of the filtered glucose; glycosuria then occurs (i.e., the glucose then appears in the urine). When excess glucose is excreted in the urine, it is accompanied by excessive loss of fluids and electrolytes. This is called *osmotic diuresis*.

Because insulin normally inhibits glycogenolysis and gluconeogenesis, these processes occur in an unrestrained fashion in people with insulin deficiency and contribute further to hyperglycemia. In addition, fat breakdown occurs, resulting in an increased production of **ketone** bodies, a highly acidic substance formed when the liver breaks down free fatty acids in the absence of insulin.

Diabetic ketoacidosis (DKA) is a metabolic derangement that occurs most commonly in persons with type 1 diabetes and results from a deficiency of insulin; highly acidic ketone bodies are formed, and metabolic acidosis occurs. The three major metabolic derangements are hyperglycemia, ketosis, and metabolic acidosis (Norris, 2019). DKA is commonly preceded by a day or more of polyuria, polydipsia, nausea, vomiting, and fatigue with eventual stupor and coma if not treated. The breath has a characteristic fruity odor due to the presence of ketoacids.



Type 2 Diabetes

Type 2 diabetes occurs more commonly among people who are older than 30 years and who have obesity, although its incidence is rapidly increasing in younger people because of the growing epidemic of obesity in children, adolescents, and young adults (CDC, 2020).

The two main problems related to insulin in type 2 diabetes are insulin resistance and impaired insulin secretion. Insulin resistance refers to a decreased tissue sensitivity to insulin. Normally, insulin binds to special receptors on cell surfaces and initiates a series of reactions involved in glucose metabolism. In type 2 diabetes, these intracellular reactions are diminished, making insulin less effective at stimulating glucose uptake by the tissues and at regulating glucose release by the liver (see Fig. 46-1). The exact mechanisms that lead to insulin resistance and impaired insulin secretion in type 2 diabetes are unknown, although genetic factors are thought to play a role.

To overcome insulin resistance and to prevent the buildup of glucose in the blood, increased amounts of insulin must be secreted to maintain the glucose level at a normal or slightly elevated level. If the beta cells cannot keep up with the increased demand for insulin, the glucose level rises and type 2 diabetes develops. Insulin resistance may also lead to metabolic syndrome, which is a constellation of symptoms, including hypertension, hypercholesterolemia, abdominal obesity, and other abnormalities (Norris, 2019).

Despite the impaired insulin secretion that is characteristic of type 2 diabetes, there is enough insulin present to prevent the breakdown of fat and the accompanying production of ketone bodies. Therefore, DKA does not typically occur in type 2 diabetes. However, uncontrolled type 2 diabetes may lead to another acute problem—**hyperglycemic hyperosmolar syndrome (HHS)** (see discussion later in chapter).

Physiology/Pathophysiology

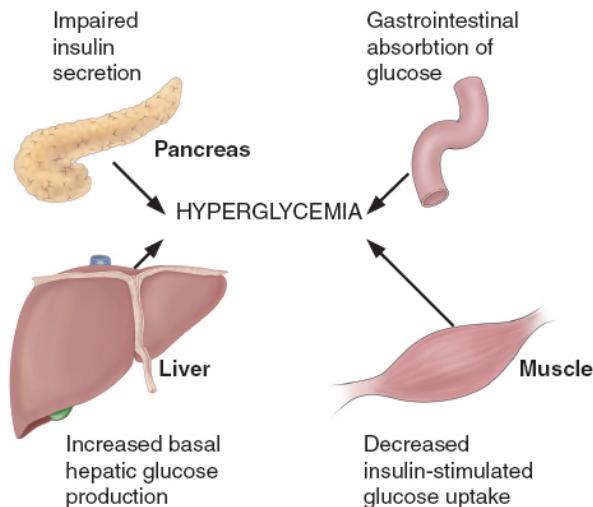


Figure 46-1 • Pathogenesis of type 2 diabetes.

Because type 2 diabetes is associated with a slow, progressive glucose intolerance, its onset may go undetected for many years. If the patient experiences symptoms, they are frequently mild and may include fatigue, irritability, polyuria, polydipsia, poorly healing skin wounds, vaginal infections, or blurred vision (if glucose levels are very high).

For most patients (approximately 75%), type 2 diabetes is detected incidentally (e.g., when routine laboratory tests or ophthalmoscopic examinations are performed). One consequence of undetected diabetes is that long-term diabetes complications (e.g., eye disease, peripheral neuropathy, peripheral vascular disease)

may have developed before the actual diagnosis of diabetes is made (ADA, 2020), signifying that the blood glucose has been elevated for a time before diagnosis.

Gestational Diabetes

Gestational diabetes is any degree of glucose intolerance with its onset during pregnancy (Norris, 2019). Hyperglycemia develops during pregnancy, particularly in the second and third trimesters, because of the secretion of placental hormones that cause insulin resistance.

Women who are considered to be at high risk for gestational diabetes and should be screened by blood glucose testing at their first prenatal visit are those with marked obesity, a personal history of gestational diabetes, glycosuria, or a strong family history of diabetes. High-risk ethnic groups include Hispanic Americans, Native Americans, Asian Americans, African Americans, and Pacific Islanders. If these high-risk women do not have gestational diabetes at initial screening, they should be retested between 24 and 28 weeks of gestation. All women of average risk should be tested at 24 to 28 weeks of gestation. Testing is not specifically recommended for women identified as being at low risk. Low-risk women are those who meet all of the following criteria: age younger than 25 years, normal weight before pregnancy, member of an ethnic group with low prevalence of gestational diabetes, no history of abnormal glucose tolerance, no known history of diabetes in first-degree relatives, and no history of poor obstetric outcomes (ADA, 2020). Women considered to be at high or average risk should have either an oral glucose tolerance test or a glucose challenge test followed by an oral glucose tolerance test in women who exceed the glucose threshold value of 140 mg/dL (7.8 mmol/L) (ADA, 2020).

Initial management includes dietary modification and blood glucose monitoring. Between 70% and 85% of women with gestational diabetes can control blood glucose levels with lifestyle modifications alone. Dietary recommendations include a daily minimum of 175 g of carbohydrates, 71-g protein, 28-g fiber, and low saturated fats (ADA, 2020). If hyperglycemia persists, insulin is prescribed. Target ranges for blood glucose levels during pregnancy are 140 to 180 mg/dL (7.8 to 10 mmol/L) (ADA, 2020).

After delivery, blood glucose levels in women with gestational diabetes usually return to normal. However, many women who have had gestational diabetes develop type 2 diabetes later in life. Women with a history of gestational diabetes should be screened for the development of diabetes or prediabetes every 3 years (ADA, 2020).

Latent Autoimmune Diabetes of Adults (LADA)

In adults, **LADA** is a subtype of diabetes in which the progression of autoimmune beta cell destruction in the pancreas is slower than in types 1 and 2 diabetes. Patients with LADA are at high risk of becoming insulin dependent. Most patients with LADA have at least two of the following: age of onset less than 50 years, body mass index (BMI) less than 25 kg/m², history of autoimmune disease, acute symptoms prior to diagnosis, or positive family history of autoimmune disease (Fischbach & Fischbach, 2018).

Prevention

The Diabetes Prevention Program Research Group (2002) reported that type 2 diabetes can be prevented with appropriate changes in lifestyle. Participants at high risk for type 2 diabetes (BMI greater than 24 kg/m², fasting and postprandial plasma glucose levels elevated but not to levels diagnostic of diabetes) received standard lifestyle recommendations plus metformin, an oral antidiabetic agent; standard lifestyle recommendations plus placebo; or an intensive program of lifestyle modifications. The 16-lesson curriculum of the intensive program of lifestyle modifications focused on weight reduction of greater than 7% of initial body weight and physical activity of moderate intensity. It also included behavior modification strategies designed to help patients achieve the goals of weight reduction and participation in exercise. Compared to the placebo group, the lifestyle intervention group had a 58% lower incidence of diabetes and the metformin group had a 31% lower incidence of diabetes. These findings were found in both genders and all racial and ethnic groups. This research demonstrates that type 2 diabetes can be prevented or delayed in persons at high risk for the disease (Diabetes Prevention Program Research Group, 2002). The Diabetes Prevention Program Outcomes Study followed participants for 15 years and demonstrated that those who enrolled in the program continued to develop type 2 diabetes at a lower rate compared to controls (Diabetes Prevention Program Research Group, 2015).

Researchers have also reported in a study of more than 7000 participants followed for 8 years that those who took glucosamine, a supplement that decreases osteoarthritis and joint pain, had a lower risk of developing type 2 diabetes compared to those not taking the supplement (Ma, Li, Zhou, et al., 2020).

Clinical Manifestations

Clinical manifestations depend on the patient's level of hyperglycemia. Classic clinical manifestations of diabetes include the “three Ps”: polyuria, polydipsia, and polyphagia. Polyuria (increased urination) and polydipsia (increased thirst) occur as a result of the excess loss of fluid associated with osmotic diuresis. Patients also experience polyphagia (increased appetite) that results from the catabolic state induced by insulin deficiency and the breakdown of proteins and fats (Norris, 2019). Other symptoms include fatigue and weakness, sudden vision changes, tingling or numbness in hands or feet, dry skin, skin lesions or wounds that are slow to heal, and recurrent infections. The onset of type 1 diabetes may also be associated with sudden weight loss or nausea, vomiting, or abdominal pains, if DKA has developed.

Chart 46-2

Criteria for the Diagnosis of Diabetes

- Symptoms of diabetes plus casual plasma glucose concentration equal to or greater than 200 mg/dL (11.1 mmol/L). Casual is defined as any time of day without regard to time since last meal. The classic symptoms of diabetes include polyuria, polydipsia, and unexplained weight loss.

Or
- Fasting plasma glucose greater than or equal to 126 mg/dL (7.0 mmol/L). Fasting is defined as no caloric intake for at least 8 hours.

Or
- Two-hour postload glucose equal to or greater than 200 mg/dL (11.1 mmol/L) during an oral glucose tolerance test. The test should use a glucose load containing the equivalent of 75-g anhydrous glucose dissolved in water.

Or
- Hemoglobin A1C $\geq 6.5\%$ (48 mmol/mol).

In the absence of unequivocal hyperglycemia with acute metabolic decompensation, these criteria should be confirmed by repeat testing on a different day. The third measure is not recommended for routine clinical use.

A1C, glycosylated hemoglobin

Adapted from American Diabetes Association (ADA). (2020). Standards of medical care in diabetes—2020. *Diabetes Care*, 43(Suppl 1), S1–S212.

Assessment and Diagnostic Findings

An abnormally high blood glucose level is the basic criterion for the diagnosis of diabetes. **Fasting plasma glucose (FPG)** (blood glucose determination obtained in the laboratory after fasting for at least 8 hours), random plasma glucose, and glucose level 2 hours after receiving glucose (2-hour postprandial load) may be used (Fischbach & Fischbach, 2018). See Chart 46-2 for the ADA’s diagnostic criteria for diabetes (ADA, 2020).

In addition to the assessment and diagnostic evaluation performed to diagnose diabetes, ongoing specialized assessment of patients with known diabetes and evaluation for complications in patients with newly diagnosed diabetes are important components of care. Parameters that should be regularly assessed are discussed in [Chart 46-3](#).



Gerontologic Considerations

Diabetes is particularly prevalent in older adults. In fact, type 2 diabetes is the seventh leading cause of death and affects approximately 20% of older adults (Eliopoulos, 2018). There is a high prevalence among African Americans and those who are 65 to 74 years of age (Eliopoulos, 2018).

Early detection is important but may be challenging because symptoms may be absent or nonspecific. A glucose tolerance test is more effective in diagnosis than urine testing for glucose in older patients due to the higher renal threshold for glucose (Eliopoulos, 2018).

Medical Management

The main goal of diabetes treatment is to normalize insulin activity and blood glucose levels to reduce the development of complications. The Diabetes Control and Complications Trial Research Group (DCCT), a 10-year prospective clinical trial conducted from 1983 to 1993, demonstrated the importance of achieving blood glucose control in the normal, nondiabetic range. This landmark trial demonstrated that intensive glucose control dramatically reduced the development and progression of complications such as **retinopathy** (damage to small blood vessels that nourish the retina), **nephropathy** (damage to kidney cells), and **neuropathy** (damage to nerve cells). Intensive treatment is defined as 3 or 4 insulin injections per day or an insulin pump (i.e., a continuous subcutaneous insulin infusion) plus frequent blood glucose monitoring and weekly contacts with diabetes educators (DCCT, 1993). The ADA recommends that all patients with diabetes strive for glucose control ($\text{HgbA}_{1\text{C}}$ less than 7%) to reduce their risk of complications (ADA, 2020a).

Chart 46-3



ASSESSMENT

Assessing the Patient with Diabetes

History

- Symptoms related to the diagnosis of diabetes:
 - Symptoms of hyperglycemia
 - Symptoms of hypoglycemia
 - Frequency, timing, severity, and resolution
- Results of blood glucose monitoring
- Status, symptoms, and management of chronic complications of diabetes:
 - Eye; kidney; nerve; genitourinary and sexual, bladder, and gastrointestinal
 - Cardiac; peripheral vascular; foot complications associated with diabetes
- Adherence to/ability to follow prescribed dietary management plan
- Adherence to prescribed exercise regimen
- Adherence to/ability to follow prescribed pharmacologic treatment (insulin or oral antidiabetic agents)
- Use of tobacco, alcohol, and prescribed and over-the-counter medications/drugs
- Lifestyle, cultural, psychosocial, and economic factors that may affect diabetes treatment
- Effects of diabetes or its complications on functional status (e.g., mobility, vision)

Physical Examination

- Blood pressure (sitting and standing to detect orthostatic changes)
- Body mass index (height and weight)
- Funduscopic examination and visual acuity
- Foot examination (lesions, signs of infection, pulses)
- Skin examination (lesions and insulin injection sites)
- Neurologic examination
 - Vibratory and sensory examination using monofilament
 - Deep tendon reflexes
- Oral examination

Laboratory Examination

- HgbA_{1C} (A1C)
- Fasting lipid profile
- Test for microalbuminuria
- Serum creatinine level
- Urinalysis
- Electrocardiogram

Need for Referrals

- Ophthalmologist
- Podiatrist
- Dietitian
- Diabetes educator
- Others if indicated

A1C, glycosylated hemoglobin

Adapted from American Diabetes Association (ADA). (2020). Standards of medical care in diabetes—2020. *Diabetes Care*, 43(Suppl 1), S1–S212.

Intensive therapy must be initiated with caution and must be accompanied by thorough education of the patient and family and by responsible behavior of the patient. Careful screening of patients for capability and responsibility is a key step in initiating intensive therapy.

The therapeutic goal for diabetes management is to achieve euglycemia (normal blood glucose levels) without **hypoglycemia** while maintaining a high quality of life. Diabetes management has five components: nutritional therapy, exercise, monitoring, pharmacologic therapy, and education. Diabetes management involves constant assessment and modification of the treatment plan by health professionals and daily adjustments in therapy by the patient. Although the health care team directs the treatment, it is the individual patient who must manage the complex therapeutic regimen. For this reason, patient and family education is an essential component of diabetes treatment and is as important as all other components of the regimen.

Nutritional Therapy

Nutrition, meal planning, weight control, and increased activity are the foundation of diabetes management (ADA, 2020; Evert, Dennison, Gardner, et al., 2019; Franz, MacLeod, Evert, et al., 2017). The most important objectives in the dietary and nutritional management of diabetes are control of total caloric intake to attain or maintain a reasonable body weight, control of blood glucose levels, and normalization of lipids and blood pressure to prevent heart disease. Success in this area alone is often associated with reversal of hyperglycemia in type 2 diabetes. However, achieving these goals is not easy. Because **medical nutrition therapy (MNT)**—nutritional therapy prescribed for management of diabetes usually given by a registered dietitian—is complex, a registered dietitian who understands the therapy has the major responsibility for designing and educating about this aspect of the therapeutic plan. Nurses and all other members of the health care team must be knowledgeable about nutritional therapy and supportive of patients who need to implement nutritional and lifestyle changes. Nutritional management of diabetes includes the following goals:

1. To achieve and maintain:
 - a. Blood glucose levels in the normal range or as close to normal as is safely possible
 - b. A lipid and lipoprotein profile that reduces the risk for vascular disease
 - c. Blood pressure levels in the normal range or as close to normal as is safely possible
2. To prevent, or at least slow, the rate of development of the chronic complications of diabetes by modifying nutrient intake and lifestyle
3. To address individual nutrition needs, taking into account personal and cultural preferences and willingness to change
4. To maintain the pleasure of eating by only limiting food choices when indicated by scientific evidence



For patients who have obesity and diabetes (especially those with type 2 diabetes), weight loss is the key to treatment. (It is also a major factor in preventing diabetes.) In general, overweight is considered to be a BMI of 25 kg/m² to 29 kg/m²; obesity is defined as 20% above ideal body weight or a BMI equal to or greater than 30 kg/m² (ADA, 2020; WHO, 2018). Calculation of BMI is discussed in [Chapter 4](#), and obesity is discussed further in [Chapter 42](#). Patients who have obesity, type 2 diabetes, and require insulin or oral agents to control blood glucose levels may be able to reduce or eliminate the need for medication through weight loss. A weight loss of 5% to 10% of total weight may significantly improve blood glucose levels. For patients who have obesity and diabetes but do not take insulin or an oral antidiabetic medication, consistent meal content or timing is important but not as critical. Rather, decreasing the overall caloric intake is of greater importance. Meals should not be skipped. Pacing food intake throughout the day decreases demands on the pancreas.

The actions of several oral antidiabetic medications include weight loss. For example, the glucagonlike peptide-1 (GLP-1) agonists are associated with delayed gastric emptying and weight loss. The dipeptidyl peptidase-4 (DPP4) and sodium-glucose cotransporter-2 (SGLT2) inhibitors improve glucose control assisting with weight loss (Keresztes & Peacock-Johnson, 2019). See discussion later in the chapter about oral antidiabetic medications.

Consistently following a meal plan is one of the most challenging aspects of diabetes management. It may be more realistic to restrict calories only moderately. For patients who have lost weight incorporating new dietary habits into their lifestyles, diet education, behavioral therapy, group support, and ongoing nutrition counseling are encouraged to maintain weight loss.

Meal Planning and Related Education

The meal plan must consider the patient's food preferences, lifestyle, usual eating times, and ethnic and cultural background. For patients who require insulin to help control blood glucose levels, maintaining as much consistency as possible in the amount of calories and carbohydrates ingested at each meal is essential. In addition, consistency in the approximate time intervals between meals, with the addition of snacks if necessary, helps prevent hypoglycemic reactions and maintain overall blood glucose control. For patients who can master the insulin-to-carbohydrate calculations, lifestyle can be more flexible and diabetes control more predictable. For those using intensive insulin therapy, there may be greater flexibility in the timing and content of meals by allowing adjustments in insulin dosage for changes in eating and exercise habits. Newer insulin analogues, insulin algorithms, and insulin pumps permit greater flexibility of schedules than was previously possible. This contrasts with the concept of maintaining a constant dose of insulin, requiring strict scheduling of meals to match the onset and duration of the insulin.

The first step in preparing a meal plan is a thorough review of the patient's diet history to identify eating habits and lifestyle and cultural eating patterns (ADA, 2020; Evert et al., 2019; Franz et al., 2017). This includes a thorough assessment of the patient's need for weight loss, gain, or maintenance. In most instances, people with type 2 diabetes require weight reduction.

In educating about meal planning, clinical dietitians use various tools, materials, and approaches. Initial education addresses the importance of consistent eating habits, the relationship of food and insulin, and the provision of an individualized meal plan. In-depth follow-up education then focuses on management skills, such as eating at restaurants; reading food labels; and adjusting the meal plan for exercise, illness, and special occasions. The nurse plays an important role in communicating pertinent information to the dietitian and reinforcing the patient's understanding. Communication between the team is important.

Certain aspects of meal planning, such as the food exchange system, may be difficult to learn. This may be related to limitations in the patient's intellectual level or to emotional issues, such as difficulty accepting the diagnosis of diabetes or feelings of deprivation and undue restriction in eating. In any case, it helps to emphasize that using the exchange system (or any food classification system) provides a new way of thinking about food rather than a new way of eating. It is also important to simplify information as much as possible and to provide opportunities for the patient to practice and repeat activities and information.

Caloric Requirements

Calorie-controlled diets are planned by first calculating a person's energy needs and caloric requirements based on age, gender, height, and weight. An activity element is then factored in to provide the actual number of calories required for weight maintenance. To promote a 1- to 2-lb weight loss per week, 500 to 1000 calories are subtracted from the daily total. The calories are distributed into carbohydrates, proteins, and fats, and a meal plan is then developed, taking into account the patient's lifestyle and food preferences.

Patients may be underweight at the onset of type 1 diabetes because of rapid weight loss from severe hyperglycemia. The goal initially may be to provide a higher-calorie diet to regain lost weight and blood glucose control.

Caloric Distribution

A meal plan for diabetes focuses on the percentages of calories that come from carbohydrates, proteins, and fats (Evert et al., 2019; Franz et al., 2017).

Carbohydrates. The caloric distribution currently recommended is higher in carbohydrates than in fat and protein. In general, carbohydrate foods have the greatest effect on blood glucose levels because they are more quickly digested than other foods and are converted into glucose rapidly. However, research into the appropriateness of a higher-carbohydrate diet in patients with decreased glucose tolerance is ongoing, and recommendations may change accordingly. Currently, the ADA and the Academy of Nutrition and Dietetics (formerly the American Dietetic Association) recommend that for all levels of caloric intake, 50% to 60% of calories should be derived from carbohydrates, 20% to 30% from fat, and the remaining 10% to 20% from protein (Evert et al., 2019). The majority of the selections for carbohydrates should come from whole grains. These recommendations are also consistent with those of the American Heart Association and American Cancer Society.

Carbohydrates consist of sugars (e.g., sucrose) and starches (e.g., rice, pasta, bread). Low glycemic index diets (described later) may reduce postprandial glucose levels. Therefore, the nutrition guidelines recommend that all carbohydrates should be eaten in moderation to avoid high postprandial blood glucose levels (Evert et al., 2019; Franz et al., 2017).

Foods high in carbohydrates, such as sucrose (concentrated sweets), are not totally eliminated from the diet but should be eaten in moderation (up to 10% of total calories), because they are typically high in fat and lack vitamins, minerals, and fiber.

Fats. The recommendations regarding fat content of the diabetic diet include both reducing the total percentage of calories from fat sources to less than 30% of total calories and limiting the amount of saturated fats to 10% of total calories. Additional recommendations include limiting the total intake of dietary cholesterol to less than 300 mg/day. This approach may help reduce risk factors such as increased serum cholesterol levels, which are associated with the development of coronary artery disease—the leading cause of death and disability among people with diabetes (ADA, 2020; Evert et al., 2019).

Protein. The meal plan may include the use of some nonanimal sources of protein (e.g., legumes, whole grains) to help reduce saturated fat and cholesterol intake. In addition, the amount of protein intake may be reduced in patients with early signs of kidney disease.

Fiber. Increased fiber in the diet may improve blood glucose levels, decrease the need for exogenous insulin, and lower total cholesterol and low-density lipoprotein levels in the blood (Evert et al., 2019).

There are two types of dietary fibers: soluble and insoluble. Soluble fiber—in foods such as legumes, oats, and some fruits—plays more of a role in lowering blood glucose and lipid levels than does insoluble

fiber, although the clinical significance of this effect is probably small (Evert et al., 2019). Soluble fiber slows stomach emptying and the movement of food through the upper digestive tract. The potential glucose-lowering effect of fiber may be caused by the slower rate of glucose absorption from foods that contain soluble fiber. Insoluble fiber is found in whole-grain breads and cereals and in some vegetables. This type of fiber along with soluble fiber increases satiety, which is helpful for weight loss. At least 28 g of fiber should be ingested daily (ADA, 2020).

One risk involved in suddenly increasing fiber intake is that it may require adjusting the dosage of insulin or oral agents to prevent hypoglycemia. Other problems may include abdominal fullness, nausea, diarrhea, increased flatulence, and constipation if fluid intake is inadequate. If fiber is added to or increased in the meal plan, it should be done gradually and in consultation with a dietitian. Exchange lists (ADA, 2020) serve as an excellent guide for increasing fiber intake. Fiber-rich food choices within the vegetable, fruit, and starch/bread exchanges are highlighted in the lists.

Food Classification Systems

To educate about diet principles and help in meal planning, several systems have been developed in which foods are organized into groups with common characteristics, such as number of calories, composition of foods (i.e., amount of protein, fat, carbohydrate in the food), or effect on blood glucose levels. Several of these are listed next.

Exchanges	Select Sample Menus from Exchange Lists		
	Sample Lunch #1	Sample Lunch #2	Sample Lunch #3
2 starch	2 slices bread	Hamburger bun	1 cup cooked pasta
3 meat	2-oz sliced turkey and 1-oz low-fat cheese	3-oz lean beef patty	3-oz boiled shrimp
1 vegetable	Lettuce, tomato, onion	Green salad	½ cup plum tomatoes
1 fat	1-tsp mayonnaise	1-tbsp salad dressing	1-tsp olive oil
1 fruit	1 medium apple	1¼ cup watermelon	1¼ cup fresh strawberries
“Free” items (optional)	Unsweetened iced tea Mustard, pickle, hot pepper	Diet soda 1 tbsp catsup, pickle, onions	Ice water with lemon Garlic, basil

Exchange Lists. A commonly used tool for nutritional management is the exchange lists for meal planning (ADA, 2020). There are six main exchange lists: bread/starch, vegetable, milk, meat, fruit, and fat. Foods within one group (in the portion amounts specified) contain equal numbers of calories and are approximately equal in grams of protein, fat, and carbohydrate. Meal plans can be based on a recommended number of choices from each exchange list. Foods on one list may be interchanged with one another, allowing for variety while maintaining as much consistency as possible in the nutrient content of foods eaten. Table 46-2 presents three sample lunch menus that are interchangeable in terms of carbohydrate, protein, and fat content.

Exchange list information on combination foods such as pizza, chili, and casseroles, as well as convenience foods, desserts, snack foods, and fast foods, is available from the ADA (see the Resources section). Some food manufacturers and restaurants publish exchange lists that describe their products.

Nutrition Labels. Food manufacturers are required to have the nutrition content of foods listed on their packaging, and reading food labels is an important skill for patients to learn and use when food shopping. The label includes information about how many grams of carbohydrate are in a serving of food. This information can be used to determine how much medication is needed. For example, a patient who takes premeal insulin may use the algorithm of 1 unit of insulin for 15 g of carbohydrate. Patients can also be educated to have a “carbohydrate budget” per meal (e.g., 45 to 60 g).

Carbohydrate counting is a nutritional tool used for blood glucose management because carbohydrates are the main nutrients in food that influence blood glucose levels. This method provides flexibility in food choices, can be less complicated to understand than the diabetic food exchange list, and allows more accurate management with multiple daily injections (insulin before each meal). However, if carbohydrate counting is not used with other meal-planning techniques, weight gain can result. A variety of methods are used to count carbohydrates. When developing a diabetic meal plan using carbohydrate counting, all food sources should be considered.

Once digested, 100% of carbohydrates are converted to glucose. Approximately 50% of protein foods (meat, fish, and poultry) are also converted to glucose, and this has minimal effect on blood glucose levels.

While carbohydrate counting is commonly used for blood glucose management with type 1 and type 2 diabetes, it is not a perfect system. All carbohydrates affect the blood glucose level to different degrees, regardless of equivalent serving size (i.e., the glycemic index—see later discussion). When carbohydrate

counting is used, reading labels on food items is the key to success. Knowing what the “carbohydrate budget” for the meal is and knowing how many grams of carbohydrate are in a serving of a food, the patient can calculate the amount in one serving.

Healthy Food Choices. An alternative to counting grams of carbohydrate is measuring servings or choices. This method is used more often by people with type 2 diabetes. It is similar to the food exchange list and emphasizes portion control of total servings of carbohydrate at meals and snacks. One carbohydrate serving is equivalent to 15 g of carbohydrate. Examples of one serving are an apple 2 inches in diameter and one slice of bread. Vegetables and meat are counted as one third of a carbohydrate serving. This system works well for those who have difficulty with more complicated systems.

MyPlate Food Guide. The Food Guide (i.e., MyPlate) is another tool used to develop meal plans. It is commonly used for patients with type 2 diabetes who have a difficult time following a calorie-controlled diet. Foods are categorized into five major groups (grains, vegetables, fruits, dairy, and protein), plus fats and oils (see Chapter 4, Fig. 4-5). Foods (grains, fruits, and vegetables) that are lowest in calories and fat and highest in fiber should make up the basis of the diet. For those with diabetes, as well as for the general population, 50% to 60% of the daily caloric intake should be from these three groups. Foods higher in fat (particularly saturated fat) should account for a smaller percentage of the daily caloric intake. Fats, oils, and sweets should be used sparingly to obtain weight and blood glucose control and to reduce the risk for cardiovascular disease. Reliance on MyPlate may result in fluctuations in blood glucose levels, however, because high-carbohydrate foods may be grouped with low-carbohydrate foods. The guide is appropriately used only as a first-step educational tool for patients who are learning how to control food portions and how to identify which foods contain carbohydrate, protein, and fat.

Glycemic Index. One of the main goals of diet therapy in diabetes is to avoid sharp, rapid increases in blood glucose levels after food is eaten. The term **glycemic index** is used to describe how much a given food increases the blood glucose level compared with an equivalent amount of glucose. The effects of the use of the glycemic index on blood glucose levels and on long-term patient outcomes are unclear, but it may be beneficial (ADA, 2020; Evert et al., 2019). Although more research is necessary, the following guidelines may be helpful when making dietary recommendations:

- Combining starchy foods with protein- and fat-containing foods tends to slow their absorption and lower the glycemic index.
- In general, eating foods that are raw and whole results in a lower glycemic index than eating chopped, puréed, or cooked foods (except meat).
- Eating whole fruit instead of drinking juice decreases the glycemic index, because fiber in the fruit slows absorption.
- Adding foods with sugars to the diet may result in a lower glycemic index if these foods are eaten with foods that are more slowly absorbed.

Patients can create their own glycemic index by monitoring their blood glucose level after ingestion of a particular food. This can help improve blood glucose control through individualized manipulation of the diet. Many patients who use frequent monitoring of blood glucose levels can use this information to adjust their insulin doses in accordance with variations in food intake.

Other Dietary Concerns

Alcohol Consumption

Patients with diabetes do not need to give up alcoholic beverages entirely, but patients and primary providers must be aware of the potential adverse effects of alcohol specific to diabetes. Alcohol is absorbed before other nutrients and does not require insulin for absorption. Large amounts can be converted to fats, increasing the risk for DKA. In general, the same precautions regarding the use of alcohol by people without diabetes should be applied to patients with diabetes. Moderation is recommended. A major danger of alcohol consumption by the patient with diabetes is hypoglycemia, especially for patients who take insulin or insulin secretagogues (medications that increase the secretion of insulin by the pancreas). Alcohol may decrease the normal physiologic reactions in the body that produce glucose (gluconeogenesis). Therefore, if a patient with diabetes consumes alcohol on an empty stomach, there is an increased likelihood of hypoglycemia. In addition, excessive alcohol intake may impair the patient’s ability to recognize and treat hypoglycemia or to follow a prescribed meal plan to prevent hypoglycemia. To reduce the risk of hypoglycemia, the patient should be cautioned to consume food along with the alcohol; however, carbohydrate consumed with alcohol may raise blood glucose.

Alcohol consumption may lead to excessive weight gain (from the high caloric content of alcohol), hyperlipidemia, and elevated glucose levels (especially with mixed drinks and liqueurs). Patient education regarding alcohol intake must emphasize moderation in the amount of alcohol consumed. Moderate intake is

considered to be one alcoholic beverage per day for women and two per day for men. Lower-calorie or less-sweet drinks (e.g., light beer, wine) and food intake along with alcohol consumption are advised (ADA, 2020; Evert et al., 2019). Patients with type 2 diabetes who wish to control their weight should incorporate the calories from alcohol into the overall meal plan.

Sweeteners

The use of artificial sweeteners is acceptable, especially if it assists in overall dietary adherence. Moderation in the amount of sweetener used is encouraged to avoid potential adverse effects. There are two main types of sweeteners: nutritive and nonnutritive. The nutritive sweeteners contain calories, and the nonnutritive sweeteners have few or no calories in the amounts normally used.

Nutritive sweeteners include fructose (fruit sugar), sorbitol, and xylitol, all of which provide calories in amounts similar to those in sucrose (table sugar). They cause less elevation in blood sugar levels than sucrose does and are often used in sugar-free foods. Sweeteners containing sorbitol may have a laxative effect.

Nonnutritive sweeteners have minimal or no calories. They are used in food products and are also available for table use. They produce minimal or no elevation in blood glucose levels, and the U.S. Food and Drug Administration (FDA) lists them as safe for people with diabetes.

Misleading Food Labels

Foods labeled “sugarless” or “sugar-free” may still provide calories equal to those of the equivalent sugar-containing products if they are made with nutritive sweeteners. Therefore, these foods should not be considered “free” foods to be eaten in unlimited quantity, because they can elevate blood glucose levels. Foods labeled “dietetic” are not necessarily reduced-calorie foods. Patients are advised that foods labeled dietetic may still contain significant amounts of sugar or fat.

Patients must read the labels of “health foods”—especially snacks—because they often contain carbohydrates (e.g., honey, brown sugar, corn syrup, flour) and saturated vegetable fats (e.g., coconut or palm oil), hydrogenated vegetable fats, or animal fats, which may be contraindicated in people with elevated blood lipid levels.

Exercise

Exercise is extremely important in diabetes management because of its effects on lowering blood glucose and reducing cardiovascular risk factors (ADA, 2020). Exercise lowers blood glucose levels by increasing the uptake of glucose by body muscles and by improving insulin utilization. It also improves circulation and muscle tone. Resistance (strength) training, such as weight lifting, can increase lean muscle mass, thereby increasing the resting metabolic rate. These effects are useful in diabetes in relation to losing weight, easing stress, and maintaining a feeling of well-being. Exercise also alters blood lipid concentrations, increasing levels of high-density lipoproteins and decreasing total cholesterol and triglyceride levels. This is especially important for people with diabetes because of their increased risk of cardiovascular disease.

Exercise Recommendations

Ideally, a person with diabetes should engage in regular exercise. General considerations for exercise in patients with diabetes are presented in [Chart 46-4](#). Exercise recommendations must be altered as necessary for patients with diabetic complications, such as retinopathy, autonomic neuropathy, sensorimotor neuropathy, and cardiovascular disease (ADA, 2020). Increased blood pressure associated with exercise may aggravate diabetic retinopathy and increase the risk of a hemorrhage into the vitreous or retina.

Chart 46-4



PATIENT EDUCATION

General Considerations for Exercise in People with Diabetes

The nurse instructs the patient to:

- Exercise three times each week with no more than 2 consecutive days without exercise.
- Perform resistance training twice a week if you have type 2 diabetes.
- Exercise at the same time of day (preferably when blood glucose levels are at their peak) and for the same duration each session.
- Use proper footwear and, if appropriate, other protective equipment (i.e., helmets for cycling).
- Avoid trauma to the lower extremities, especially if you have numbness due to peripheral neuropathy.
- Inspect feet daily after exercise.
- Avoid exercise in extreme heat or cold.
- Avoid exercise during periods of poor metabolic control.
- Stretch for 10 to 15 minutes before exercising.

Adapted from American Diabetes Association (ADA). (2020). Standards of medical care in diabetes—2020. *Diabetes Care*, 43(Suppl 1), S1–S212.

In general, a slow, gradual increase in the exercise period is encouraged. For many patients, walking is a safe and beneficial form of exercise that requires no special equipment (except for proper shoes) and can be performed anywhere. People with diabetes should discuss an exercise program with their primary provider and undergo a careful medical evaluation with appropriate diagnostic studies before beginning a program (ADA, 2020).

For patients who are older than 30 years and who have two or more risk factors for heart disease, an exercise stress test is recommended prior to starting an exercise program (ADA, 2020). Risk factors for heart disease include hypertension, obesity, high cholesterol levels, abnormal resting electrocardiogram (ECG), sedentary lifestyle, smoking, male gender, and a family history of heart disease. An abnormal stress test may indicate cardiac ischemia. Typically, an abnormal stress test is followed by a cardiac catheterization and, in some cases, with an intervention such as angioplasty, stent placement, or cardiac surgery.

Exercise Precautions

Patients who have blood glucose levels exceeding 250 mg/dL (14 mmol/L) and who have ketones in their urine should not begin exercising until the urine test results are negative for ketones and the blood glucose level is closer to normal. Exercising with elevated blood glucose levels increases the secretion of glucagon, growth hormone, and catecholamines. The liver then releases more glucose, and the result is an increase in the blood glucose level (ADA, 2020).

The physiologic decrease in circulating insulin that normally occurs with exercise cannot occur in patients treated with insulin. Initially, patients who require insulin should be taught to eat a 15-g carbohydrate snack (a fruit exchange) or a snack of complex carbohydrates with a protein before engaging in moderate exercise to prevent unexpected hypoglycemia. The exact amount of food needed varies from person to person and should be determined by blood glucose monitoring.

Another concern for patients who take insulin is hypoglycemia that occurs many hours after exercise. To avoid post-exercise hypoglycemia, especially after strenuous or prolonged exercise, the patient may need to eat a snack at the end of the exercise session and at bedtime and monitor the blood glucose level more frequently. Patients who are capable, knowledgeable, and responsible can learn to adjust their own insulin doses by working closely with a diabetes educator. Others need specific instructions on what to do when they exercise.

Patients taking insulin and participating in extended periods of exercise should test their blood glucose levels before, during, and after the exercise period, and they should snack on carbohydrates as needed to maintain blood glucose levels. Other participants or observers should be aware that the person exercising has diabetes, and they should know what assistance to give if severe hypoglycemia occurs.

In people with type 2 diabetes who are overweight or have obesity, exercise in addition to dietary management both improves glucose metabolism and enhances loss of body fat. Exercise coupled with weight loss improves insulin sensitivity and may decrease the need for insulin or oral antidiabetic agents (ADA, 2020). Eventually, the patient's glucose tolerance may return to normal. Patients with type 2 diabetes who are not taking insulin or an oral agent may not need extra food before exercise.



Gerontologic Considerations

Physical activity that is consistent and realistic is beneficial to older adults with diabetes. Physical fitness in the older adult population with diabetes may lead to improved glycemic control, decreased risk for chronic vascular disease, and an improved quality of life (Eliopoulos, 2018). Advantages of exercise in this population include a decrease in hyperglycemia, a general sense of well-being, and better use of ingested calories, resulting in weight reduction. Because there is an increased incidence of cardiovascular problems in older adults, a physical examination and exercise stress test may be warranted before an exercise program is initiated. A pattern of gradual, consistent exercise, including a combination of stretching, aerobic exercise, and resistance training, should be planned that does not exceed the patient's physical capacity. Physical impairment because of other chronic diseases must also be considered. In some cases, a physical therapy evaluation may be indicated, with the goal of determining exercises specific to the patient's needs and abilities. Tools such as the Go4Life senior fitness booklet may be helpful (see Resources section at the end of chapter).

Monitoring Glucose Levels and Ketones

Blood glucose monitoring is a cornerstone of diabetes management, and **self-monitoring of blood glucose (SMBG)** levels have dramatically altered diabetes care. SMBG is a method of capillary blood glucose testing in which the patient pricks their finger and applies a drop of blood to a test strip that is read by a meter. It is recommended that SMBG occurs when circumstances call for it (e.g., before meals, snacks, exercise) for many patients taking insulin (ADA, 2020).

Self-Monitoring of Blood Glucose

Using SMBG and learning how to respond to the results enable people with diabetes to individualize their treatment regimen to obtain optimal blood glucose control. This allows for detection and prevention of hypoglycemia and hyperglycemia and plays a crucial role in normalizing blood glucose levels, which in turn may reduce the risk of long-term diabetic complications.

Various methods for SMBG are available. Most involve obtaining a drop of blood from the fingertip, applying the blood to a special reagent strip, and allowing the blood to stay on the strip for the amount of time specified by the manufacturer (usually 5 to 30 seconds). The meter gives a digital readout of the blood glucose value. The meters available for SMBG offer various features and benefits such as monthly averages, tracking of events such as exercise and food consumption, and downloading capacity. Most meters are biosensors that can use blood obtained from alternative test sites, such as the forearm. There is a special lancing device that is useful for patients who have painful fingertips or experience pain with fingersticks.

Because laboratory methods measure plasma glucose, most blood glucose monitors approved for patient use in the home and some test strips calibrate blood glucose readings to plasma values. Plasma glucose values are 10% to 15% higher than whole blood glucose values, and it is crucial for patients with diabetes to know whether their monitor and strips provide whole blood or plasma results.

Methods for SMBG must match the skill level and physical capabilities of patients. Factors affecting SMBG performance include visual acuity, fine motor coordination, cognitive ability, comfort with technology and willingness to use it, and cost (Eliopoulos, 2018). Some meters can be used by patients with visual impairments; these meters have audio components to assist in performing the test and obtaining the result. In addition, meters are available to check both blood glucose and blood ketone levels by those who are particularly susceptible to DKA. Most insurance companies, and programs such as Medicare and Medicaid, cover some or all of the costs of meters and strips.

All methods of SMBG carry the risk that patients may obtain and report erroneous blood glucose values as a result of incorrect techniques. Some common sources of error include improper application of blood (e.g., drop too small), damage to the reagent strips caused by heat or humidity, the use of outdated strips, and improper meter cleaning and maintenance.

Nurses play an important role in providing initial education about SMBG techniques. Equally important is evaluating the techniques of patients who are experienced in self-monitoring. Every 6 to 12 months, patients should conduct a comparison of their meter result with a simultaneous laboratory-measured blood glucose level in their provider's office and have their technique observed. The accuracy of the meter and strips can also be assessed with control solutions specific to that meter whenever a new vial of strips is used and whenever the validity of the reading is in doubt.

Candidates for Self-Monitoring of Blood Glucose

SMBG is a useful tool for managing self-care for everyone with diabetes. It is a key component of treatment for any intensive insulin therapy regimen (i.e., 2 to 4 injections per day or the use of an insulin pump) and for diabetes management during pregnancy. It is also recommended for patients with the following conditions:

- Unstable diabetes (severe swings from very high to very low blood glucose levels within a 24-hour day)
- A tendency to develop severe ketosis or hypoglycemia
- Hypoglycemia without warning symptoms

For patients not taking insulin, SMBG is helpful for monitoring the effectiveness of exercise, diet, and oral antidiabetic agents. For patients with type 2 diabetes, SMBG is recommended during periods of suspected hyperglycemia (e.g., illness) or hypoglycemia (e.g., unusual increased activity levels) and when the medication or dosage of medication is modified (ADA, 2020).

Frequency of Self-Monitoring of Blood Glucose

For most patients who require insulin, SMBG is recommended two to four times daily (usually before meals and at bedtime). For patients who take insulin before each meal, SMBG is required at least three times daily before meals to determine each dose (ADA, 2020). Those not receiving insulin may be instructed to assess their blood glucose levels at least two or three times per week, including a 2-hour postprandial test. For all patients, testing is recommended whenever hypoglycemia or hyperglycemia is suspected; with changes in medications, activity, or diet; and with stress or illness.

Responding to Self-Monitoring of Blood Glucose Results

Patients are asked to keep a record or logbook of blood glucose levels so that they can detect patterns. Testing is done at the peak action time of the medication to evaluate the need for dosage adjustments. To evaluate basal insulin and determine bolus insulin doses, testing is performed before meals. To determine the need for bolus doses of regular or rapid-acting insulin (lispro, aspart, or glulisine), testing is done 2 hours after meals. Patients with type 2 diabetes are encouraged to test daily before and 2 hours after the largest meal of the day until individualized blood glucose levels are reached. Thereafter, testing should be done periodically before and after meals. Patients who take insulin at bedtime or who use an insulin infusion pump should also test at 3 am once a week to document that the blood glucose level is not decreasing during the night. If the patient is unwilling or cannot afford to test frequently, then once or twice a day may be sufficient if the time of testing is varied (e.g., before breakfast one day, before lunch the next day).

Patients are more likely to discontinue SMBG if they are not instructed how to use the results to alter the treatment regimen, if they receive no positive reinforcement, and if testing costs increase. At the very least, the patient should be given parameters for contacting the primary provider. Patients using intensive insulin therapy regimens may be instructed in the use of algorithms (rules or decision trees) for changing the insulin doses based on patterns of values greater or less than the target range and the amount of carbohydrate to be consumed. Baseline patterns should be established by SMBG for 1 to 2 weeks.

Using a Continuous Glucose Monitoring System

A continuous glucose monitoring (CGM) system is an advanced way that people living with diabetes can use to monitor blood glucose levels. A CGM can be used with or without an insulin pump (see Fig. 46-2A). A sensor attached to a transmitter is inserted subcutaneously in the abdomen or back of the arm and connected to a wireless monitoring device, where the glucose levels are displayed in real time (see Fig. 46-2B). Sensors are replaced every 7 to 14 days. The data from the CGM device are downloaded, and blood glucose readings are analyzed. The newest CGM is implantable and can be worn for 90 days (Kropff, Choudhary, Neupane, et al., 2017). Although the CGM cannot be used for making decisions about specific insulin doses, it can be used to determine whether treatment is adequate over a 24-hour period. This device is most useful in patients with type 1 diabetes (ADA, 2020).

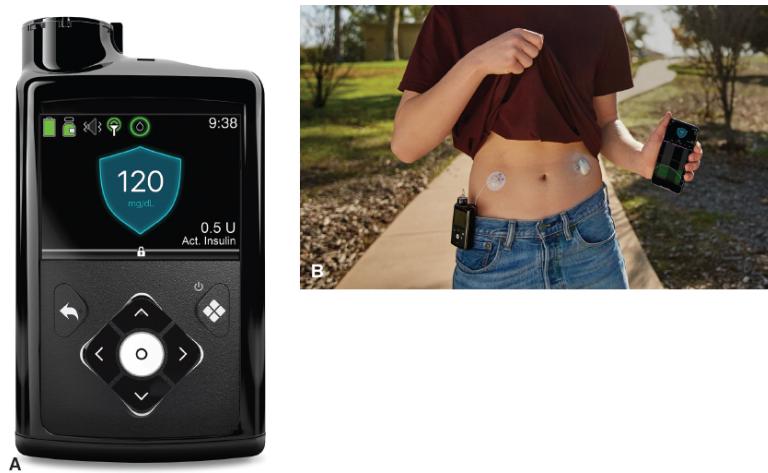


Figure 46-2 • A. The MiniMed™ 770G Hybrid Closed Loop System offers SmartGuard™, a feature using continuous glucose monitoring to automatically adjust insulin delivery. **B.** Patient using this insulin pump for self-management of blood glucose and insulin doses. Parts A and B: Manufactured by the diabetes division of Medtronic, Inc. Used with permission.

Testing for Glycated Hemoglobin

Glycated hemoglobin (also referred to as glycosylated hemoglobin, HgbA_{1C}, or A1C) is a measure of glucose control for the past 3 months (ADA, 2020). When blood glucose levels are elevated, glucose molecules attach to hemoglobin in red blood cells. The longer the amount of glucose in the blood remains above normal, the more glucose binds to hemoglobin and the higher the glycated hemoglobin level becomes. This complex (hemoglobin attached to the glucose) is permanent and lasts for the life of an individual red blood cell, approximately 120 days. If near-normal blood glucose levels are maintained, with only occasional increases, the overall value will not be greatly elevated. However, if the blood glucose values are consistently high, then the test result is also elevated. If the patient reports mostly normal SMBG results but the glycated hemoglobin is high, there may be errors in the methods used for glucose monitoring, errors in recording results, or frequent elevations in glucose levels at times during the day when the patient is not usually monitoring blood sugar levels. Normal values typically range from 4% to 6% and indicate consistently near-normal blood glucose concentrations. The target range for people with diabetes is less than 7% (53 mmol/mol) (ADA, 2020).

Testing for Ketones

Ketones (or ketone bodies) are by-products of fat breakdown, and they accumulate in the blood and urine. Ketones in the urine signal that there is a deficiency of insulin and control of type 1 diabetes is deteriorating. When there is almost no effective insulin available, the body starts to break down stored fat for energy.

The patient may use a urine dipstick (Ketostix or Chemstrip uK) to detect ketonuria. The reagent pad on the strip turns purple when ketones are present. (One of the ketone bodies is called *acetone*, and this term is frequently used interchangeably with the term *ketones*.) Other strips are available for measuring both urine glucose and ketones (Keto-Diastix or Chemstrip uGK). Large amounts of ketones may depress the color response of the glucose test area, meters that test the blood for ketones are available.

Urine ketone testing should be performed whenever patients with type 1 diabetes have glycosuria or persistently elevated blood glucose levels (more than 240 mg/dL or 13.2 mmol/L for two testing periods in a row) and during illness, in pregnancy with preexisting diabetes, and in gestational diabetes (ADA, 2020).

Pharmacologic Therapy

Insulin is secreted by the beta cells of the islets of Langerhans and lowers the blood glucose level after meals by facilitating the uptake and utilization of glucose by muscle, fat, and liver cells. In the absence of adequate insulin, pharmacologic therapy is essential.

Insulin Therapy

In type 1 diabetes, exogenous insulin must be given for life because the body loses the ability to produce insulin. In type 2 diabetes, insulin may be necessary on a long-term basis to control glucose levels if meal planning and oral agents are ineffective or when insulin deficiency occurs. In addition, some patients in

whom type 2 diabetes is usually controlled by meal planning alone or by meal planning and an oral antidiabetic agent may require insulin temporarily during illness, infection, pregnancy, surgery, or some other stressful event. In many cases, insulin injections are given two or more times daily to control the blood glucose level. Because the insulin dose required by the individual patient is determined by the level of glucose in the blood, accurate monitoring of blood glucose levels is essential; thus, SMBG is a cornerstone of insulin therapy.

TABLE 46-3 Select Categories of Insulin

Time Course	Agent	Onset	Peak	Duration	Indications
Rapid acting	lispro aspart glulisine	15–30 min 15 min 5–15 min	30–90 min 1–3 h 1 h	≤5 h 3–4 h 5 h	Used for rapid reduction of glucose level, to treat postprandial hyperglycemia, or to prevent nocturnal hypoglycemia
Short acting	regular	30–60 min	2–3 h	4–6 h	Usually given 15 min before a meal; may be taken alone or in combination with longer-acting insulin
Intermediate acting	NPH (neutral protamine Hagedorn)	1–1.5 h	4–12 h	Up to 24 h	Food should be taken around the time of onset and peak
Long acting	glargine detemir	3–6 h unknown	Continuous (no peak)	24 h 24 h	Used for basal dose
Rapid-acting inhalation powder	Afrezza	<15 min	~50 min	2–3 h	Administer at the beginning of a meal

Adapted from Comerford, K. C., & Durkin, M. T. (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer; Keresites, P., & Peacock-Johnson, A. (2019). Type 2 diabetes: A pharmacological update. *American Journal of Nursing*, 119(2), 32–40.

Preparations

A number of insulin preparations are available. They vary according to three main characteristics: time course of action, species (source), and manufacturer (Comerford & Durkin, 2020). Human insulins are produced by recombinant deoxyribonucleic acid (DNA) technology and are the only type of insulin available in the United States.

Time Course of Action. Insulins may be grouped into several categories based on the onset, peak, and duration of action (see Table 46-3).

Rapid-acting insulins produce a more rapid effect that is of shorter duration than regular insulin. Because of their rapid onset, the patient should be instructed to eat no more than 5 to 15 minutes after injection. Because of the short duration of action of these insulin analogues, patients with type 1 diabetes and some patients with type 2 or gestational diabetes also require a long-acting insulin (basal insulin) to maintain glucose control. Basal insulin is necessary to maintain blood glucose levels irrespective of meals. A constant level of insulin is required at all times. Intermediate-acting insulins function as basal insulins but may have to be split into 2 injections to achieve 24-hour coverage.

Short-acting insulins are called *regular insulin* (marked R on the bottle). Regular insulin is a clear solution and is usually given 15 minutes before a meal, either alone or in combination with a longer-acting insulin. Regular insulin can be administered IV (Comerford & Durkin, 2020).

Intermediate-acting insulins are called *NPH insulin* (neutral protamine Hagedorn), which are similar in their time course of action, appear uniformly milky and cloudy. If an NPH insulin is taken alone, it is not crucial that it be taken before a meal but patients should eat some food around the time of the onset and peak of these insulins.

“Peakless” basal or long-acting insulins are used as a basal insulin—that is, the insulin is absorbed very slowly over 24 hours and can be given once a day (Comerford & Durkin, 2020). Because the insulin is in a suspension with a pH of 4, it cannot be mixed with other insulins because this would cause precipitation. It is administered once a day at any time of the day but must be given at the same time each day to prevent overlap of action. Many patients fall asleep, forgetting to take their bedtime insulin, or may be wary of taking insulin before going to sleep. Having these patients take their insulin in the morning ensures that the dose is taken.



Quality and Safety Nursing Alert

When administering insulin, it is very important to read the label carefully and to be sure that the correct type and dose of insulin is given.

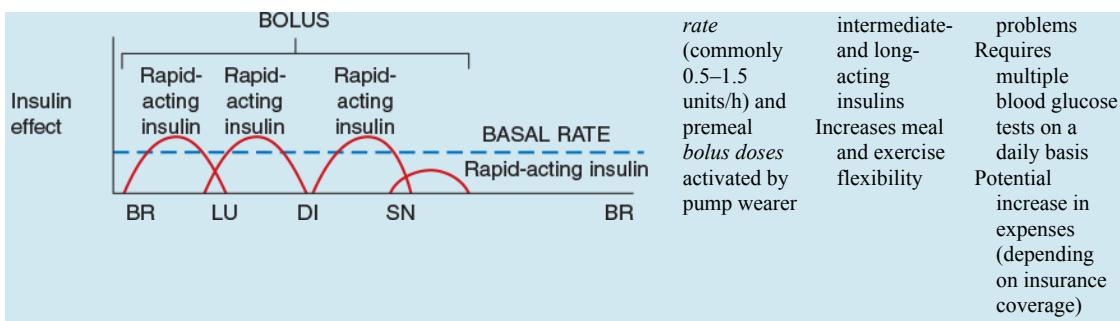
The nurse should emphasize which meals—and snacks—are being “covered” by which insulin doses. In general, the rapid- and short-acting insulins are expected to cover the increase in glucose levels after meals, immediately after the injection; the intermediate-acting insulins are expected to cover subsequent meals; and the long-acting insulins provide a relatively constant level of insulin and act as a basal insulin.

Insulin Regimens

Insulin regimens vary from 1 to 4 injections per day. Usually, there is a combination of a short-acting insulin and a longer-acting insulin. The normally functioning pancreas continuously secretes small amounts of insulin during the day and night. In addition, whenever blood glucose increases after ingestion of food, there is a rapid burst of insulin secretion in proportion to the glucose-raising effect of the food. The goal of all but the simplest, 1-injection insulin regimens is to mimic this normal pattern of insulin secretion in response to food intake and activity patterns. [Table 46-4](#) describes several insulin regimens and the advantages and disadvantages of each.

TABLE 46-4 Insulin Regimens

Schematic Representation	Description	Advantages	Disadvantages
<p>Normal pancreas</p> <p>100 μU/mL</p> <p>0</p> <p>BR LU DI SN BR</p>	<p>Insulin release increases when blood glucose levels rise and continues at a low steady rate between meals</p>		
<p>1 injection per day</p> <p>Insulin effect</p> <p>Rapid-acting insulin</p> <p>NPH</p> <p>BR LU DI SN BR</p>	<p>Before breakfast:</p> <ul style="list-style-type: none"> • NPH or • NPH with rapid-acting insulin 	<p>Simple regimen</p>	<p>Difficult to control fasting blood glucose if effects of NPH do not last</p> <p>Afternoon hypoglycemia may result from attempts to control fasting glucose level by increasing NPH dose</p>
<p>2 injections per day—mixed</p> <p>Insulin effect</p> <p>Rapid-acting insulin</p> <p>NPH</p> <p>Rapid-acting insulin</p> <p>NPH</p> <p>BR LU DI SN BR</p>	<p>Before breakfast and dinner:</p> <ul style="list-style-type: none"> • NPH or • NPH with rapid-acting insulin or • Premixed (rapid-acting insulin) insulin 	<p>Simplest regimen that attempts to mimic normal pancreas</p>	<p>Need relatively fixed schedule of meals and exercise</p> <p>Cannot independently adjust NPH or regular if premixed insulin is used</p>
<p>3 or 4 injections per day</p> <p>Insulin effect</p> <p>Rapid-acting insulin</p> <p>Rapid-acting insulin</p> <p>Rapid-acting insulin</p> <p>NPH</p> <p>BR LU DI SN BR</p> <p>Insulin effect</p> <p>Rapid-acting insulin</p> <p>Rapid-acting insulin</p> <p>Rapid-acting insulin</p> <p>Long-acting insulin</p> <p>BR LU DI SN BR</p>	<p>Rapid-acting insulin before each meal with:</p> <ul style="list-style-type: none"> • NPH at dinner or • NPH at bedtime or • Long-acting insulin one or two times a day 	<p>More closely mimics normal pancreas than 3-injection regimen</p> <p>Each premeal dose of regular insulin decided independently</p> <p>More flexibility with meals and exercise</p>	<p>Requires more injections than other regimens</p> <p>Requires multiple blood glucose tests on a daily basis</p> <p>Requires intensive education and follow-up</p>
Insulin pump	<p>Uses ONLY rapid-acting insulin infused at continuous, low rate called <i>basal</i></p>	<p>Most closely mimics normal pancreas</p> <p>Decreases unpredictable peaks of</p>	<p>Requires intensive training and frequent follow-up</p> <p>Potential for mechanical</p>



Note: Rapid-acting insulin—lispro, aspart, or glulisine.

BR, breakfast; DI, dinner; ↑, insulin injections; LU, lunch; NPH, neutral protamine Hagedorn; regular; SN, snack.

Adapted from Comerford, K. C., & Durkin, M. T. (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer.

There are two general approaches to insulin therapy: conventional and intensive (described in detail later). The patient can learn to use SMBG results and carbohydrate counting to vary the insulin doses. This allows more flexibility in timing and content of meals and exercise periods. However, complex insulin regimens require a strong level of commitment, intensive education, and close follow-up by the health care team.

The patient should be very involved in the decision regarding which insulin regimen to use. The patient should compare the potential benefits of different regimens with the potential costs (e.g., time involved, number of injections, fingersticks for glucose testing, amount of record keeping). There are no set guidelines as to which insulin regimen should be used for which patient. Members of the health care team should not assume that older patients should automatically be given a simplified regimen, or that all people want to be involved in a complex treatment regimen. The nurse plays an important role in educating the patient about the various approaches to insulin therapy. The nurse should refer the patient to a diabetes and education care specialist, certified diabetes educator (CDE), or a diabetes education center, if available, for further training and education in the insulin treatment regimens.

Conventional Regimen. One approach is to simplify the insulin regimen as much as possible, with the aim of avoiding the acute complications of diabetes (hypoglycemia and symptomatic hyperglycemia). With this type of simplified regimen (e.g., one or more injections of a mixture of short- and intermediate-acting insulins per day), the patient should not vary meal patterns and activity levels. The simplified regimen would be appropriate for the terminally ill, the older adult who is frail and has limited self-care abilities, or patients who are completely unwilling or unable to engage in the self-management activities that are part of a more complex insulin regimen.

Intensive Regimen. The second approach is to use a more complex insulin regimen to achieve as much control over blood glucose levels as is safe and practical. A more complex insulin regimen allows the patient more flexibility to change the insulin doses from day to day in accordance with changes in eating and activity patterns, with stress and illness, and as needed for variations in the prevailing glucose level.

While intensive treatment (3 or 4 injections of insulin per day) reduces the risk of complications, not all people with diabetes are candidates for very tight control of blood glucose. The risk of severe hypoglycemia increases threefold in patients receiving intensive treatment (ADA, 2020). Patients who have received a kidney transplant because of nephropathy and chronic kidney disease should follow an intensive insulin regimen to preserve function of the new kidney.

Those who are not candidates include those with:

- Nervous system disorders rendering them unaware of hypoglycemic episodes (e.g., those with autonomic neuropathy)
- Recurring severe hypoglycemia
- Irreversible diabetic complications, such as blindness or ESKD
- Severe cerebrovascular or cardiovascular disease
- Ineffective self-care skills

Complications of Insulin Therapy

Local Allergic Reactions. A local allergic reaction (redness, swelling, tenderness, and induration or a 2- to 4-cm wheal) may appear at the injection site 1 to 2 hours after the administration of insulin. Reactions usually resolve in a few hours or days. If they do not resolve, another type of insulin can be prescribed (Comerford & Durkin, 2020).

Systemic Allergic Reactions. Systemic allergic reactions to insulin are rare. When they do occur, there is an immediate local skin reaction that gradually spreads into generalized urticaria (hives). These rare

reactions are occasionally associated with generalized edema or anaphylaxis. The treatment is desensitization, with small doses of insulin given in gradually increasing amounts using a desensitization kit.

Insulin Lipodystrophy. Lipodystrophy refers to a localized reaction, in the form of either lipoatrophy or lipohypertrophy, occurring at the site of insulin injections. Lipoatrophy is the loss of subcutaneous fat; it appears as slight dimpling or more serious pitting of subcutaneous fat. The use of human insulin has almost eliminated this disfiguring complication.

Lipohypertrophy, the development of fibrofatty masses at the injection site, is caused by the repeated use of an injection site. If insulin is injected into scarred areas, absorption may be delayed. This is one reason that rotation of injection sites is so important. Patients should avoid injecting insulin into these areas until the hypertrophy disappears.

Resistance to Injected Insulin. Patients may develop insulin resistance and require large insulin doses to control symptoms of diabetes (Comerford & Durkin, 2020). In most patients with diabetes who take insulin, immune antibodies develop and bind the insulin, thereby decreasing the insulin available for use. All insulins cause some antibody production in humans.

Very few patients who are resistant develop high levels of antibodies. Many of these patients have a history of insulin therapy interrupted for several months or longer. Treatment consists of administering a more concentrated insulin preparation, such as U-500, which is available by special order (Comerford & Durkin, 2020). U-500 insulin is never stored with other insulin preparations due to the risk of overdose if accidentally given to the wrong patient (Comerford & Durkin, 2020). Occasionally, corticosteroid therapy is needed to block the production of antibodies. This may be followed by a gradual reduction in the insulin requirement. Therefore, patients must monitor their blood for hypoglycemia.

Morning Hyperglycemia. An elevated blood glucose level on arising in the morning is caused by an insufficient level of insulin, which may be caused by several factors: the dawn phenomenon, the Somogyi effect, or insulin waning. The dawn phenomenon is characterized by a relatively normal blood glucose level until approximately 3 AM, when blood glucose levels begin to rise. The phenomenon is thought to result from nocturnal surges in growth hormone secretion, which creates a greater need for insulin in the early morning hours in patients with type 1 diabetes. It must be distinguished from insulin waning (the progressive increase in blood glucose from bedtime to morning) and from the Somogyi effect (nocturnal hypoglycemia followed by rebound hyperglycemia). Insulin waning is frequently seen if the evening NPH dose is given before dinner; it is prevented by moving the evening dose of NPH insulin to bedtime.

Chart 46-5

Characteristics and Treatment of Morning Hyperglycemia

Insulin Waning

Progressive rise in blood glucose from bedtime to morning.

Treated by increasing evening (predinner or bedtime) dose of intermediate- or long-acting insulin, or instituting a dose of insulin before the evening meal if one is not already part of the treatment regimen.

Dawn Phenomenon

Relatively normal blood glucose until early morning hours when levels begin to rise.

Treated by changing time of injection of evening intermediate-acting insulin from dinnertime to bedtime.

Somogyi Effect

Normal or elevated blood glucose at bedtime, early morning hypoglycemia, and a subsequent increased blood glucose caused by the production of counter-regulatory hormones.

Treated by decreasing evening (predinner or bedtime) dose of intermediate-acting insulin, or increasing bedtime snack.

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

It may be difficult to tell from a patient's history what the cause is for morning hyperglycemia. To determine the cause, the patient must be awakened once or twice during the night to test blood glucose levels. Testing at bedtime, at 3 am, and on awakening provides information that can be used to make adjustments in insulin to avoid morning hyperglycemia.

Chart 46-5 summarizes the differences among insulin waning, the dawn phenomenon, and the Somogyi effect.

Methods of Insulin Delivery

Methods of insulin delivery include traditional subcutaneous injections, insulin pens, jet injectors, and insulin pumps. (See later Nursing Management discussion of traditional subcutaneous injections.)

Insulin Pens. Insulin pens use small (150- to 300-unit) prefilled insulin cartridges that are loaded into a penlike holder. A disposable needle is attached to the device for insulin injection. Insulin is delivered by dialing in a dose or pushing a button for every 1- or 2-unit increment given. People using these devices still need to insert the needle for each injection (see Fig. 46-3); however, they do not need to carry insulin bottles or draw up insulin before each injection. These devices are most useful for patients who need to inject only one type of insulin at a time (e.g., premeal rapid-acting insulin three times a day, bedtime NPH insulin) or who can use the premixed insulins. These pens are convenient for those who administer insulin before dinner if eating out or traveling. They are also useful for patients with impaired manual dexterity, vision, or cognitive function, which makes the use of traditional syringes difficult.

Jet Injectors. As an alternative to needle injections, jet injection devices deliver insulin through the skin under pressure in an extremely fine stream. These devices are more expensive and require thorough training and supervision when first used. In addition, patients should be cautioned that absorption rates, peak insulin activity, and insulin levels may be different when changing to a jet injector. (Insulin given by jet injector is usually absorbed faster.) The use of jet injectors has been associated with bruising in some patients.



Figure 46-3 • Prefilled insulin syringe.

Insulin Pumps. Continuous subcutaneous insulin infusion involves the use of small, externally worn devices called insulin pumps (ADA, 2020). This technology mimics the functions of a healthy pancreas by providing automated systems that can adjust insulin delivery based on basal (background) insulin every 5 minutes. Insulin pumps contain a 3-mL syringe attached to a long (24- to 42-inch), thin, narrow-lumen tube with a needle or Teflon catheter attached to the end. The patient inserts the needle or catheter into subcutaneous tissue (usually on the abdomen) and secures it with tape or a transparent dressing. The needle or catheter is changed at least every 3 days. The pump is then worn either on the patient's clothing or in a pocket. Some women keep the pump tucked into the front or side of the bra. Additional accessories, such as belt, clip or pouch can be used to carry an insulin pump.

When an insulin pump is used, insulin is delivered by subcutaneous infusion at a basal rate that ranges from 0.25 to 2 units per hour depending on the device. When a meal is consumed, the patient calculates a dose of insulin to metabolize the meal by counting the total amount of carbohydrate for the meal using a predetermined insulin-to-carbohydrate ratio; for example, a ratio of 1 unit of insulin for every 15 g of carbohydrate would require 3 units for a meal with 45 g of carbohydrate. This allows flexibility of meal timing and content.

Possible disadvantages of insulin pumps are unexpected disruptions in the flow of insulin from the pump that may occur if the tubing or needle becomes occluded, if the supply of insulin runs out, or if the battery is depleted, increasing the risk of DKA. Effective education to produce knowledgeable patients minimizes this risk. There is the potential for infection at needle insertion sites. Hypoglycemia may occur with insulin pump therapy; however, this is usually related to the lowered blood glucose levels that many patients achieve rather than to a specific problem with the pump itself. The tight diabetes control associated with the use of an insulin pump may increase the incidence of hypoglycemia unawareness because of the very gradual decline in serum glucose level, from more than 70 mg/dL (3.9 mmol/L) to less than 60 mg/dL (3.3 mmol/L).

Some patients find that wearing the pump for 24 hours each day is inconvenient. However, the pump can easily be disconnected, per patient preference, for limited periods, such as for showering, exercise, swimming, or sexual activity.

Candidates for the insulin pump must be willing to assess their blood glucose level several times daily with either SMBG or CGM (ADA, 2020). In addition, they must be psychologically stable and comfortable about having diabetes, because the insulin pump is often a visible sign to others and a constant reminder to patients that they have diabetes. Most important, patients using insulin pumps must have extensive education in the use of the pump and in self-management of blood glucose and insulin doses. They must work closely with a team of health care professionals who are experienced in insulin pump therapy—specifically, a diabetologist/endocrinologist, a dietitian, and a diabetes and education specialist or CDE.

The most common risk of insulin pump therapy is DKA which can occur if there is an occlusion in the infusion set or tubing. Because only rapid-acting insulin is used in the pump, any interruption in the flow of insulin may rapidly cause the patient to be without insulin. The patient should be taught to administer insulin by manual injection if an insulin interruption is suspected (e.g., no response in blood glucose level after a meal bolus).

Many insurance companies cover the cost of pump therapy. If not, the extra expense of the pump and associated supplies may be a deterrent for some patients. Medicare covers insulin pump therapy for patients with type 1 diabetes.

Insulin pumps have been used in patients with type 2 diabetes whose beta-cell function has diminished and who require insulin. Patients with a hectic lifestyle often do well with an insulin pump. There is no risk of DKA when there is an interruption of the flow of insulin in people with type 2 diabetes wearing an insulin pump.

Transplantation of Pancreatic Cells. Transplantation of the whole pancreas or a segment of the pancreas is being performed on a limited population (mostly patients with diabetes who are receiving a kidney transplantation simultaneously) (Aref, Zayan, Pararajasingam, et al., 2019). Patients must weigh the risks of antirejection medications against the advantages of pancreas transplantation. Implantation of insulin-producing pancreatic islet cells is another approach. This latter approach involves a less extensive surgical procedure and a potentially lower incidence of immunogenic problems. However, thus far, independence from exogenous insulin has been limited to 2 years after transplantation of islet cells. Results of studies of patients younger than 50 years of age with islet cell transplants using less toxic antirejection drugs have shown some promise (Aref et al., 2019). These procedures are not widely available due to a shortage of organs for transplantation.

Oral Antidiabetic Agents

Oral antidiabetic agents may be effective for patients who have type 2 diabetes that cannot be treated effectively with MNT and exercise alone. In the United States, oral antidiabetic agents include second-generation sulfonylureas, biguanides, alpha-glucosidase inhibitors, non-sulfonylurea insulin secretagogues (meglitinides, phenylalanine derivatives), thiazolidinediones (glitazones), dipeptide peptidase-4 (DPP-4) inhibitors, glucagonlike peptide-1 receptor agonists (GLP-1), and sodium-glucose cotransporter 2 (SGL-2) inhibitors (see Table 46-5). The thiazolidinediones are a class of oral antidiabetic medications that reduce insulin resistance in target tissues, enhancing insulin action without directly stimulating insulin secretion. Second-generation sulfonylureas and meglitinides are insulin secretagogues (Keresztes & Peacock-Johnson, 2019). Patients must understand that oral agents are prescribed as an addition to (not a substitute for) other treatment modalities, such as MNT and exercise. The use of oral antidiabetic medications may need to be

halted temporarily and insulin prescribed if hyperglycemia develops that is attributable to infection, trauma, or surgery. See later section on glycemic control in the patient who is hospitalized.

Because mechanisms of action vary (see Fig. 46-4), effects may be enhanced with the use of a multidose, or more than one medication (ADA, 2020). A combination of oral agents with insulin, usually glargine at bedtime, has also been used as a treatment for some patients with type 2 diabetes. Insulin therapy may be used from the onset for newly diagnosed patients with type 2 diabetes who are symptomatic and have high blood glucose and A1C levels (ADA, 2020).

Other Pharmacologic Therapy

Additional medications are available for use in the pharmacologic management of diabetes. These injectables are adjunct therapies, not a substitute for insulin if insulin is required to control diabetes.

Pramlintide, a synthetic analogue of human amylin, a hormone that is secreted by the beta cells of the pancreas, is approved for treatment of both type 1 and type 2 diabetes (Comerford & Durkin, 2020). It is used to control hyperglycemia in adults who have not achieved acceptable levels of glucose control despite the use of insulin at mealtimes. It is used with insulin, not in place of insulin. It acts to slow the rate at which food leaves the stomach and reduces appetite (Comerford & Durkin, 2020). The goal of therapy is to minimize fluctuations in daily glucose levels and provide better glucose control. Pramlintide must be injected subcutaneously 2 in from an insulin injection site (Comerford & Durkin, 2020). Patients are instructed to monitor their blood glucose levels closely during the initial period of use of pramlintide.

Nursing Management

The logo consists of the words "WATCH & LEARN" in a blue sans-serif font. The ampersand is replaced by a magnifying glass icon. To the left of the text is a small circular icon containing a stylized eye.

Nursing management of patients with diabetes can involve treatment of a wide variety of physiologic disorders, depending on the patient's health status and whether the patient is newly diagnosed or seeking care for an unrelated health problem. Glucose control in patients diagnosed with diabetes as well as those who have not been diagnosed is an important consideration in the hospital setting. Nursing management of patients with DKA and HHS and of those with diabetes as a secondary diagnosis is discussed in subsequent sections of this chapter.

Because all patients with diabetes must master the concepts and skills necessary for long-term management and avoidance of potential complications of diabetes, a solid educational foundation is necessary for competent self-care and is an ongoing focus of nursing care.

TABLE 46-5



Select Oral Antidiabetic Medications

Generic Name	Action/Indications	Side Effects	Nursing Implications
Alpha-Glucosidase Inhibitors			
acarbose miglitol	Delay absorption of complex carbohydrates in the intestine and slow entry of glucose into systemic circulation Does not increase insulin secretion Can be used alone or in combination with sulfonylureas, metformin, or insulin to improve glucose control	Hypoglycemia (risk increased if used with insulin or other antidiabetic agents) GI side effects (abdominal discomfort or distention, diarrhea, flatulence) Drug–drug interactions	Must be taken with first bite of food to be effective Monitor for GI side effects (diarrhea, abdominal distention). Monitor for blood glucose levels to assess effectiveness of therapy. Monitor liver function studies every 3 mo for 1 yr, then periodically. Contraindicated in patients with GI or kidney dysfunction, or cirrhosis. Alert: Hypoglycemia must be treated with glucose, not sucrose.
Biguanides			
metformin metformin with glyburide	Inhibit production of glucose by the liver Increase body tissue sensitivity to insulin Decrease hepatic synthesis of cholesterol	Lactic acidosis Hypoglycemia if metformin is used in combination with insulin or other antidiabetic agents Drug–drug interaction GI disturbances Contraindicated in patients with impaired kidney or liver function, respiratory insufficiency, severe infection, or alcohol abuse	Monitor for lactic acidosis and hypoglycemia. Monitor kidney function. Patients taking metformin are at increased risk for acute kidney injury and lactic acidosis with the use of iodinated contrast material for diagnostic studies; metformin should be stopped 48 h prior to and for 48 h after the use of contrast agent or until kidney function is evaluated and normal. Check for interactions with other medications.
Dipeptidyl Peptidase-4 (DPP-4) Inhibitors			
alogliptin linagliptin saxagliptin sitagliptin vildagliptin	Increase and prolong the action of incretin, a hormone that increases insulin release and decreases glucagon levels, with the result of improved glucose control	Upper respiratory infection Stuffy or runny nose and sore throat Headache Stomach discomfort and diarrhea Hypoglycemia, if used with sulfonylurea	Usually given once a day. Used alone or with other oral antidiabetic agents. Instruct patient about signs and symptoms of hypoglycemia and other adverse effects to report. Monitor kidney function.
Glucagonlike peptide-1 agonist (GLP-1)			
dulaglutide liraglutide	Enhances glucose-dependent insulin secretion and exhibit other antihyperglycemic actions following their release into the circulation from the gastrointestinal tract.	Pancreatitis, weight loss, diarrhea, nausea, vomiting, reaction at injection site, cough	Given once a day by subcutaneous injection.
Non-Sulfonylurea Insulin Secretagogues			
nateglinide categorized as a D-phenylalanine derivative repaglinide categorized as a meglitinide	Stimulate pancreas to secrete insulin Can be used alone or in combination with metformin or thiazolidinediones to improve glucose control	Hypoglycemia/weight gain less likely than sulfonylureas Drug–drug interactions (with ketoconazole, fluconazole, erythromycin, rifampin, isoniazid)	Monitor blood glucose levels to assess effectiveness of therapy. Has rapid action and short half-life Should be taken only if able to eat a meal immediately. Educate patients about symptoms of hypoglycemia. Monitor patients with impaired liver function and renal impairment. Has no effect on plasma lipids. Is taken before each meal. Check for interactions with other medications.
Second-Generation Sulfonylureas			
glimepiride	Stimulate beta cells of the	Hypoglycemia	Monitor patient for hypoglycemia.

glipizide glyburide	pancreas to secrete insulin; may improve binding between insulin and insulin receptors or increase the number of insulin receptors Have more potent effects than first-generation sulfonylureas May be used in combination with metformin or insulin to improve glucose control	Mild GI symptoms Weight gain Drug-drug interactions (NSAIDs, warfarin, sulfonamides)	Monitor blood glucose and urine ketone levels to assess effectiveness of therapy. Patients at high risk for hypoglycemia: advanced age, renal insufficiency. When taken with beta-adrenergic blocking agents, may mask usual warning signs and symptoms of hypoglycemia. Instruct patients to avoid the use of alcohol. Contraindicated with sulfa allergy.
Sodium-glucose co-transporter 2 (SGL-2) Inhibitors			
anagliptin dapagliflozin empagliflozin	Prevents the kidneys from reabsorbing glucose back into the blood, therefore lowering glucose by releasing glucose into the urine	Urinary tract infections Hypoglycemia May increase LDL and HDL cholesterol	Should be taken once daily before first meal in the morning. Monitor for genital or urinary tract infections.
Thiazolidinediones (or Glitazones)			
pioglitazone rosiglitazone	Sensitize body tissue to insulin; stimulate insulin receptor sites to lower blood glucose and improve action of insulin May be used alone or in combination with sulfonylurea, metformin, or insulin	Hypoglycemia (risk increased with the use of insulin or other antidiabetic agents) Anemia Weight gain, edema Decrease effectiveness of oral contraceptives Possible liver dysfunction Drug-drug interactions Hyperlipidemia (has variable effect on lipids; pioglitazone may be preferred choice in patients with lipid abnormalities) Impaired platelet function	Monitor blood glucose levels to assess effectiveness of therapy. Monitor liver function tests. Arrange dietary education to establish weight control program. Instruct patient taking oral contraceptives about increased risk of pregnancy.

GI, gastrointestinal; NSAIDs, nonsteroidal anti-inflammatory drugs.

Adapted from Comerford, K. C., & Durkin, M. T. (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer.

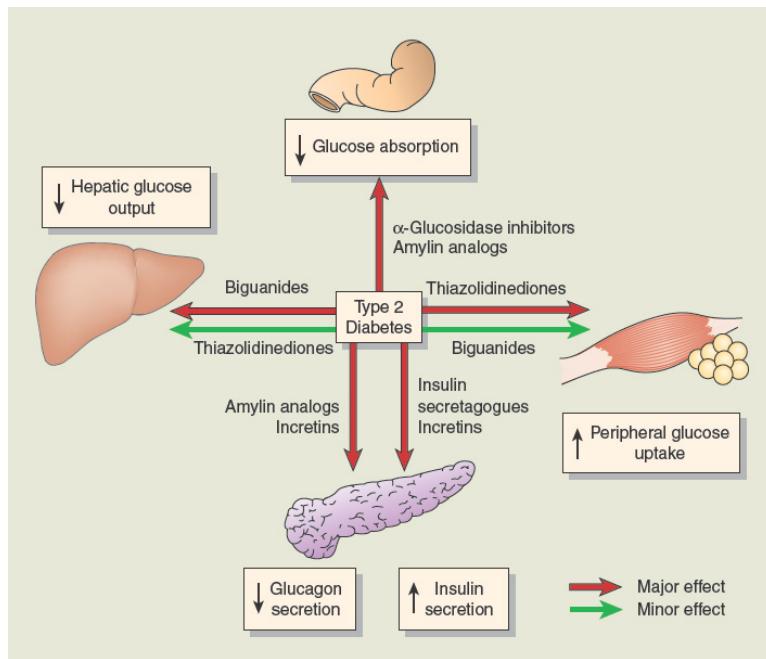


Figure 46-4 • Action sites of hypoglycemic agents and mechanisms of lowering blood glucose in type 2 diabetes. The incretins are the dipeptidyl peptidase-4 inhibitors and glucagonlike peptide-1 agonists.

Managing Glucose Control in the Hospital Setting

Hyperglycemia can prolong lengths of stay and increase infection rates and mortality; thus, nurses need to address glucose management in all hospital patients. Hyperglycemia occurs most often in patients with known diabetes (i.e., type 1, type 2, gestational) and in those newly diagnosed with diabetes or stress hyperglycemia. Nursing management of hyperglycemia in the hospital uses the following principles (ADA, 2020):

- Blood glucose targets are 140 to 180 mg/dL.
- Insulin (subcutaneous or IV) is preferred to oral antidiabetic agents to manage hyperglycemia.
- Hospital insulin protocols or order sets should minimize complexity, ensure adequate staff training, include standardized hypoglycemic treatment, and make guidelines available for glycemic goals and insulin dosing.
- Appropriate timing of blood glucose checks, meal consumption, and insulin dose are all crucial for glucose control and to avoid hypoglycemia.



Providing Patient Education

Diabetes is a chronic illness that requires a lifetime of special self-management behaviors (ADA, 2020). Because MNT, physical activity, medication, and physical and emotional stress affect diabetic control, patients must learn to balance a multitude of factors.

Developing a Diabetes Education Plan

Patients with new-onset type 1 diabetes are hospitalized for short periods or may be managed completely on an outpatient basis. Patients with new-onset type 2 diabetes are rarely hospitalized for initial care. Outpatient diabetes education and training programs have proliferated with the availability of third-party reimbursement. All encounters with patients with diabetes are opportunities for reinforcement of self-management skills, regardless of the setting.

Many health systems employ nurses and registered dietitians who specialize in diabetes education and management and who are certified by the National Certification Board for Diabetes Educators as CDEs. However, because of the large number of patients with diabetes all nurses play a vital role in identifying patients with diabetes, assessing self-care skills, providing basic education, reinforcing the education

provided by the specialist, and referring patients for follow-up care. Diabetes patient education programs that have been peer-reviewed by the ADA as meeting National Standards for Diabetes Self-Management Education can be reimbursed for education (Davidson, Ross, & Castor, 2018).

Unfolding Patient Stories: Skyler Hansen • Part 2



Recall from [Chapter 5 Skyler Hansen](#), a high school student recently diagnosed with type 1 diabetes. Outline a diabetes education plan for Skyler and his parents. What are important patient education topics, resources, and methods for the nurse to consider? How would the nurse evaluate whether the patient and his family understand the education provided?

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

Organizing Information

There are various strategies for organizing and prioritizing the vast amount of information that must be taught to patients with diabetes. In addition, many hospitals and outpatient diabetes centers have devised written guidelines, care plans, and documentation forms (often based on ADA guidelines) that may be used to document and evaluate education (Davidson et al., 2018).

A general approach is to organize information and skills into two main categories: basic or initial, and in-depth (advanced) or continuing education.

Educating Patients About Basic Skills. Basic skills must be learned by all patients with newly diagnosed type 1 or type 2 diabetes and all patients receiving insulin for the first time. Basic information that patients must know is included in [Chart 46-6](#).

For patients with newly diagnosed type 2 diabetes, emphasis is initially placed on meal planning, exercise, and weight loss if applicable. Those who are starting to take oral antidiabetic agents need to know about detecting, preventing, and treating hypoglycemia. If diabetes has gone undetected for many years, the patient may already be experiencing some chronic complications from diabetes. Therefore, for some patients with newly diagnosed type 2 diabetes, basic diabetes education must include information on preventive skills, such as foot care (Davidson et al., 2018) and eye care (e.g., planning yearly or more frequent complete [dilated eye] examinations by an ophthalmologist, understanding that retinopathy is largely asymptomatic until advanced stages).

Patients also need to realize that once they master the basic skills and information, diabetes education is a life-long process. Acquiring in-depth and advanced diabetes knowledge occurs both formally through programs of continuing education and informally through experience and sharing of information with other people with diabetes.

Chart 46-6



PATIENT EDUCATION

Basic Skills for People with Diabetes

The nurse includes the following basic information in education:

- 1. Pathophysiology**
 - a. Basic definition of diabetes (having a high blood glucose level)
 - b. Normal blood glucose ranges and target blood glucose levels
 - c. Effect of insulin and exercise (decrease glucose)
 - d. Effect of food and stress, including illness and infections (increase glucose)
 - e. Basic treatment approaches
- 2. Treatment modalities**
 - a. Administration of insulin and oral antidiabetic medications
 - b. Meal planning (food groups, timing of meals)
 - c. Monitoring of blood glucose and urine ketones
- 3. Recognition, treatment, and prevention of acute complications**
 - a. Hypoglycemia
 - b. Hyperglycemia
- 4. Pragmatic information**
 - a. Where to buy and store insulin, syringes, and glucose monitoring supplies
 - b. When and how to contact the primary provider

Adapted from American Diabetes Association (ADA). (2020). Standards of medical care in diabetes—2020. *Diabetes Care*, 43(Suppl 1), S1–S212.

Planning In-Depth and Continuing Education. This education involves more details related to basic skills (e.g., learning to vary food choices [carbohydrate counting], type of insulin, preparing for travel) as well as learning preventive measures for avoiding long-term complications from diabetes. Preventive measures include foot care, eye care, general hygiene (e.g., skin care, oral hygiene), and risk factor management (e.g., control of blood pressure, blood glucose, cholesterol, weight) (ADA, 2020; Davidson et al., 2018).

More advanced continuing education may include alternative methods for insulin delivery, such as the insulin pump, CGM, and algorithms or rules for evaluating and adjusting insulin doses. The degree of advanced diabetes education to be provided depends on the patient's interest and ability. However, learning preventive measures (especially foot and eye care) is mandatory for early detection and treatment to reduce the occurrence of amputations and blindness in patients with diabetes.

Assessing Readiness to Learn

Before initiating diabetes education, the nurse assesses the patient's (and family's) readiness to learn. When patients are first diagnosed with diabetes (or first told of their need for insulin), they often go through stages of the grieving process. These stages may include shock and denial, anger, depression, negotiation, and acceptance. The amount of time it takes for the patient and family members to work through the grieving process varies from patient to patient. They may experience helplessness, guilt, altered body image, loss of self-esteem, and concern about the future. The nurse must assess the patient's coping strategies and reassure the patient and family that feelings of depression and shock are normal.

Asking the patient and family about their major concerns or fears is an important way to learn about any misinformation that may be contributing to anxiety. Simple, direct information should be provided to dispel misconceptions. Once the patient masters basic skills, more information is provided.

Patients who are in the hospital rarely have the luxury of waiting until they feel ready to learn; short lengths of hospital stay necessitate initiation of basic skill education as early as possible. This gives the patient the opportunity to practice skills with supervision by the nurse before discharge. Follow-up in the home is often necessary for reinforcement of skills.

The nurse evaluates the patient's social situation for factors that may influence the diabetes treatment and education plan, such as:

- Low literacy level (may be evaluated while assessing for visual deficits by having the patient read from educational materials)
- Limited financial resources or lack of health insurance
- Presence or absence of family support
- Typical daily schedule (the patient is asked about the timing and number of usual daily meals, work and exercise schedule, plans for travel)

- Cognitive deficits or other disabling conditions, obtained from the patient's health history and physical assessment (the patient is assessed for aphasia or decreased ability to follow simple commands)
- Cultural beliefs may also impact adherence to a regimen.

Educating Experienced Patients

Nurses need to annually assess the skills and self-care behaviors of patients who have had diabetes for many years (Davidson et al., 2018). Assessment of these patients must include direct observation of skills, not just the patient's self-report of self-care behaviors. In addition, these patients must be fully aware of preventive measures related to foot care, eye care, and risk factor management. Those experiencing long-term complications from diabetes for the first time may go through the grieving process again. Some patients may have a renewed interest in diabetes self-care in the hope of delaying further complications. Others may have guilt and depression. The patient is encouraged to discuss feelings and fears related to complications. Meanwhile, the nurse provides appropriate information regarding complications from diabetes.

Determining Education Methods

Maintaining flexibility with regard to education approaches is important. Providing education on skills and information in a logical sequence is not always the most helpful method for patients. For example, many patients fear self-injection. Before they learn how to prepare, purchase, store, and mix insulins, they should be taught to insert the needle and inject insulin (or practice with saline solution).

Various tools can be used to complement education. Many of the companies that manufacture products for diabetes self-care also provide booklets, videotapes, DVDs, or on-line materials to assist in patient education. Educational materials are also available from many sources (see the Resources section). It is important to use a variety of written handouts that match the patient's learning needs (including different languages, low-literacy information, and large print) and reading level and to ensure that these materials are technically accurate. Patients can continue learning about diabetes care by participating in community-based educational programs and other sources such as Web-based programs (Davidson et al., 2018).

Educating Patients to Self-Administer Insulin

Insulin injections are self-administered into the subcutaneous tissue with the use of special insulin syringes. Basic information includes explanations of the equipment, insulins, and syringes and how to mix insulin, if necessary.

Storing Insulin

Whether insulin is the short- or the long-acting preparation, vials not in use, including spare vials or pens, should be refrigerated. Extremes of temperature should be avoided; insulin should not be allowed to freeze and should not be kept in direct sunlight or in a hot car. The insulin vial in use should be kept at room temperature to reduce local irritation at the injection site, which may occur if cold insulin is injected. If a vial of insulin will be used up within 1 month, it may be kept at room temperature. The patient should be instructed to always have a spare vial of the type or types of insulin used (ADA, 2020). Cloudy insulins should be thoroughly mixed by gently inverting the vial or rolling it between the hands before drawing the solution into a syringe or a pen (Comerford & Durkin, 2020). The patient needs to be educated to pay attention to the expiration date on any type of insulin.

Bottles of intermediate-acting insulin should also be inspected for flocculation, which is a frosted, whitish coating inside the bottle. This occurs most commonly with insulins that are exposed to extremes of temperature. If a frosted, adherent coating is present, some of the insulin is bound, inactive, and should not be used.

Selecting Syringes

Syringes must be matched with the insulin concentration (e.g., U-100). Currently, three sizes of U-100 insulin syringes are available:

- 1-mL syringe, 100-unit capacity
- 0.5-mL syringe, 50-unit capacity
- 0.3-mL syringe, 30-unit capacity

The concentration of insulin used in the United States is U-100; that is, there are 100 units per milliliter (or cubic centimeter). Small syringes allow patients who require small amounts of insulin to measure and draw up the amount of insulin accurately. There is a U-500 (500 units/mL) concentration of insulin available by special order for patients who have severe insulin resistance and require massive doses of insulin.

Most insulin syringes have a disposable 27- to 29-gauge needle that is approximately 0.5 in long. The smaller syringes are marked in 1-unit increments and may be easier to use for patients with visual deficits

and those taking very small doses of insulin. The 1-mL syringes are marked in 1- and 2-unit increments. A small disposable insulin needle (31 gauge, 8 mm long) is available for very thin patients and children.

Mixing Insulins

When rapid- or short-acting insulins are to be given simultaneously with longer-acting insulins, they are usually mixed together in the same syringe; the longer-acting insulins must be mixed thoroughly before drawing into the syringe. It is important that patients prepare their insulin injections consistently from day to day.

There are varying opinions regarding which type of insulin (short- or longer-acting) should be drawn up into the syringe first when they are going to be mixed, but the ADA recommends that the regular insulin be drawn up first. The most important issues are that patients are consistent in technique, so as not to draw up the wrong dose in error or the wrong type of insulin, and that patients not inject one type of insulin into the bottle containing a different type of insulin. Injecting cloudy insulin into a vial of clear insulin contaminates the entire vial of clear insulin and alters its action.

For patients who have difficulty mixing insulins, several options are available. They may use a premixed insulin, they may have prefilled syringes prepared (see Fig. 46-3), or they may take 2 injections. Premixed insulins are available in many different ratios of NPH insulin to regular insulin (Comerford & Durkin, 2020). The ratio of 70/30 (70% NPH and 30% regular insulin in one bottle) is most common. Combinations with a ratio of 75% NPL (neutral protamine lispro) and 25% insulin lispro are also available. The appropriate initial dosage of premixed insulin must be calculated so that the ratio of NPH to regular insulin most closely approximates the separate doses needed.

For patients who can inject insulin but who have difficulty drawing up a single or mixed dose, syringes may be prefilled with the help of home health nurses or family and friends. A 3-week supply of insulin syringes may be prepared and kept in the refrigerator but warmed to room temperature before administration. The prefilled syringes should be stored with the needle in an upright position to avoid clogging of the needle; they should be mixed thoroughly by inverting syringe several times before the insulin is injected.

Withdrawing Insulin

Most (if not all) of the printed materials available on insulin dose preparation instruct patients to inject air into the bottle of insulin equivalent to the number of units of insulin to be withdrawn. The rationale for this is to prevent the formation of a vacuum inside the bottle, which would make it difficult to withdraw the proper amount of insulin.

Selecting and Rotating the Injection Site

The four main areas for injection are the abdomen, upper arms (posterior surface), thighs (anterior surface), and hips (see Fig. 46-5). Insulin is absorbed faster in some areas of the body than others. The speed of absorption is greatest in the abdomen and decreases progressively in the arm, thigh, and hip, respectively.

Systematic rotation of injection sites within an anatomic area is recommended to prevent lipodystrophy (localized changes in fatty tissue). In addition, to promote consistency in insulin absorption, the patient should be encouraged to use all available injection sites within one area rather than randomly rotating sites from area to area. For example, some patients almost exclusively use the abdominal area, administering each injection 0.5 to 1 inch away from the previous injection. Another approach to rotation is always to use the same area at the same time of day. For example, patients may inject morning doses into the abdomen and evening doses into the arms or legs.

A few general principles apply to all rotation patterns. First, the patient should try not to use the exact same site more than once in 2 to 3 weeks. In addition, if the patient is planning to exercise, insulin should not be injected into the limb that will be exercised because this will cause the drug to be absorbed faster, which may result in hypoglycemia.

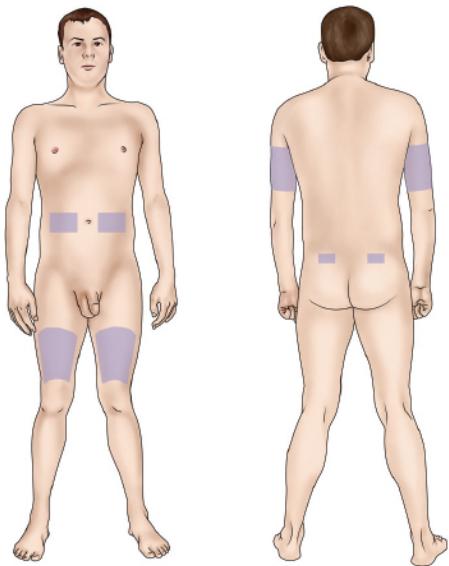


Figure 46-5 • Suggested areas for insulin injection.

Chart 46-7

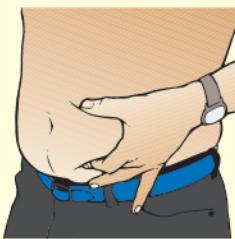


PATIENT EDUCATION

Self-Injection of Insulin

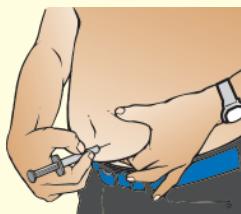
The nurse instructs the patient to:

1. With one hand, stabilize the skin by spreading it or pinching up a large area.



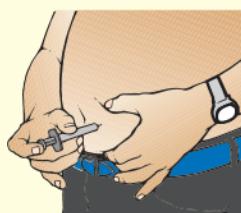
Pinching the skin

2. Pick up syringe with the other hand, and hold it as you would a pencil. Insert needle straight into the skin.^a



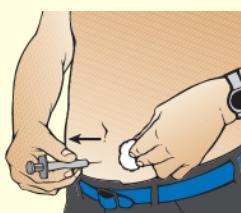
Inserting the needle into the skin

3. To inject the insulin, push the plunger all the way in.



Injecting the insulin

4. Pull needle straight out of skin. Press cotton ball over injection site for several seconds.



Removing the needle and holding cotton ball over site

5. Use disposable syringe *only once* and discard into hard plastic container (with a tight-fitting top) such as an empty bleach or detergent container.^b Follow state regulations for disposal of syringes and needles.



Disposing of syringe

^aSome patients may be taught to insert the needle at a 45-degree angle.

^bAlthough some studies suggest that reusing disposable syringes may be safe, it is recommended that this be done only in the absence of poor personal hygiene, an acute concurrent illness, open wounds on the hands, or decreased resistance to infection.

Preparing the Skin

The use of alcohol to cleanse the skin is not necessary, but patients who have learned this technique often continue to use it. They should be cautioned to allow the skin to dry after cleansing with alcohol. If the skin is not allowed to dry before the injection, the alcohol may be carried into the tissues, resulting in a localized reddened area and a burning sensation.

Inserting the Needle

There are varying approaches to inserting the needle for insulin injections. The correct technique is based on the need for the insulin to be injected into the subcutaneous tissue (see [Chart 46-7](#)). Injection that is too deep (e.g., intramuscular) or too shallow (intradermal) may affect the rate of absorption of the insulin. For a normal or overweight person, a 90-degree angle is the best insertion angle. Aspiration (inserting the needle and then pulling back on the plunger to assess for blood being drawn into the syringe and needle in vein) is not necessary. Many patients who have been using insulin for an extended period have eliminated this step from their insulin injection routine with no apparent adverse effects. [Chart 46-8](#) details how to evaluate the effectiveness of self-injection of insulin education.

Disposing of Syringes and Needles

Insulin syringes and pens, needles, and lancets should be disposed of according to local regulations. If community disposal programs are unavailable, used sharps should be placed in a puncture-resistant container. The patient should contact local trash authorities for instructions about proper disposal of filled containers, which should not be mixed with containers to be recycled.

Promoting Home, Community-Based, and Transitional Care



Educating the Patient About Self-Care

If poor glucose control or preventable complications occur, the nurse needs to assess the reasons for the patient's ineffective management of the treatment regimen. It should not be assumed that problems with diabetes management are related to the patient's decision to ignore self-management. The patient may have forgotten or may have never learned certain information, or there may be cultural or religious beliefs that interfere with adherence. The problem may be correctable simply through providing complete information and ensuring that the patient understands the information. The focus of diabetes education should be patient empowerment. Patient education must address behavior change, self-efficacy, and health beliefs.

Chart 46-8

Criteria for Determining Effectiveness of Self-Injection of Insulin Education

Equipment

Insulin

1. Identifies information on label of insulin bottle:
 - Type (e.g., NPH, regular, 70/30)
 - Manufacturer
 - Concentration (e.g., U-100)
 - Expiration date
2. Checks appearance of insulin:
 - Clear or milky white
 - Checks for flocculation (clumping, frosted appearance)
 - Identifies where to purchase and store insulin:
 - Indicates approximately how long bottle will last (1000 units per bottle U-100 insulin)
 - Indicates how long opened bottles can be used

Syringes

1. Identifies concentration (U-100) marking on syringe
2. Identifies size of syringe (e.g., 100 units, 50 units, 30 units)
3. Describes appropriate disposal of used syringe

Preparation and Administration of Insulin Injection

1. Draws up correct amount and type of insulin
2. Properly mixes 2 insulins if necessary
3. Inserts needle and injects insulin
4. Describes site rotation:
 - Demonstrates injection with all anatomic areas to be used
 - Describes pattern for rotation, such as using abdomen only or using certain areas at the same time of day
 - Describes system for remembering site locations, such as horizontal pattern across the abdomen as if drawing a dotted line

Knowledge of Insulin Action

1. Lists prescription:
 - Type and dosage of insulin
 - Timing of insulin injections
2. Describes approximate time course of insulin action:
 - Identifies long- and short-acting insulins by name
 - States approximate time delay until the onset of insulin action
 - Identifies need to delay food until 5 to 15 minutes after injection of rapid-acting insulin (lispro, aspart, glulisine)
 - Knows that longer time delays are safe when blood glucose level is high and that time delays may need to be shortened when blood glucose level is low

Incorporation of Insulin Injections into Daily Schedule

1. Recites proper order of premeal diabetes activities:
 - May use mnemonic device such as the word "tie," which helps the patient remember the order of activities ("t" = test [blood glucose], "i" = insulin injection, "e" = eat)
 - Describes daily schedule, such as test, insulin, eat before breakfast and dinner; test and eat, before lunch and bedtime
2. Describes information regarding hypoglycemia:
 - Symptoms: shakiness, sweating, nervousness, hunger, weakness
 - Causes: too much insulin, too much exercise, not enough food
 - Treatment: 15-g concentrated carbohydrate, such as 2 or 3 glucose tablets, 1 tube glucose gel, 0.5 cup juice
 - After initial treatment, follow with snack including starch and protein, such as cheese and crackers, milk and crackers, half sandwich.
3. Describes information regarding prevention of hypoglycemia:
 - Avoids delays in meal timing

- Eats a meal or snack approximately every 4 to 5 hours (while awake)
 - Does not skip meals
 - Increases food intake before exercise if blood glucose level is less than 100 mg/dL
 - Checks blood glucose regularly
 - Identifies safe modification of insulin doses consistent with management plan
 - Carries a form of fast-acting sugar at all times
 - Wears a medical identification bracelet
 - Educates family, friends, and coworkers about signs and treatment of hypoglycemia
 - Has family, roommates, and traveling companions learn to use injectable glucagon for severe hypoglycemic reactions
4. Maintains regular follow-up for evaluation of diabetes control:
- Keeps written record of blood glucose, insulin doses, hypoglycemic reactions, variations in diet
 - Keeps all appointments with health professionals
 - Sees primary provider regularly (usually two to four times per year)
 - States how to contact primary provider in case of emergency
 - States when to call primary provider to report variations in blood glucose levels

If knowledge deficit is not the issue, physical or emotional factors may be impairing the patient's ability to perform self-care skills. For example, decreased visual acuity may impair the patient's ability to administer insulin accurately, measure the blood glucose level, or inspect the skin and feet. In addition, decreased joint mobility (especially in older adults) or preexisting disability may impair the patient's ability to inspect the bottom of the feet. Denial of the diagnosis or depression may impair the patient's ability to carry out multiple daily self-care measures. The patient whose family, personal, or work problems may be of higher priority may benefit from assistance in establishing priorities. The nurse must also assess the patient for infection or emotional stress, which may lead to elevated blood glucose levels despite adherence to the treatment regimen.

The following approaches are helpful for promoting self-care management skills:

- Address any underlying factors (e.g., knowledge deficit, self-care deficit, illness) that may affect control of diabetes.
- Simplify the treatment regimen if it is too difficult for the patient to follow.
- Adjust the treatment regimen to meet patient requests (e.g., adjust diet or insulin schedule to allow increased flexibility in meal content or timing).
- Establish a specific plan or contract with each patient with simple, measurable goals.
- Provide positive reinforcement of self-care behaviors performed instead of focusing on behaviors that were neglected (e.g., positively reinforce blood glucose tests that were performed instead of focusing on the number of missed tests).
- Help the patient identify personal motivating factors rather than focusing on wanting to please primary providers.
- Encourage the patient to pursue life goals and interests, and discourage an undue focus on diabetes.

Continuing and Transitional Care

The degree to which patients interact with primary providers to obtain ongoing care depends on many factors. Age, socioeconomic level, existing complications, type of diabetes, and comorbid conditions may dictate the frequency of follow-up visits. Many patients with diabetes are seen by home health, community-based, or transitional care nurses for diabetes education, wound care, insulin preparation, or assistance with glucose monitoring. Even patients who achieve excellent glucose control and have no complications can expect to see their primary provider at least twice a year for ongoing evaluation and should receive routine nutrition updates. In addition, the nurse should remind the patient to participate in recommended health promotion activities (e.g., annual flu vaccines) and age-appropriate health screenings (e.g., pelvic examinations, mammograms).

Participation in support groups (in person or online) is encouraged for patients who have had diabetes for many years as well as for those who are newly diagnosed. Such participation may help the patient and family cope with changes in lifestyle that occur with the onset of diabetes and its complications. People who participate in support groups often share valuable information and experiences and learn from others. Support groups provide an opportunity for discussion of strategies to deal with diabetes and its management and to clarify and verify information with nurses or other health care professionals leading to healthier behaviors.

Acute Complications of Diabetes



There are three major acute complications of diabetes related to short-term imbalances in blood glucose levels: hypoglycemia, DKA, and HHS (Fayfman, Pasquel, & Umpeirrez, 2017).

Hypoglycemia (Insulin Reactions)

Hypoglycemia means low (hypo) sugar in the blood (glycemia) and occurs when the blood glucose falls to less than 70 mg/dL (3.9 mmol/L) (ADA, 2020). It can occur when there is too much insulin or oral hypoglycemic agents, too little food, or excessive physical activity. Hypoglycemia may occur at any time of the day or night. It often occurs before meals, especially if meals are delayed or snacks are omitted. For example, midmorning hypoglycemia may occur when the morning insulin is peaking, whereas hypoglycemia that occurs in the late afternoon coincides with the peak of the morning NPH insulin. Middle-of-the-night hypoglycemia may occur because of peaking evening or predinner NPH insulins, especially in patients who have not eaten a bedtime snack.



Gerontologic Considerations

In older patients with diabetes, hypoglycemia is a particular concern for many reasons:

- Older adults frequently live alone and may not recognize the symptoms of hypoglycemia.
- With decreasing kidney function, it takes longer for oral hypoglycemic agents to be excreted by the kidneys.
- Skipping meals may occur because of decreased appetite or financial limitations.
- Decreased visual acuity may lead to errors in insulin administration.

Clinical Manifestations

The clinical manifestations of hypoglycemia may be grouped into two categories: adrenergic symptoms and central nervous system (CNS) symptoms.

In mild hypoglycemia, as the blood glucose level falls, the sympathetic nervous system is stimulated, resulting in a surge of epinephrine and norepinephrine. This causes symptoms such as sweating, tremor, tachycardia, palpitation, nervousness, and hunger.

In moderate hypoglycemia, the drop in blood glucose level deprives the brain cells of needed fuel for functioning. Signs of impaired function of the CNS may include inability to concentrate, headache, lightheadedness, confusion, memory lapses, numbness of the lips and tongue, slurred speech, impaired coordination, emotional changes, irrational or combative behavior, double vision, and drowsiness. Any combination of these symptoms (in addition to adrenergic symptoms) may occur with moderate hypoglycemia.

In severe hypoglycemia, CNS function is so impaired that the patient needs the assistance of another person for treatment of hypoglycemia. Symptoms may include disoriented behavior, seizures, difficulty arousing from sleep, or loss of consciousness.



Concept Mastery Alert

It is important to check the patient's blood glucose level and correlate it with the patient's symptoms. If the blood glucose level is low but the patient is not exhibiting any symptoms, the nurse should double-check the glucose level to ensure that it is correct.

Assessment and Diagnostic Findings

Symptoms of hypoglycemia may occur suddenly and vary considerably from person to person. Decreased hormonal (adrenergic) response to hypoglycemia may contribute to lack of symptoms of hypoglycemia. This occurs in some patients who have had diabetes for many years. It may be related to autonomic neuropathy, which is a chronic diabetic complication (see later discussion). As the blood glucose level falls, the normal surge in adrenalin does not occur, and the usual adrenergic symptoms, such as sweating and shakiness, do not take place. The hypoglycemia may not be detected until moderate or severe CNS impairment occurs.

Affected patients must perform SMBG on a frequent regular basis, especially before driving or engaging in other potentially dangerous activities.

Management

Treating with Carbohydrates

Immediate treatment must be given when hypoglycemia occurs (ADA, 2020). The usual recommendation is for 15 to 20 g of a fast-acting concentrated source of carbohydrate. It is not necessary to add sugar to juice, even if it is labeled as unsweetened juice, because the fruit sugar in juice contains enough carbohydrate to raise the blood glucose level. Adding table sugar to juice may cause a sharp increase in the blood glucose level, and patients may experience hyperglycemia for hours after treatment.

Initiating Emergency Measures

In adults whose glucose level is less than 54 mg/dL (3.0 mmol/L) or who are unconscious and cannot swallow, an injection of glucagon 1 mg can be given either subcutaneously or intramuscularly (ADA, 2020). Glucagon is a hormone produced by the alpha cells of the pancreas that stimulates the liver to breakdown glycogen, the stored glucose. Injectable glucagon is packaged as a powder in 1-mg vials and must be mixed with a diluent immediately before being injected. After injection of glucagon, the patient may take as long as 20 minutes to regain consciousness. A concentrated source of carbohydrate followed by a snack should be given to the patient on awakening to prevent recurrence of hypoglycemia (because the duration of the action of 1 mg of glucagon is brief—its onset is 8 to 10 minutes, and its action lasts 12 to 27 minutes) and to replenish liver stores of glucose. Some patients experience nausea after the administration of glucagon. If this occurs, the patient should be turned to the side to prevent aspiration in case the patient vomits.

Glucagon is sold by prescription only and should be part of the emergency supplies available to patients with diabetes who require insulin. Family members, caregivers, and coworkers should be instructed in the use of glucagon, especially for patients who have little or no warning of hypoglycemic episodes (ADA, 2020). Patients should be instructed to notify their primary provider after severe hypoglycemia has occurred and been treated. Close monitoring for 24 hours following a hypoglycemic episode is indicated because the patient is at increased risk of another episode (ADA, 2020).

In hospitals and emergency departments, for patients who are unconscious or cannot swallow, 25 to 50 mL of dextrose 50% in water ($D_{50}W$) may be administered IV. The effect is usually seen within minutes. The patient may complain of a headache and of pain at the injection site. Ensuring patency of the IV line used for injection of 50% dextrose is essential because hypertonic solutions such as 50% dextrose are very irritating to veins.



Providing Patient Education

Hypoglycemia is prevented by a consistent pattern of eating, administering insulin, and exercising. Between-meal and bedtime snacks may be needed to counteract the maximum insulin effect. In general, the patient should cover the time of peak activity of insulin by eating a snack and by taking additional food when physical activity is increased. Routine blood glucose tests are performed so that changing insulin requirements may be anticipated and the dosage adjusted. Because unexpected hypoglycemia can occur, all patients treated with insulin should wear an identification bracelet or tag stating that they have diabetes.

Patients, family members, and coworkers must be instructed to recognize the symptoms of hypoglycemia. Family members in particular must be made aware that any subtle (but unusual) change in behavior may be an indication of hypoglycemia. They should be taught to encourage and even insist that the person with diabetes assess blood glucose levels if hypoglycemia is suspected. Some patients become very resistant to testing or eating and become angry with family members who are trying to treat the hypoglycemia. Family members must be taught to persevere and to understand that the hypoglycemia can cause irrational behavior, due to low supply of glucose to the brain.

Autonomic neuropathy or beta-blockers such as propranolol to treat hypertension or cardiac arrhythmias may mask the typical symptoms of hypoglycemia. It is very important that patients taking these medications perform blood glucose tests on a frequent and regular basis. Patients who have type 2 diabetes and who take oral sulfonylurea agents may also develop hypoglycemia, which can be prolonged and severe; this is a particular risk for older adult patients.

It is important that patients with diabetes, especially those receiving insulin, learn to carry some form of simple sugar with them at all times (ADA, 2020; Davidson et al., 2018). There are commercially prepared

glucose tablets and gels that the patient may find convenient to carry. If the patient has a hypoglycemic reaction and does not have any of the recommended emergency foods available, any available food (preferably a carbohydrate food) should be eaten.

Patients are advised to refrain from eating high-calorie, high-fat dessert foods (e.g., cookies, cakes, doughnuts, ice cream) to treat hypoglycemia because their high-fat content may slow the absorption of the glucose and resolution of the hypoglycemic symptoms. The patient may subsequently eat more of the foods when symptoms do not resolve rapidly, which may cause very high blood glucose levels for several hours and may contribute to weight gain.

Patients who feel unduly restricted by their meal plan may view hypoglycemic episodes as a time to reward themselves with desserts. Instructing these patients to incorporate occasional desserts into the meal plan may be more effective, because this may make it easier for them to limit their treatment of hypoglycemic episodes to simple (low-calorie) carbohydrates such as juice or glucose tablets. Patients should be instructed to report all severe hypoglycemic episodes in addition to any increase in the incidence, frequency, and severity to the primary provider.



Diabetic Ketoacidosis

DKA is caused by an absence or markedly inadequate amount of insulin. This deficit in available insulin results in disorders in the metabolism of carbohydrate, protein, and fat. The three main clinical features of DKA are as follows:

- Hyperglycemia
- Dehydration and electrolyte loss
- Acidosis

Pathophysiology

Without insulin, the amount of glucose entering the cells is reduced, and gluconeogenesis (the production and release of glucose by the liver) is increased, leading to hyperglycemia (see Fig. 46-6). In an attempt to rid the body of the excess glucose, the kidneys excrete the glucose along with water and electrolytes (e.g., sodium, potassium). This osmotic diuresis, which is characterized by polyuria, leads to dehydration and marked electrolyte loss (Norris, 2019). Patients with severe DKA may lose up to 6.5 L of water and up to 400 to 500 mEq each of sodium, potassium, and chloride over a 24-hour period.

Another effect of insulin deficiency or deficit is lipolysis, the breakdown of fat into free fatty acids and glycerol. The free fatty acids are converted into ketone bodies by the liver. Ketone bodies are acids; their accumulation in the circulation due to lack of insulin leads to metabolic acidosis.

Three main causes of DKA are decreased or missed dose of insulin, illness or infection, and undiagnosed and untreated diabetes (DKA may be the initial manifestation of type 1 diabetes). An insulin deficiency may result from an insufficient dosage of insulin prescribed or from insufficient insulin being given by the patient. Errors in insulin dosage may be made by patients who are ill and who assume that if they are eating less or if they are vomiting, they must decrease their insulin doses. (Because illness, especially infections, can cause increased blood glucose levels, the patient does not need to decrease the insulin dose to compensate for decreased food intake when ill and may even need to increase the insulin dose.)

Physiology/Pathophysiology

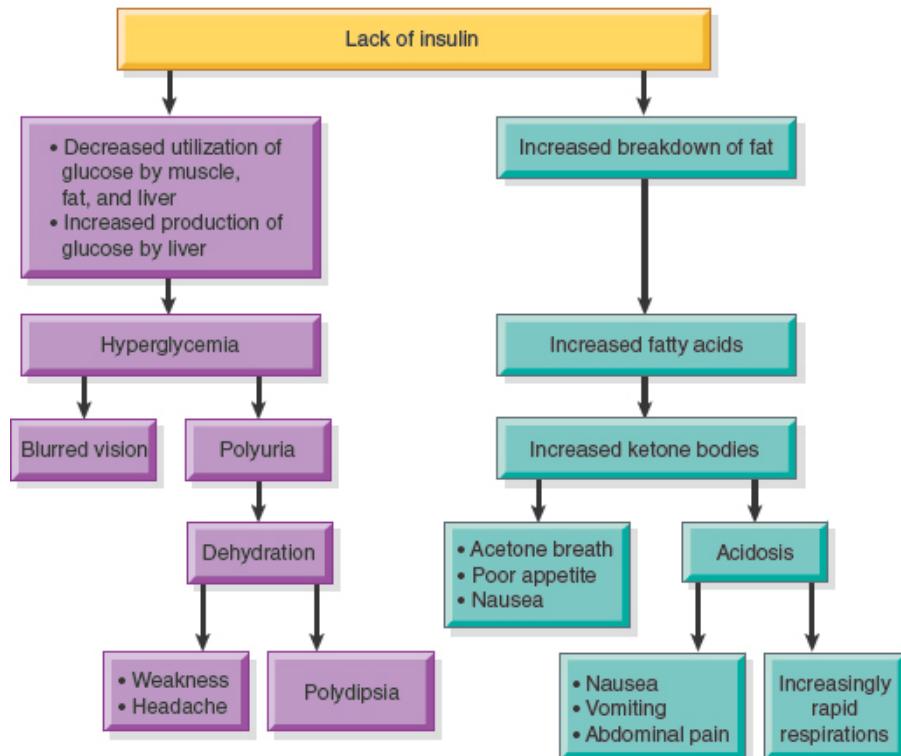


Figure 46-6 • Abnormal metabolism causes signs and symptoms of diabetic ketoacidosis. Redrawn from Pearce, M. A., Rosenberg, C. S., & Davidson, M. D. (2003). Patient education. In Davidson, M. B. (Ed.). *Diabetes mellitus: Diagnosis and treatment*. New York: Churchill Livingstone.

Other potential causes of decreased insulin include patient error in drawing up or injecting insulin (especially in patients with visual impairments), intentional skipping of insulin doses (especially in adolescents with diabetes who are having difficulty coping with diabetes or other aspects of their lives), or equipment problems (e.g., occlusion of insulin pump tubing). Illness and infections are associated with insulin resistance. In response to physical (and emotional) stressors, there is an increase in the level of "stress" hormones—glucagon, epinephrine, norepinephrine, cortisol, and growth hormone. These hormones promote glucose production by the liver and interfere with glucose utilization by muscle and fat tissue, counteracting the effect of insulin. If insulin levels are not increased during times of illness and infection, hyperglycemia may progress to DKA (ADA, 2020).

Prevention

For prevention of DKA related to illness, “sick day rules” for managing diabetes when ill (see [Chart 46-9](#)) should be reviewed with patients. The most important concept in this is to never eliminate insulin doses when nausea and vomiting occur. Instead, the patient should take the usual insulin dose (or previously prescribed special sick day doses) and then attempt to consume frequent small portions of carbohydrates (including foods usually avoided, such as juices, regular sodas, and gelatin). Drinking fluids every hour is important to prevent dehydration. Blood glucose and urine ketones must be assessed every 3 to 4 hours.

If the patient cannot take fluids without vomiting, or if elevated glucose or ketone levels persist, the provider must be contacted. Patients are taught to have foods available for use on sick days (Down, 2018). In addition, a supply of urine test strips (for ketone testing) and blood glucose test strips should be available. The patient must know how to contact their primary provider 24 hours a day. These materials should be assembled in a “sick day” kit.

Chart 46-9



PATIENT EDUCATION

Guidelines to Follow During Periods of Illness (“Sick Day Rules”)

The nurse instructs the patient to:

- Take insulin or oral antidiabetic agents as usual.
- Test blood glucose and urine ketones every 3 to 4 hours.
- Report elevated glucose levels as specified or urine ketones to your primary provider.
- Take supplemental doses of regular insulin every 3 to 4 hours, if needed, if you take insulin.
- Substitute soft foods (e.g., 1/3 cup regular gelatin, 1 cup cream soup, ½ cup custard, 3 squares graham crackers) six to eight times a day if you cannot follow your usual meal plan.
- Take liquids (e.g., ½ cup regular cola or orange juice, ½ cup broth, 1 cup sports drink [Gatorade]) every ½ to 1 hour to prevent dehydration and to provide calories, if vomiting, diarrhea, or fever persists.
- Report nausea, vomiting, and diarrhea to your primary provider, because extreme fluid loss may be dangerous.
- Be aware that if you are unable to retain oral fluids, you may require hospitalization to avoid diabetic ketoacidosis and possibly coma.

After the acute phase of DKA has resolved, the nurse should assess for underlying causes. If there are psychological reasons for the patient missing

insulin doses, the patient and family may be referred for evaluation and counseling or therapy.

Clinical Manifestations

The hyperglycemia of DKA leads to polyuria, polydipsia, and marked fatigue. In addition, the patient may experience blurred vision, weakness, and headache. Patients with marked intravascular volume depletion may have orthostatic hypotension (drop in systolic blood pressure of 20 mm Hg or more on changing from a reclining to a standing position). Volume depletion may also lead to frank hypotension with a weak, rapid pulse.

The ketosis and acidosis of DKA lead to gastrointestinal symptoms, such as anorexia, nausea, vomiting, and abdominal pain. The patient may have acetone breath (a fruity odor), which occurs with elevated ketone levels. In addition, hyperventilation (with very deep, but not labored, respirations) may occur. These Kussmaul respirations represent the body's attempt to decrease the acidosis, counteracting the effect of the ketone buildup (Norris, 2019). In addition, mental status in DKA varies widely. The patient may be alert, lethargic, or comatose.

Assessment and Diagnostic Findings

Blood glucose levels may vary between 250 and 800 mg/dL (16.6 and 44.4 mmol/L). Some patients have lower glucose values, and others have values of 1000 mg/dL (55.5 mmol/L) or higher (usually depending on the degree of dehydration). The severity of DKA is not necessarily related to the blood glucose level. Evidence of ketoacidosis is reflected in low serum bicarbonate (0 to 15 mEq/L) and low pH (6.8 to 7.3) values. A low partial pressure of carbon dioxide (PaCO_2 10 to 30 mm Hg) reflects respiratory compensation (Kussmaul respirations) for the metabolic acidosis. Accumulation of ketone bodies (which precipitates the acidosis) is reflected in blood and urine ketone measurements (Down, 2018).

Sodium and potassium concentrations may be low, normal, or high, depending on the amount of dehydration present. Despite the plasma concentration, there has been a marked total body depletion of these (and other) electrolytes, and they will need to be replaced. Increased levels of creatinine, blood urea nitrogen (BUN), and hematocrit may also be seen with dehydration. After rehydration, continued elevation in the serum creatinine and BUN levels suggests underlying renal insufficiency.

Management

In addition to treating hyperglycemia, management of DKA is aimed at correcting dehydration, electrolyte loss, and acidosis before correcting the hyperglycemia with insulin (Fayfman et al., 2017; Joyner Blair, Hamilton, & Spurlock, 2018).

Rehydration

In dehydrated patients, rehydration is important for maintaining tissue perfusion. In addition, fluid replacement enhances the excretion of excessive glucose by the kidneys. The patient may need as much as 6 to 10 L of IV fluid to replace fluid losses caused by polyuria, hyperventilation, diarrhea, and vomiting.

Initially, 0.9% sodium chloride (normal saline [NS]) solution is given at a rapid rate, usually 0.5 to 1 L per hour for the first 2 to 4 hours (Fayfman et al., 2017). Half-strength NS (0.45%) solution (also known as hypotonic saline solution) may be used for patients with hypertension or hypernatremia and those at risk for heart failure. After the first few hours, half-strength NS solution is the fluid of choice for continued rehydration, provided the blood pressure is stable and the sodium level is not low. Moderate to high rates of infusion (200 to 500 mL per hour) may be needed for several more hours. When the blood glucose level reaches 300 mg/dL (16.6 mmol/L) or less, the IV solution may be changed to dextrose 5% in water (D_5W) to prevent a precipitous decline in the blood glucose level (Fayfman et al., 2017).

Monitoring of fluid volume status involves frequent measurements of vital signs (including monitoring for orthostatic changes in blood pressure and heart rate), lung assessment, and monitoring of intake and output. Initial urine output lags behind IV fluid intake as dehydration is corrected. Plasma expanders may be necessary to correct severe hypotension that does not respond to IV fluid treatment. Monitoring for signs of fluid overload is especially important for patients who are older, have renal impairment, or are at risk for heart failure.

Restoring Electrolytes

The major electrolyte of concern during treatment of DKA is potassium. The initial plasma concentration of potassium may be low, normal, or high, but more often than not, tends to be high (hyperkalemia) from disruption of the cellular sodium-potassium pump (in the face of acidosis). Therefore, the serum potassium level must be monitored frequently. Some of the factors related to treating DKA that affect potassium concentration include rehydration, which leads to increased plasma volume and subsequent decreases in the concentration of serum potassium. Rehydration also leads to increased urinary excretion of potassium. Insulin administration enhances the movement of potassium from the extracellular fluid into the cells.

Cautious but timely potassium replacement is vital to avoid arrhythmias that may occur with hypokalemia. As much as 40 mEq/h may be needed for several hours. Because extracellular potassium levels decrease during DKA treatment, potassium must be infused even if the plasma potassium level is normal.

Frequent (every 2 to 4 hours initially) ECGs and laboratory measurements of potassium are necessary during the first 8 hours of treatment. Potassium replacement is withheld only if hyperkalemia is present or if the patient is not urinating.



Quality and Safety Nursing Alert

Because a patient's serum potassium level may drop quickly as a result of rehydration and insulin treatment, potassium replacement must begin once potassium levels drop to normal in the patient with DKA.

Reversing Acidosis

Ketone bodies (acids) accumulate as a result of fat breakdown. The acidosis that occurs in DKA is reversed with insulin, which inhibits fat breakdown, thereby ending ketone production and acid buildup. Insulin is usually infused IV at a slow, continuous rate (e.g., 5 units per hour). Hourly blood glucose values must be measured. IV fluid solutions with higher concentrations of glucose, such as NS solution (e.g., D₅NS, D₅ 0.45% NS), are given when blood glucose levels reach 250 to 300 mg/dL (13.9 to 16.6 mmol/L) to avoid too rapid a drop in the blood glucose level (i.e., hypoglycemia) during treatment.

Regular insulin, the only type of insulin approved for IV use, may be added to IV solutions. The nurse must convert hourly rates of insulin infusion (frequently prescribed as units per hour) to IV drip rates. For example, if 100 units of regular insulin are mixed into 500 mL of 0.9% NS, then 1 unit of insulin equals 5 mL; therefore, an initial insulin infusion rate of 5 units per hour would equal 25 mL per hour. The insulin is often infused separately from the rehydration solutions to allow frequent changes in the rate and content of the latter (Fayfman et al., 2017).

Insulin must be infused continuously until subcutaneous administration of insulin can be resumed. Any interruption in administration may result in the reaccumulation of ketone bodies and worsening acidosis. Even if blood glucose levels are decreasing and returning to normal, the insulin drip must not be stopped until subcutaneous insulin therapy has been started. Rather, the rate or concentration of the dextrose infusion may be increased to prevent hypoglycemia. Blood glucose levels are usually corrected before the acidosis

is corrected. Therefore, IV insulin may be continued for 12 to 24 hours, until the serum bicarbonate level increases (to at least 15 to 18 mEq/L) and until the patient can eat. In general, bicarbonate infusion to correct severe acidosis is avoided during treatment of DKA because it precipitates further, sudden (and potentially fatal) decreases in serum potassium levels. Continuous insulin infusion is usually sufficient for reversal of DKA (Down, 2018; Fayfman et al., 2017).



Quality and Safety Nursing Alert

When hanging the insulin drip, the nurse must flush the insulin solution through the entire IV infusion set and discard the first 50 mL of fluid. Insulin molecules adhere to the inner surface of plastic IV infusion sets; therefore, the initial fluid may contain a decreased concentration of insulin.



Hyperglycemic Hyperosmolar Syndrome

HHS is a metabolic disorder most often of type 2 diabetes resulting from a relative insulin deficiency initiated by an illness that raises the demand for insulin. This is a serious condition in which hyperosmolality and hyperglycemia predominate, with alterations of the sensorium (sense of awareness). At the same time, ketosis is usually minimal or absent. The basic biochemical defect is the lack of effective insulin (i.e., insulin resistance). Persistent hyperglycemia causes osmotic diuresis, which results in losses of water and electrolytes. To maintain osmotic equilibrium, water shifts from the intracellular fluid space to the extracellular fluid space. With glycosuria and dehydration, hypernatremia and increased osmolarity occur. [Table 46-6](#) compares DKA and HHS.

HHS occurs most often in older adults (50 to 70 years of age) who have no known history of diabetes or who have type 2 diabetes (Fayfman et al., 2017). HHS often can be traced to an infection or a precipitating event such as an acute illness (e.g., stroke), medications that exacerbate hyperglycemia (e.g., thiazides), or treatments such as dialysis. The history includes days to weeks of polyuria with adequate fluid intake. What distinguishes HHS from DKA is that ketosis and acidosis generally do not occur in HHS, partly because of differences in insulin levels. In DKA, no insulin is present, and this promotes the breakdown of stored glucose, protein, and fat, which leads to the production of ketone bodies and ketoacidosis. In HHS, the insulin level is too low to prevent hyperglycemia (and subsequent osmotic diuresis), but it is high enough to prevent fat breakdown. Patients with HHS do not have the ketosis-

related gastrointestinal symptoms that lead them to seek medical attention. Instead, they may tolerate polyuria and polydipsia until neurologic changes or an underlying illness (or family members or others) prompts them to seek treatment.

TABLE 46-6 Comparison of Diabetic Ketoacidosis and Hyperglycemic Hyperosmolar Syndrome

Characteristics	DKA	HHS
Patients most commonly affected	Can occur in type 1 or type 2 diabetes; more common in type 1 diabetes	Can occur in type 1 or type 2 diabetes; more common in type 2 diabetes, especially older adults with type 2 diabetes
Precipitating event	Omission of insulin; physiologic stress (infection, surgery, stroke, MI, untreated type 1 diabetes)	Physiologic stress (infection, surgery, stroke, MI), medications (e.g., thiazides), treatments (e.g., dialysis)
Onset	Rapid (<24 h)	Slower (over several days)
Blood glucose levels	Usually >250 mg/dL (>13.9 mmol/L)	Usually >600 mg/dL (>33.3 mmol/L)
Arterial pH level	<7.3	Normal
Serum and urine ketones	Present	Absent
Serum osmolality	275–320 mOsm/L	>320 mOsm/L
Plasma bicarbonate level	<15 mEq/L	Normal
BUN and creatinine levels	Elevated	Elevated
Mortality rate	<1%	5–16%

BUN, blood urea nitrogen; DKA, diabetic ketoacidosis; HHS, hyperglycemic hyperosmolar syndrome; MI, myocardial infarction.

Adapted from Fayfman, M., Pasquel, F. J., & Umpeirrez, G. E. (2017). Management of hyperglycemic crises: Diabetic ketoacidosis and hyperglycemic hyperosmolar state. *Medical Clinics of North America*, 101(3), 587–606.

Clinical Manifestations

The clinical picture of HHS is one of hypotension, profound dehydration (dry mucous membranes, poor skin turgor), tachycardia, and variable neurologic signs (e.g., alteration of consciousness, seizures, hemiparesis) (Down, 2018; Fayfman et al., 2017) (see Table 46-6).

Assessment and Diagnostic Findings

Diagnostic assessment includes a range of laboratory tests, including blood glucose, electrolytes, BUN, complete blood count, serum osmolality, and arterial blood gas analysis. The blood glucose level is greater than 600 mg/dL, the osmolality exceeds 320 mOsm/kg, and ketoacidosis is absent (Fayfman et al., 2017). Electrolyte and BUN levels are consistent with the clinical picture of severe dehydration (see [Chapter 10](#)). Mental status changes, focal neurologic deficits, and hallucinations are common secondary to the cerebral dehydration that results from extreme hyperosmolality. Orthostatic hypotension accompanies the dehydration (Fayfman et al., 2017).

Management

The overall approach to the treatment of HHS is similar to that of DKA: fluid replacement, correction of electrolyte imbalances, and insulin administration. Because patients with HHS are typically older, close monitoring of volume and electrolyte status is important for prevention of fluid overload, heart failure, and cardiac arrhythmias. Fluid treatment is started with 0.9% or 0.45% NS, depending on the patient's sodium level and the severity of volume depletion. Central venous or hemodynamic pressure monitoring guides fluid replacement. Potassium is added to IV fluids when urinary output is adequate and is guided by continuous ECG monitoring and frequent laboratory determinations of potassium (Fayfman et al., 2017).

Extremely elevated blood glucose concentrations decrease as the patient is rehydrated. Insulin plays a less important role in the treatment of HHS because it is not needed for reversal of acidosis, as in DKA. Nevertheless, insulin is usually given at a continuous low rate to treat hyperglycemia, and replacement IV fluids with dextrose are given (as in DKA) after the glucose level has decreased to the range of 250 to 300 mg/dL (13.8 to 16.6 mmol/L) (Fayfman et al., 2017).

Other therapeutic modalities are determined by the underlying illness and the results of continuing clinical and laboratory evaluation. It may take 3 to 5 days for neurologic symptoms to clear, and treatment of HHS usually continues well after metabolic abnormalities have resolved. After recovery from HHS, many patients can control their diabetes with MNT alone or with MNT and oral antidiabetic medications. Insulin may not be needed once the acute hyperglycemic complication is resolved. Frequent SBGM is important in prevention of recurrence of HHS (Fayfman et al., 2017).

NURSING PROCESS

The Patient with Diabetic Ketoacidosis or Hyperglycemic Hyperosmolar Syndrome

Assessment

For the patient with DKA, the nurse monitors the ECG for arrhythmias indicating abnormal potassium levels. Vital signs (especially blood pressure and pulse), arterial blood gases, breath sounds, and mental status are assessed every hour and recorded on a flow sheet. Neurologic status checks are included as part of the hourly assessment because cerebral edema can be a severe and sometimes fatal outcome. Blood glucose levels are checked every hour (Fayfman et al., 2017).

For the patient with HHS, the nurse assesses vital signs, fluid status, and laboratory values. Fluid status and urine output are closely monitored because of the high risk of kidney failure secondary to severe dehydration. Because HHS tends to occur in older patients, the physiologic changes that occur with aging should be considered. Careful assessment of cardiovascular, pulmonary, and kidney function throughout the acute and recovery phases of HHS is important (Fayfman et al., 2017).

Diagnosis

NURSING DIAGNOSES

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

- Risk for hypovolemia associated with polyuria and dehydration
- Fluid imbalance associated with fluid loss or shifts
- Lack of knowledge about diabetes self-care skills or information
- Anxiety associated with loss of control, fear of inability to manage diabetes, misinformation associated with diabetes, fear of diabetes complications

COLLABORATIVE PROBLEMS/POTENTIAL COMPLICATIONS

Potential complications may include the following:

- Fluid overload, pulmonary edema, and heart failure
- Hypokalemia
- Hyperglycemia and ketoacidosis
- Hypoglycemia
- Cerebral edema

Planning and Goals

The major goals for the patient may include maintenance of fluid and electrolyte balance, increased knowledge about diabetes basic skills and self-care, decreased anxiety, and absence of complications.

Nursing Interventions

MAINTAINING FLUID AND ELECTROLYTE BALANCE

Intake and output are measured. IV fluids and electrolytes are given as prescribed, and oral fluid intake is encouraged when it is permitted. Laboratory values of serum electrolytes (especially sodium and potassium) are monitored. Vital signs are monitored hourly for signs of dehydration (tachycardia, orthostatic hypotension) along with assessment of breath sounds, level of consciousness, presence of edema, and cardiac status (ECG rhythm strips).

INCREASING KNOWLEDGE ABOUT DIABETES MANAGEMENT

The development of DKA or HHS suggests the need for the nurse to carefully assess the patient's understanding of and adherence to the diabetes management plan. Furthermore, factors that may have led to the development of DKA or HHS are explored with the patient and family. If the patient's blood glucose monitoring, dietary intake, use of antidiabetic medications (insulin or oral agents), and exercise patterns differ from those identified in the diabetes management plan, their relationship to the development of DKA or HHS is discussed, along with early manifestations of DKA or HHS. If other factors, such as trauma, illness, surgery, or stress, are implicated, appropriate strategies to respond to these and similar situations in the future are described so that the patient can respond in the future without developing life-threatening complications. The nurse may need to provide education about basic skills again to patients who may not be able to recall the instructions. If the patient has omitted insulin or oral antidiabetic agents that have been prescribed, the nurse explores the reasons for doing so and addresses those issues to prevent future recurrence and readmissions for treatment of these complications.

If the patient has not previously been diagnosed with diabetes, the opportunity is used to educate the patient about the need for maintaining blood glucose at a normal level and learning about diabetes management and basic skills.

DECREASING ANXIETY

Educating the patient about cognitive strategies may be useful for relieving tension, overcoming anxiety, decreasing fear, and achieving relaxation (see Chapter 3). Examples include:

- *Imagery*: The patient concentrates on a pleasant experience or restful scene.
- *Distraction*: The patient thinks of an enjoyable story or recites a favorite poem or song.
- *Optimistic self-recitation*: The patient recites optimistic thoughts ("I know all will go well").

- *Music:* The patient listens to soothing music (an easy-to-administer, inexpensive, noninvasive intervention).

MONITORING AND MANAGING POTENTIAL COMPLICATIONS

Fluid Overload. Fluid overload can occur because of the administration of a large volume of fluid at a rapid rate, which is often required to treat patients with DKA or HHS. This risk is increased in older patients and in those with preexisting cardiac or kidney disease. To avoid fluid overload and resulting heart failure and pulmonary edema, the nurse monitors the patient closely during treatment by measuring vital signs and intake and output at frequent intervals. Central venous pressure monitoring and hemodynamic monitoring may be initiated to provide additional measures of fluid status. Physical examination focuses on assessment of cardiac rate and rhythm, breath sounds, venous distention, skin turgor, and urine output. The nurse monitors fluid intake and keeps careful records of IV and other fluid intake, along with urine output measurements.

Hypokalemia. Hypokalemia is a potential complication during the treatment of DKA. Low serum potassium levels may result from rehydration, increased urinary excretion of potassium, movement of potassium from the extracellular fluid into the cells with insulin administration, and restoration of the cellular sodium–potassium pump. Prevention of hypokalemia includes cautious replacement of potassium; however, before its administration, it is important to ensure that a patient's kidneys are functioning. Because of the adverse effects of hypokalemia on cardiac function, monitoring of the cardiac rate, cardiac rhythm, ECG, and serum potassium levels is essential.

Cerebral Edema. Although the exact cause of cerebral edema is unknown, rapid correction of hyperglycemia, resulting in fluid shifts, is thought to be the cause. Cerebral edema, which occurs more often in children than in adults, can be prevented by a gradual reduction in the blood glucose level. An hourly flow sheet is used to enable close monitoring of the blood glucose level, serum electrolyte levels, fluid intake, urine output, mental status, and neurologic signs. Precautions are taken to minimize activities that could increase intracranial pressure.



EDUCATING PATIENTS ABOUT SELF-CARE

The patient is educated about basic skills, including treatment modalities (diet, insulin administration, monitoring of blood glucose, and, for type 1 diabetes, monitoring of urine ketones); the patient is also educated about recognition, treatment, and prevention of DKA and HHS (Down, 2018; Fayfman et al., 2017). Education addresses those factors leading to DKA or HHS. Follow-up education is arranged with a home health nurse and

dietitian or an outpatient diabetes education center. This is particularly important for patients who have experienced DKA or HHS because of the need to address factors that led to its occurrence (e.g., dehydration). For patients who have had HHS, avoiding dehydration and paying attention to increased urination or thirst are even more important than insulin administration. The importance of self-monitoring and of monitoring and follow-up by primary providers is reinforced, and the patient is reminded about the importance of keeping follow-up appointments.

Evaluation

Expected patient outcomes may include:

1. Achieves fluid and electrolyte balance
 - a. Demonstrates intake and output balance
 - b. Exhibits electrolyte values within normal limits
 - c. Exhibits vital signs that remain stable, with resolution of orthostatic hypotension and tachycardia
2. Demonstrates knowledge about DKA and HHS
 - a. Identifies factors leading to DKA and HHS
 - b. Describes signs and symptoms of DKA and HHS
 - c. Describes short- and long-term consequences of DKA and HHS
 - d. Identifies strategies to prevent the development of DKA and HHS
 - e. States when contact with primary provider is needed to treat early signs of DKA and HHS
3. Decreased anxiety
 - a. Identifies strategies to decrease anxiety and fear
4. Absence of complications
 - a. Exhibits normal cardiac rate and rhythm and normal breath sounds
 - b. Exhibits no jugular venous distention
 - c. Exhibits blood glucose and urine ketone levels within target range
 - d. Exhibits no manifestations of hypoglycemia or hyperglycemia
 - e. Shows improved mental status without signs of cerebral edema

Long-Term Complications of Diabetes



The number of deaths attributable to ketoacidosis and infection in patients with diabetes has steadily declined, but diabetes related complications have increased. Long-term complications are becoming more common as more people live longer with diabetes; these complications can affect almost every organ system of the body and are a major cause of disability. The general

categories of long-term diabetic complications are macrovascular disease, microvascular disease, and neuropathy.

The causes and pathogenesis of each type of complication are still being investigated. However, it appears that increased levels of blood glucose play a role in neuropathic disease, microvascular complications, and risk factors contributing to macrovascular complications. Hypertension may also be a major contributing factor, especially in macrovascular and microvascular diseases (ADA, 2020).

Long-term complications are seen in both type 1 and type 2 diabetes but usually do not occur within the first 5 to 10 years after diagnosis. However, evidence of these complications may be present at the time of diagnosis of type 2 diabetes, because patients may have had undiagnosed diabetes for many years. Kidney (microvascular) disease is more prevalent in patients with type 1 diabetes, and cardiovascular (macrovascular) complications are more prevalent in older patients with type 2 diabetes.

Macrovascular Complications

Diabetic macrovascular complications result from changes in the medium to large blood vessels. Blood vessel walls thicken, sclerose, and become occluded by plaque that adheres to the vessel walls. Eventually, blood flow is blocked. These atherosclerotic changes tend to occur more often and at an earlier age in patients with diabetes. Coronary artery disease, cerebrovascular disease, and peripheral vascular disease are the three main types of macrovascular complications that occur frequently in patients with diabetes.

Myocardial infarction (MI) is twice as common in men with diabetes and three times as common in women with diabetes, compared with people without diabetes. There is also an increased risk of complications resulting from MI and an increased likelihood of a second MI. Coronary artery disease accounts for an increased incidence of death among patients with diabetes. The typical ischemic symptoms may be absent in patients with diabetes. Therefore, the patient may not experience the early warning signs of decreased coronary blood flow and may have “silent” MIs, which may be discovered only as changes on the ECG. In some cases, ECG changes may not be apparent. This lack of ischemic symptoms may be secondary to autonomic neuropathy (see later discussion). See [Chapter 23](#) for a detailed discussion of coronary vascular disorders.

Cerebral blood vessels are similarly affected by accelerated atherosclerosis. Occlusive changes or the formation of an embolus elsewhere in the vasculature that lodges in a cerebral blood vessel can lead to transient ischemic attacks and strokes. People with diabetes have twice the risk of developing cerebrovascular disease and an increased risk of death from stroke (Virani et

al., 2020). In addition, recovery from a stroke may be impaired in patients who have elevated blood glucose levels at the time of and immediately after a stroke. Because symptoms of a stroke may be similar to symptoms of acute diabetic complications (HHS or hypoglycemia), it is very important to assess the blood glucose level (and treat abnormal levels) rapidly in patients with these symptoms so that testing and treatment of a stroke can be initiated promptly if indicated.

Atherosclerotic changes in the large blood vessels of the lower extremities are responsible for the increased incidence (two to three times higher than in people without diabetes) of occlusive peripheral arterial disease in patients with diabetes (ADA, 2020). Signs and symptoms of peripheral vascular disease include diminished peripheral pulses and intermittent claudication (pain in the buttock, thigh, or calf during walking). The severe form of arterial occlusive disease in the lower extremities is largely responsible for the increased incidence of gangrene and subsequent amputation in patients with diabetes. Neuropathy and impairments in wound healing also play a role in diabetic foot disease (see later discussion).

Role of Diabetes in Macrovascular Diseases

Researchers continue to investigate the relationship between diabetes and macrovascular diseases. The main feature unique to diabetes is elevated blood glucose; however, a direct link has not been found between hyperglycemia and atherosclerosis. Although it may be tempting to attribute the increased prevalence of macrovascular diseases to the increased prevalence of certain risk factors (e.g., obesity, increased triglyceride levels, hypertension) in patients with diabetes, there is a higher-than-expected rate of macrovascular diseases among patients with diabetes compared with patients without diabetes who have the same risk factors (ADA, 2020). Therefore, diabetes itself is seen as an independent risk factor for accelerated atherosclerosis. Other potential factors that may play a role in diabetes-related atherosclerosis include platelet and clotting factor abnormalities, decreased flexibility of red blood cells, decreased oxygen release, changes in the arterial wall related to hyperglycemia, and possibly hyperinsulinemia.

Management

The focus of management is an aggressive modification and reduction of risk factors. This involves prevention and treatment of the commonly accepted risk factors for atherosclerosis. MNT and exercise are important in managing obesity, hypertension, and hyperlipidemia. In addition, the use of medications to control hypertension and hyperlipidemia is indicated. Smoking cessation is essential. Control of blood glucose levels may reduce triglyceride

concentrations and can significantly reduce the incidence of complications (ADA, 2020; Evert et al., 2019).

Microvascular Complications

Diabetic microvascular disease (or microangiopathy) is characterized by capillary basement membrane thickening. The basement membrane surrounds the endothelial cells of the capillary. Researchers believe that increased blood glucose levels react through a series of biochemical responses to thicken the basement membrane to several times its normal thickness. Two areas affected by these changes are the retina and the kidneys (Norris, 2019).

Diabetic Retinopathy

Diabetic retinopathy is the leading cause of blindness among people between 20 and 74 years of age in the United States; it occurs in both type 1 and type 2 diabetes (ADA, 2020).

People with diabetes are subject to many visual complications (see [Table 46-7](#)). The pathology referred to as diabetic retinopathy is caused by changes in the small blood vessels in the retina, which is the area of the eye that receives images and sends information about the images to the brain (see Fig. 46-7). The retina is richly supplied with blood vessels of all kinds: small arteries and veins, arterioles, venules, and capillaries. Retinopathy has three main stages: nonproliferative (background), preproliferative, and proliferative.

Almost all patients with type 1 diabetes and the majority of patients with type 2 diabetes have some degree of retinopathy after 20 years (ADA, 2020). Changes in the microvasculature include microaneurysms, intraretinal hemorrhage, hard exudates, and focal capillary closure. Although most patients do not develop visual impairment, it can be devastating if it occurs. A complication of nonproliferative retinopathy—macular edema—occurs in approximately 10% of people with type 1 or type 2 diabetes and may lead to visual distortion and loss of central vision (ADA, 2020).

TABLE 46-7 Ocular Complications of Diabetes

Eye Disorder	Characteristics
Retinopathy	Damage to the small blood vessels that nourish the retina.
Background	Early-stage, asymptomatic retinopathy. Blood vessels within the retina develop microaneurysms that leak fluid, causing swelling and forming exudates (deposits). In some cases, macular edema causes distorted vision.
Preproliferative	Represents increased destruction of retinal blood vessels.
Proliferative	Abnormal growth of new blood vessels on the retina. New vessels rupture, bleeding into the vitreous and blocking light. Ruptured blood vessels in the vitreous form scar tissue, which can pull on and detach the retina.
Cataracts	Opacity of the lens of the eye; cataracts occur at an earlier age in patients with diabetes.
Lens Changes	The lens of the eye can swell when blood glucose levels are elevated. For some patients, visual changes related to lens swelling may be the first symptoms of diabetes. It may take up to 2 mo of improved blood glucose control before hyperglycemic swelling subsides and vision stabilizes. Therefore, patients are advised not to change eyeglass prescriptions during the 2 mo after discovery of hyperglycemia.
Extraocular Muscle Palsy	This may occur as a result of diabetic neuropathy. The involvement of various cranial nerves responsible for ocular movements may lead to double vision. This usually resolves spontaneously.
Glucoma	Results from occlusion of the outflow channels by new blood vessels. Glaucoma may occur with slightly higher frequency among patients with diabetes.

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

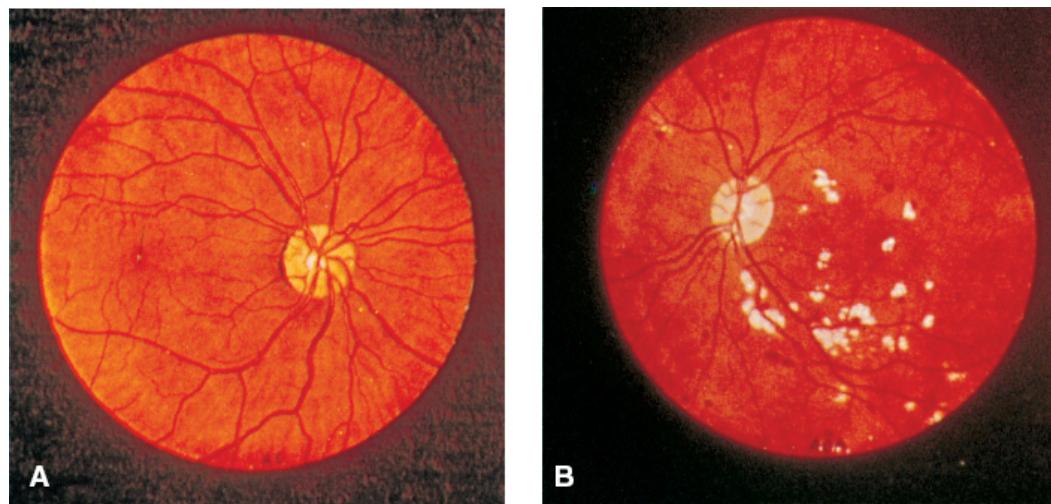


Figure 46-7 • Diabetic retinopathy. **A.** In the fundus photograph of a normal eye, the light circular area over which a number of blood vessels converge is the optic disc, where the optic nerve meets the back of the eye. **B.** The fundus photograph of a patient with diabetic retinopathy shows characteristic waxy-looking retinal lesions, microaneurysms of the vessels, and hemorrhages. Courtesy of American Optometric Association.

An advanced form of background retinopathy—preproliferative retinopathy—is considered to be a precursor to the more serious proliferative retinopathy. In preproliferative retinopathy, there are more widespread vascular changes and loss of nerve fibers. Epidemiologic evidence suggests that 10% to 50% of patients with preproliferative retinopathy will develop proliferative retinopathy within a short time (possibly as little as 1 year). As with background retinopathy, if visual changes occur during the preproliferative stage, they are usually caused by macular edema.

Proliferative retinopathy represents the greatest threat to vision and is characterized by the proliferation of new blood vessels growing from the retina into the vitreous. These new vessels are prone to bleeding. The visual loss associated with proliferative retinopathy is caused by this vitreous hemorrhage, retinal detachment, or both. The vitreous is normally clear, allowing light to be transmitted to the retina. When there is a hemorrhage, the vitreous becomes clouded and cannot transmit light, resulting in loss of vision. Another consequence of vitreous hemorrhage is that resorption of the blood in the vitreous leads to the formation of fibrous scar tissue. This scar tissue may place traction on the retina, resulting in retinal detachment and subsequent visual loss.

Clinical Manifestations

Retinopathy is a painless process. In non- and preproliferative retinopathy, blurry vision secondary to macular edema occurs in some patients, although many patients are asymptomatic. Even patients with a significant degree of proliferative retinopathy and some hemorrhaging may not experience major visual changes. However, symptoms indicative of hemorrhaging include floaters or cobwebs in the visual field, sudden visual changes including spotty or hazy vision, or complete loss of vision.

Assessment and Diagnostic Findings

Diagnosis is by direct visualization of the retina through dilated pupils with an ophthalmoscope or with a technique known as fluorescein angiography. Fluorescein angiography can document the type and activity of the retinopathy. Dye is injected into an arm vein and is carried to various parts of the body through the blood, but especially through the vessels of the retina of the eye. This technique allows an ophthalmologist, using special instruments, to see the retinal vessels in bright detail and gives useful information that cannot be obtained with just an ophthalmoscope.

Side effects of this diagnostic procedure may include nausea during the dye injection; yellowish, fluorescent discoloration of the skin and urine lasting 12 to 24 hours; and occasionally allergic reactions, usually manifested by hives or itching. However, the diagnostic procedure is generally safe.

Medical Management

The first focus of management of retinopathy is on primary and secondary prevention. The DCCT study (1993) demonstrated that in patients without preexisting retinopathy, maintenance of blood glucose to a normal or near-normal level in type 1 diabetes through intensive insulin therapy and patient education decreased the risk of retinopathy by 76%, compared with conventional therapy. The progression of retinopathy was decreased by 54% in patients with very mild to moderate nonproliferative retinopathy at the time of initiation of treatment. Additional research demonstrated similar results in patients with type 2 diabetes (Action to Control Cardiovascular Risk in Diabetes Follow-On [ACCORDIAN] Eye Study Group, 2016). Thus, control of blood glucose levels in patients with both types of diabetes reduces the risk of retinopathy as well (ADA, 2020).

Other strategies that may slow the progression of diabetic retinopathy include control of hypertension, control of blood glucose, and cessation of smoking.

For advanced cases of diabetic retinopathy, the main treatment is argon laser photocoagulation. The laser treatment destroys leaking blood vessels and areas of neovascularization. For patients who are at increased risk for

hemorrhage, panretinal photocoagulation may significantly reduce the rate of progression to blindness. Panretinal photocoagulation involves the systematic application of multiple (more than 1000) laser burns throughout the retina (except in the macular region). This stops the widespread growth of new vessels and hemorrhaging of damaged vessels. The role of “mild” panretinal photocoagulation (with only one third to one half as many laser burns) in the early stages of proliferative retinopathy or in patients with preproliferative changes is being investigated. For patients with macular edema, focal photocoagulation is used to apply smaller laser burns to specific areas of microaneurysms in the macular region. This may reduce the rate of visual loss from macular edema (ADA, 2020).

Photocoagulation treatments are usually performed on an outpatient basis, and most patients can return to their usual activities by the next day. Limitations may be placed on activities involving weight bearing or bearing down. In most cases, the treatment does not cause intense pain, although patients may report varying degrees of discomfort such as a headache. Usually, anesthetic eye drop is all that is needed during the treatment. A few patients may experience slight visual loss, loss of peripheral vision, or impairments in adaptation to the dark. However, the risk of slight visual changes from the laser treatment itself is much less than the potential for loss of vision from progression of retinopathy.

A major hemorrhage into the vitreous may occur, with the vitreous fluid becoming mixed with blood, preventing light from passing through the eye; this can cause blindness. A vitrectomy is a surgical procedure in which vitreous humor filled with blood or fibrous tissue is removed with a special drill-like instrument and replaced with saline or another liquid. A vitrectomy is performed for patients who already have visual loss and in whom the vitreous hemorrhage has not cleared on its own after 6 months. The purpose is to restore useful vision; recovery to near-normal vision is not usually expected.

Nursing Management

Nursing management of patients with diabetic retinopathy or other eye disorders involves implementing the individual plan of care and providing patient education. Education focuses on prevention through regular ophthalmologic examinations, blood glucose control, and self-management of eye care regimens. The effectiveness of early diagnosis and prompt treatment is emphasized in educating the patient and family.

If vision loss occurs, nursing care must also address the patient’s adjustment to impaired vision and the use of adaptive devices for diabetes self-care as well as activities of daily living. See [Chapter 58](#) for discussion of nursing care for patients with low vision and blindness.

Promoting Home, Community-Based, and Transitional Care



Educating Patients About Self-Care

Because the course of the retinopathy may be long and stressful, patient education is essential. In educating and counseling patients, it is important to stress the following:

- Retinopathy may appear after many years of diabetes, and its appearance does not necessarily mean that the diabetes is on a downhill course.
- The odds for maintaining vision are in the patient's favor, especially with adequate control of glucose levels and blood pressure.
- Frequent eye examinations allow for the detection and prompt treatment of retinopathy.

A patient's response to vision loss depends on personality, self-concept, and coping mechanisms. Acceptance of blindness occurs in stages; some patients may learn to accept blindness in a rather short period, and others may never do so. An important issue in educating patients is that several complications of diabetes may occur simultaneously. For example, a patient who is blind due to diabetic retinopathy may also have peripheral neuropathy and may experience impairment of manual dexterity and tactile sensation, or kidney failure. This can be devastating to the patient and family. Psychological counseling may be warranted. To prevent further losses, glycemic control remains a priority.

Continuing and Transitional Care

The importance of careful diabetes management is emphasized as one means of slowing the progression of visual changes. The patient is reminded of the need to see an ophthalmologist regularly. If eye changes are progressive and unrelenting, the patient should be prepared for inevitable blindness. Therefore, consideration is given to making referrals for educating the patient in Braille and for training the patient with guide (i.e., service) dogs. Referral to state agencies should be made to ensure that the patient receives services for the blind. Family members are also taught how to assist the patient to remain as independent as possible despite decreasing visual acuity.

Referral for home care may be indicated for some patients, particularly those who live alone, those who are not coping well, and those who have other health problems or complications of diabetes that may interfere with their ability to perform self-care. During home visits, the nurse can assess the patient's home environment and their ability to manage diabetes despite visual impairments. See [Chapter 58](#) for a detailed discussion of medical management and nursing care for patients with visual disturbances.

Nephropathy

Nephropathy, or kidney disease secondary to diabetic microvascular changes in the kidney, is a common complication of diabetes (ADA, 2020). In the United States each year, people with diabetes account for almost 50% of new cases of ESKD, and about 25% of those require dialysis or transplantation. About 20% to 30% of people with type 1 or types 2 diabetes develop nephropathy, but fewer of those with type 2 diabetes progress to ESKD. Native American, Latino, African American, Asian American, and Pacific Island people with type 2 diabetes are at greater risk for ESKD than non-Latino Whites (ADA, 2020).

Patients with type 1 diabetes frequently show initial signs of kidney disease after 10 to 15 years, while patients with type 2 diabetes tend to develop kidney disease within 10 years after the diagnosis of diabetes. Many patients with type 2 diabetes have had diabetes for many years before the diabetes is diagnosed and treated. Therefore, they may have evidence of nephropathy at the time of diagnosis (ADA, 2020). If blood glucose levels are elevated consistently for a significant period of time, the kidney's filtration mechanism is stressed, allowing blood proteins to leak into the urine. As a result, the pressure in the blood vessels of the kidney increases. It is thought that this elevated pressure serves as the stimulus for the development of nephropathy. Various medications and diets are being tested to prevent these complications.

The DCCT (1993) results showed that intensive treatment for type 1 diabetes with a goal of achieving a glycolated hemoglobin level as close to the nondiabetic range as possible reduced the occurrence of early signs of nephropathy. Similarly, the United Kingdom Prospective Diabetes Study Group (UKPDS, 1998) demonstrated a reduced incidence of overt nephropathy in patients with type 2 diabetes who controlled their blood glucose levels.

Clinical Manifestations

Most of the signs and symptoms of kidney dysfunction in patients with diabetes are similar to those seen in patients without diabetes (see [Chapter 48](#)). In addition, as kidney failure progresses, the catabolism (breakdown) of both exogenous and endogenous insulin decreases, and frequent hypoglycemic episodes may result. Insulin needs change as a result of changes in the catabolism of insulin, changes in diet related to the treatment of nephropathy, and changes in insulin clearance that occur with decreased kidney function.

Assessment and Diagnostic Findings

Albumin is one of the most important blood proteins that leak into the urine. Although small amounts may leak undetected for years, its leakage into the

urine is among the earliest signs that can be detected. Clinical nephropathy eventually develops in more than 85% of people with microalbuminuria but in fewer than 5% of people without microalbuminuria. The urine should be checked annually for the presence of microalbumin. If the microalbuminuria exceeds 30 mg/24 hours on two consecutive random urine tests, a 24-hour urine sample should be obtained and tested. If results are positive, treatment is indicated (see later discussion).

In addition, tests for serum creatinine and BUN levels should be conducted annually. Diagnostic testing for cardiac or other systemic disorders may also be required with progression of other complications, and caution is indicated if contrast agents are used with these tests. Contrast agents and dyes used for some diagnostic tests may not be easily cleared by the damaged kidney, and the potential benefits of these diagnostic tests must be weighed against their potential risks.

Hypertension often develops in patients (with and without diabetes) who are in the early stages of kidney disease. However, hypertension is the most common complication in all people with diabetes (ADA, 2020). Therefore, this symptom may or may not be due to kidney disease; other diagnostic criteria must also be present.

Management

In addition to achieving and maintaining near-normal blood glucose levels, management for all patients with diabetes should include careful attention to the following:

- Control of hypertension (the use of angiotensin-converting enzyme [ACE] inhibitors, such as captopril), because control of hypertension may also decrease or delay the onset of early proteinuria
- Prevention or vigorous treatment of urinary tract infections
- Avoidance of nephrotoxic medications and contrast dye
- Adjustment of medications as kidney function changes
- Low-sodium diet
- Low-protein diet

If the patient has already developed microalbuminuria with levels that exceed 30 mg/24 hours on two consecutive tests, an ACE inhibitor should be prescribed. ACE inhibitors lower blood pressure and reduce microalbuminuria, thereby protecting the kidney. Alternatively, angiotensin receptor-blocking agents may be prescribed. This preventive strategy should be part of the standard of care for all people with diabetes (ADA, 2020). Carefully designed low-protein diets also appear to reverse early leakage of small amounts of protein from the kidney.

In chronic or ESKD, two types of treatment are available: dialysis (hemodialysis or peritoneal dialysis) and transplantation from a relative or a deceased donor. Hemodialysis for patients with diabetes is similar to that for patients without the disease (see [Chapter 48](#)). Because hemodialysis creates additional stress on patients with cardiovascular disease, it may not be appropriate for some patients.

Continuous ambulatory peritoneal dialysis is being used by patients with diabetes, mainly because of the independence it allows. In addition, insulin can be mixed into the dialysate, which may result in better blood glucose control and end the need for insulin injections. Some patients may require higher doses of insulin because the dialysate contains glucose. Major risks of peritoneal dialysis are infection and peritonitis. The mortality rate for patients with diabetes undergoing dialysis is higher than that for patients without diabetes undergoing dialysis and is closely related to the severity of cardiovascular problems.

Kidney disease is frequently accompanied by advancing retinopathy that may require laser treatments and surgery. Severe hypertension also worsens eye disease because of the additional stress it places on the blood vessels. Patients being treated with hemodialysis who require eye surgery may be changed to peritoneal dialysis and have their hypertension aggressively controlled for several weeks before surgery to prevent bleeding and damage to the retina. The rationale for this change is that hemodialysis requires anticoagulant agents that can increase the risk of bleeding after the surgery, and peritoneal dialysis minimizes pressure changes in the eyes.

In medical centers performing large numbers of transplantations, the chances are 75% to 80% that the transplanted kidney will continue to function in patients with diabetes for at least 5 years. Like the original kidneys, transplanted kidneys can eventually be damaged if blood glucose levels are consistently high after the transplantation. Therefore, monitoring blood glucose levels frequently and adjusting insulin levels in patients with diabetes are essential for long-term success of kidney transplantation. Optimal treatment is a simultaneous kidney and pancreatic transplantation (Aref et al., 2019).

Diabetic Neuropathies

Diabetic neuropathy refers to a group of diseases that affect all types of nerves, including peripheral (sensorimotor), autonomic, and spinal nerves. The disorders appear to be clinically diverse and depend on the location of the affected nerve cells. The prevalence increases with the age of the patient and the duration of the disease (National Institute of Diabetes and Digestive and Kidney Diseases [NIDDK], 2018).

The etiology of neuropathy may involve elevated blood glucose levels over a period of years. Control of blood glucose levels to normal or near-normal levels decreases the incidence of neuropathy. The pathogenesis of neuropathy may be attributed to either a vascular or metabolic mechanism or both. Capillary basement membrane thickening and capillary closure may be present. In addition, there may be demyelination of the nerves, which is thought to be related to hyperglycemia. Nerve conduction is disrupted when there are aberrations of the myelin sheaths.

The two most common types of diabetic neuropathy are sensorimotor polyneuropathy and autonomic neuropathy. Sensorimotor polyneuropathy is also called *peripheral neuropathy*. Cranial mononeuropathies—those affecting the oculomotor nerve—also occur in diabetes, especially in older adults.

Peripheral Neuropathy

Peripheral neuropathy most commonly affects the distal portions of the nerves, especially the nerves of the lower extremities; it affects both sides of the body symmetrically and may spread in a proximal direction.

Clinical Manifestations

Although approximately half of patients with diabetic neuropathy do not have symptoms, initial symptoms may include paresthesias (prickling, tingling, or heightened sensation) and burning sensations (especially at night). As the neuropathy progresses, the feet become numb. In addition, a decrease in proprioception (awareness of posture and movement of the body and of position and weight of objects in relation to the body) and a decreased sensation of light touch may lead to an unsteady gait. Decreased sensations of pain and temperature place patients with neuropathy at increased risk for injury due to falls and undetected foot infections (Hickey & Strayer, 2020). Deformities of the foot may also occur; neuropathy-related joint changes are sometimes referred to as Charcot joints. These joint deformities result from the abnormal weight distribution on joints resulting from lack of proprioception.

On physical examination, a decrease in deep tendon reflexes and vibratory sensation is found. For patients with fewer or no symptoms of neuropathy, these physical findings may be the only indication of neuropathic changes. For patients with signs or symptoms of neuropathy, it is important to rule out other possible causes, including alcohol-induced and vitamin-deficiency neuropathies.

Management

Intensive insulin therapy and control of blood glucose levels delay the onset and slow the progression of neuropathy. Pain, particularly of the lower extremities, is a disturbing symptom in some people with neuropathy secondary to diabetes. In some cases, neuropathic pain spontaneously resolves within 6 months; for others, pain persists for many years. Various approaches to pain management can be tried. These include analgesic agents (preferably nonopioid); tricyclic antidepressants and other antidepressant medications (duloxetine); anticonvulsant medications (pregabalin or gabapentin); mexiletine, an antiarrhythmic agent; and transcutaneous electrical nerve stimulation (Hickey & Strayer, 2020).

Autonomic Neuropathies

Neuropathy of the autonomic nervous system results in a broad range of dysfunctions affecting almost every organ system of the body (NIDDK, 2018).

Clinical Manifestations

Three manifestations of autonomic neuropathy are related to the cardiac, gastrointestinal, and renal systems. Cardiovascular symptoms range from a fixed, slightly tachycardic heart rate and orthostatic hypotension to silent, or painless, myocardial ischemia and infarction. Research suggests that cardiovascular health may also be impacted by diabetes distress and depressive symptoms (McCarthy, Whittemore, Gholson, et al., 2019). See the Nursing Research Profile in [Chart 46-10](#).

Delayed gastric emptying may occur with the typical gastrointestinal symptoms of early satiety, bloating, nausea, and vomiting. “Diabetic” constipation or diarrhea (especially nocturnal diarrhea) may occur as a result. In addition, there may be unexplained wide swings in blood glucose levels related to inconsistent absorption of the glucose from ingested foods secondary to the inconsistent gastric emptying.

Urinary retention, a decreased sensation of bladder fullness, and other urinary symptoms of neurogenic bladder result from autonomic neuropathy. The patient with a neurogenic bladder is predisposed to development of urinary tract infections because of the inability to empty the bladder completely. This is especially true of patients with poorly controlled diabetes because hyperglycemia impairs resistance to infection.

Hypoglycemic Unawareness

Autonomic neuropathy affecting the adrenal medulla is responsible for diminished or absent adrenergic symptoms of hypoglycemia. Patients may report that they no longer feel the typical shakiness, sweating, nervousness, and palpitations associated with hypoglycemia. Frequent blood glucose

monitoring is recommended for these patients. The inability to detect and treat these warning signs of hypoglycemia puts patients at risk for development of dangerously low blood glucose levels. Therefore, goals for blood glucose levels may need to be adjusted to reduce the risk for hypoglycemia. Patients and families need to be taught to recognize subtle and atypical symptoms of hypoglycemia, such as numbness around the mouth and impaired ability to concentrate.

Sudomotor Neuropathy

The neuropathic condition called *sudomotor neuropathy* refers to a decrease or absence of anhidrosis (sweating) of the extremities, with a compensatory increase in upper body anhidrosis. Dryness of the feet increases the risk for the development of foot ulcerations.

Sexual Dysfunction

Sexual dysfunction, especially erectile dysfunction in men, is a complication of diabetes. The effects of autonomic neuropathy on female sexual functioning are not well documented. Reduced vaginal lubrication has been mentioned as a possible neuropathic effect. Other possible changes in sexual function in women with diabetes include decreased libido and lack of orgasm. Vaginal infection, which increases in the incidence in women with diabetes, may be associated with decreased lubrication and vaginal pruritus (itching) and tenderness. Urinary tract infections and vaginitis may also affect sexual function.

Chart 46-10



NURSING RESEARCH PROFILE

Health Factors in Adults with Type 1 Diabetes

McCarthy, M. M., Whittemore, R., Gholson, G., et al. (2019). Diabetes distress, depressive symptoms, and cardiovascular health in adults with type 1 diabetes. *Nursing Research*, 68(6), 445–452.

Purpose

The prevalence of type 1 diabetes in adults is increasing. The purpose of this study was to describe the relationships among the psychological factors of depressive symptoms and diabetes distress and six cardiovascular risk factors.

Design

This was a cross-sectional survey of a sample of 83 adults with type 1 diabetes. Data were collected about sociodemographic information, depressive symptoms, diabetes distress, and cardiovascular health factors (body mass index [BMI], blood pressure, cholesterol, smoking, hemoglobin A1C, and physical activity).

Findings

The mean age of participants was 45 years, with a mean duration of 20 years with type 1 diabetes. The majority had low scores on the Diabetes Distress Scale while 18% had moderate, and another 18% had high scores. Twenty-two percent of participants had an increased level of depressive symptoms. Significant correlations were found between diabetes distress and fear of hypoglycemia ($r = .65, p < .0001$), depressive symptoms ($r = .55, p < .0001$), hemoglobin A1C ($r = .41, p < .001$), and total cholesterol ($r = .26, p < .05$). There were significant correlations between depressive symptom scores and fear of hypoglycemia ($r = .35, p < .01$), as well as hemoglobin A1C ($r = .25, p < .05$). There were small to medium nonsignificant correlations between depressive symptoms scores and weekly step counts and BMI.

Nursing Implications

Nurses working with patients with type 1 diabetes need to be aware that elevated diabetes distress and depressive symptoms may further affect these patients' already high risk of cardiovascular disease. Interventions are needed that affect both physical and psychological factors that have the potential to adversely affect cardiovascular health.

Erectile dysfunction occurs with greater frequency in men with diabetes than in other men of the same age. Some men with autonomic neuropathy have normal erectile function and can experience orgasm but do not ejaculate normally. Retrograde ejaculation occurs; seminal fluid is propelled backward through the posterior urethra and into the urinary bladder. Examination of the

urine confirms the diagnosis because of the large number of active sperm present. Fertility counseling may be necessary for couples attempting conception.

Diabetic neuropathy is not the only cause of erectile dysfunction in men with diabetes. Medications such as antihypertensive agents, psychological factors, and other medical conditions (e.g., vascular insufficiency) that may affect other men also play a role in erectile dysfunction in men with diabetes (see [Chapter 53](#)).

Management

Management strategies for autonomic neuropathy focus on alleviating symptoms and on modification and management of risk factors. Detection of painless cardiac ischemia is important so that education about avoiding strenuous exercise can be provided. Orthostatic hypotension may respond to a diet high in sodium, discontinuation of medications that impede autonomic nervous system responses, the use of sympathomimetic and other agents (e.g., caffeine) that stimulate an autonomic response, mineralocorticoid therapy, and the use of lower-body elastic garments that maximize venous return and prevent pooling of blood in the extremities.

Treatment of delayed gastric emptying includes a low-fat diet, frequent small meals, frequent blood glucose monitoring, and the use of agents that increase gastric motility (e.g., metoclopramide, bethanechol). Treatment of diabetic diarrhea may include bulk-forming laxatives or antidiarrheal agents. Constipation is treated with a high-fiber diet and adequate hydration; medications, laxatives, and enemas may be necessary if constipation is severe. Management of sexual dysfunction in women and men is discussed in Chapters 51 and 53, respectively. Intermittent straight catheterization may be necessary to prevent urinary tract infections in patients with neurogenic bladders.

Treatment of sudomotor dysfunction focuses on education about skin care and heat intolerance.

Foot and Leg Problems

Lower limb amputations in adults with diabetes increased by 50% between 2009 and 2015 compared to prior years (Virani et al., 2020). Amputations are preventable, provided patients are taught appropriate foot care measures and practice them on a daily basis (ADA, 2020). Complications of diabetes that contribute to the increased risk of foot problems and infections include the following:

- ***Neuropathy:*** Sensory neuropathy leads to loss of pain and pressure sensation, and autonomic neuropathy leads to increased dryness and fissuring of the skin (secondary to decreased sweating). Motor neuropathy results in muscular atrophy, which may lead to changes in the shape of the foot.
- ***Peripheral vascular disease:*** Poor circulation of the lower extremities contributes to poor wound healing and the development of gangrene.
- ***Immunocompromise:*** Hyperglycemia impairs the ability of specialized leukocytes to destroy bacteria. Therefore, in poorly controlled diabetes, there is a lowered resistance to certain infections.



Figure 46-8 • Neuropathic ulceration occurs on pressure points in areas with diminished sensation in diabetic polyneuropathy. Because pain is absent, the ulceration may go unnoticed.

The typical sequence of events in the development of a diabetic foot ulceration begins with a soft tissue injury of the foot, formation of a fissure between the toes or in an area of dry skin, or formation of a callus (see Fig. 46-8). Patients with an insensitive foot do not feel injuries, which may be thermal (e.g., from using heating pads, walking barefoot on hot concrete, testing bathwater with the foot), chemical (e.g., burning the foot while using caustic agents on calluses, corns, or bunions), or traumatic (e.g., injuring skin while cutting nails, walking with an undetected foreign object in the shoe, or wearing ill-fitting shoes and socks).

If the patient is not in the habit of thoroughly inspecting both feet on a daily basis, the injury or fissure may go unnoticed until a serious infection has developed. Drainage, swelling, redness of the leg (from cellulitis), or gangrene may be the first sign of foot problems that the patient notices. Treatment of foot ulcerations involves keeping the patient off their feet, antibiotics, and débridement. In addition, controlling glucose levels, which tend to increase when infections occur, is important for promoting wound healing. When peripheral vascular disease is present, foot ulcerations may not heal because of the decreased ability of oxygen, nutrients, and antibiotics to reach the injured tissue. Amputation (see [Chapter 37](#)) may be necessary to prevent the spread of infection, particularly if it involves the bone (osteomyelitis) (see [Chapter 36](#)).

Foot assessment and foot care instructions are most important when caring for patients who are at high risk for foot infections (Johnson, Osbourne, Rispoli, et al., 2018). Some of the high-risk characteristics include:

- Duration of diabetes more than 5 years
- Age greater than 40 years
- Current smoker and history of smoking
- Decreased peripheral pulses
- Decreased sensation
- Anatomic deformities or pressure areas (e.g., bunions, calluses, hammer toes)
- History of previous foot ulcerations or amputation



Figure 46-9 • The monofilament test is used to assess the sensory threshold in patients with diabetes. The test instrument—a monofilament—is gently applied to about five pressure points on the foot (as shown in image on *left*). **A.** Example of a monofilament used for advanced quantitative assessment. **B.** Semmes-Weinstein monofilament used by clinicians. **C.** Disposable monofilament used by patients. The examiner applies the monofilament to the test area to determine whether the patient feels the device. Adapted with permission from Cameron, B. L. (2002). Making diabetes management routine. *American Journal of Nursing*, 102(2), 26–32.

Medical Management

The feet of a patient with diabetes should be examined during every health care visit or at least once per year (more often if there is an increase in risk) by a podiatrist, physician, or nurse (ADA, 2020; Johnson et al., 2018). All patients should be assessed for neuropathy and undergo evaluation of neurologic status by an experienced examiner using a monofilament device (ADA, 2020; Johnson et al., 2018) (see [Fig. 46-9](#)). Pressure areas, such as calluses, or thick toenails should be treated by a podiatrist in addition to routine trimming of nails. Blood glucose control is essential for avoiding decreased resistance to infections and for preventing diabetic neuropathy.

Nursing Management

The nurse facilitates or conducts a foot assessment at each visit to a health care provider (Johnson et al., 2018). Educating patients about proper foot care is an essential nursing intervention that can prevent costly and painful complications that result in disability (see [Chart 46-11](#)).

SPECIAL ISSUES IN DIABETES CARE

Patients with Diabetes Who Are Undergoing Surgery

During periods of physiologic stress, such as surgery, blood glucose levels tend to increase, because levels of stress hormones (epinephrine, norepinephrine, glucagon, cortisol, and growth hormone) increase. If hyperglycemia is not controlled during surgery, the resulting osmotic diuresis may lead to excessive loss of fluids and electrolytes. Patients with type 1 diabetes also risk developing DKA during periods of stress.

Hypoglycemia is also a concern in patients with diabetes who are undergoing surgery. This is a special concern during the preoperative period if surgery is delayed beyond the morning in a patient who received a morning injection of intermediate-acting insulin.

There are various approaches to managing glucose control during the perioperative period. Frequent blood glucose monitoring is essential throughout the pre- and postoperative periods, regardless of the method used for glucose control. Examples of these approaches are described in [Chart 46-12](#). The use of IV insulin and dextrose has become widespread with the increased availability of intraoperative glucose monitoring.

During the postoperative period, patients with diabetes must also be closely monitored for cardiovascular complications because of the increased prevalence of atherosclerosis, wound infections, and skin breakdown (especially in patients with decreased sensation in the extremities due to

neuropathy). Maintaining adequate nutrition and blood glucose control promote wound healing.

Management of Patients with Diabetes Who Are Hospitalized

At any one time, as many as 25% of hospitalized general medical-surgical patients have diabetes (ADA, 2020). Often, diabetes is not the primary medical diagnosis, yet problems with control of diabetes frequently result from changes in the patient's normal routine or from surgery or illness. Patients who are hospitalized and have a diagnosis of diabetes should have this clearly indicated on their electronic health record (EHR), and glucose monitoring needs to be prescribed (ADA, 2020). During the course of treatment, blood glucose control may worsen. Control of blood glucose levels is important because hyperglycemia in patients who are hospitalized can increase the length of hospital stay, the risk of infection, and mortality (ADA, 2020).

In addition, this is an opportunity for patients with diabetes to update their knowledge about diabetes self-care and prevention of complications. Nurses caring for patients with diabetes should focus attention on the diabetes as well as the primary health issue.

Chart 46-11



PATIENT EDUCATION

Foot Care Tips

The nurse instructs the patient to:

Take care of your diabetes.

- Work with your health care team to keep your blood glucose level within a normal range.

Inspect your feet every day.

- Look at your bare feet every day for cuts, blisters, red spots, and swelling.
- Use a mirror to check the bottoms of your feet, or ask a family member for help if you have trouble seeing.
- Check for changes in temperature.

Wash your feet every day.

- Wash your feet in warm, not hot, water.
- Dry your feet well. Be sure to dry between the toes.
- Do not soak your feet.
- Do not check water temperature with your feet; use a thermometer or elbow.

Keep the skin soft and smooth.

- Rub a thin coat of skin lotion over the tops and bottoms of your feet, but not between your toes.

Smooth corns and calluses gently.

- Use a pumice stone to smooth corns and calluses.
- Do not shave calluses.
- See a podiatrist as needed.

Trim your toenails each week or when needed.

- Trim your toenails straight across, and file the edges with an emery board or nail file.

Wear shoes and socks at all times.

- Never walk barefoot.
- Wear comfortable shoes that fit well and protect your feet.
- Feel inside your shoes before putting them on each time to make sure that the lining is smooth and there are no objects inside.
- Be aware that a podiatrist can provide inserts (orthotics) to remove pressure from pressure points on the foot.
- Break in new shoes slowly (i.e., wear for 1 to 2 hours initially, with gradual increases in the length of time worn) to avoid blister formation.
- If you have bony deformities, custom-made shoes with extra width or depth may be needed.

Protect your feet from hot and cold.

- Wear shoes at the beach or on hot pavement.
- Wear socks at night if your feet get cold.

Keep the blood flowing to your feet.

- Put your feet up when sitting.
- Wiggle your toes and move your ankles up and down for 5 minutes, two or three times a day.
- Do not cross your legs for long periods of time.
- Do not smoke.

Check with your primary provider.

- Have your primary provider check your bare feet and find out whether you are likely to have serious foot problems. Remember that you may not feel the pain of an injury.
- Call your primary provider right away if a cut, sore, blister, or bruise on your foot does not begin to heal after 1 day.
- Follow your primary provider's advice about foot care.
- Do not self-medicate or use home remedies or over-the-counter agents to treat foot problems.

Adapted from Johnson, R., Osbourne, A., Rispoli, J., et al. (2018). The diabetic foot assessment. *Orthopaedic Nursing*, 37(1) 13–21.

Chart 46-12

Approaches to Management of Glucose Control During the Perioperative Period for Those with a Diagnosis of Diabetes

- Monitor blood glucose levels frequently (every 1 to 2 hours).
- For patients taking insulin:
 1. The morning of surgery, all subcutaneous insulin doses are withheld, unless the blood glucose level is elevated (e.g., >200 mg/dL [11.1 mmol/L]), in which case a small dose of subcutaneous regular insulin may be prescribed. The blood glucose level is controlled during surgery with the IV infusion of regular insulin, which is balanced by an infusion of dextrose. The insulin and dextrose infusion rates are adjusted according to frequent (hourly) capillary glucose determinations. After surgery, the insulin infusion may be continued until the patient can eat. If IV insulin is discontinued, subcutaneous regular insulin may be given at set intervals (every 4 to 6 hours), or intermediate-acting insulin may be given every 12 hours with supplemental regular insulin as necessary until the patient is eating and the usual pattern of insulin dosing is resumed.
 - Carefully monitor the insulin infusion rate and blood glucose levels in a patient with diabetes who is receiving IV insulin. IV insulin has a much shorter duration of action than subcutaneous insulin. If the infusion is interrupted or discontinued, hyperglycemia will develop rapidly (within 1 hour in type 1 diabetes and within a few hours in type 2 diabetes).
 - Ensure that subcutaneous insulin is given 30 minutes before the IV insulin infusion is discontinued.
 2. One half to two thirds of the patient's usual morning dose of insulin (either intermediate-acting insulin alone or both short- and intermediate-acting insulins) is given subcutaneously in the morning before surgery. The remainder is then given after surgery.
 3. The patient's usual daily dose of subcutaneous insulin is divided into 4 equal doses of regular insulin. These are then given at 6-hour intervals. The last 2 approaches do not provide the control achieved by IV administration of insulin and dextrose.
 - Patients with type 2 diabetes who do not usually take insulin may require insulin during the perioperative period to control blood glucose elevations. Patients who are taking metformin may be instructed to discontinue the oral agent 24 to 48 hours before surgery, if possible. Some of these patients may resume their usual regimen of diet and oral agent during the recovery period. Other patients (whose diabetes is probably not well controlled with diet and an oral antidiabetic agent before surgery) need to continue with insulin injections after discharge.
 - For patients with type 2 diabetes who are undergoing minor surgery but who do not normally take insulin, glucose levels may remain stable provided no dextrose is infused during the surgery. After surgery, these patients may require small doses

of regular insulin until the usual diet and oral agent are resumed.

Adapted from Comerford, K. C., & Durkin M. T. (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer.

Self-Care Issues

For patients who are actively involved in diabetes self-management (especially insulin dose adjustment), relinquishing control over meal timing, insulin timing, and insulin dosage can be particularly difficult and anxiety provoking. The patient may fear hypoglycemia and express much concern over possible delays in receiving attention from the nurse if hypoglycemic symptoms occur or may disagree with a planned dose of insulin.

The nurse acknowledges the patient's concerns and involves the patient in the plan of care as much as possible. If the patient disagrees with certain aspects of the care related to diabetes, the nurse must communicate this to other members of the health care team. Nurses and other health care providers must pay particular attention to patients who are successful in managing self-care; they should assess these patients' self-care management skills and encourage them to continue if their performance is correct and effective.

Hospitalization of a patient with diabetes should be considered an opportunity to evaluate the patient's self-care skills and to reinforce or deliver education that might be needed. The nurse observes the patient preparing and injecting the insulin, monitoring blood glucose, and performing foot care. Simply questioning the patient about these skills without actually observing performance of the skills is not sufficient. The patient's knowledge about diet can be assessed with the help of a dietitian through direct questioning and review of the patient's menu choices. The patient's understanding about signs and symptoms, treatment, and prevention of hypoglycemia and hyperglycemia is assessed, along with knowledge of risk factors for macrovascular disease, including hypertension, increased lipids, and smoking. In addition, the patient is asked the date of their last eye examination (including dilation of the pupils). Education about these issues is critical.

Hyperglycemia During Hospitalization

Hyperglycemia may occur in patients who are hospitalized as a result of the original illness that led to the need for hospitalization. A number of other factors may contribute to hyperglycemia; examples include:

- Changes in the usual treatment regimen (e.g., increased food, decreased insulin, decreased activity)

- Medications (e.g., corticosteroids such as prednisone, which are used in the treatment of a variety of inflammatory disorders)
- IV dextrose, which may be part of the maintenance fluids or may be used for the administration of antibiotics and other medications, without adequate insulin therapy
- Overly vigorous treatment of hypoglycemia
- Inappropriate withholding of insulin or inappropriate use of “sliding scales”
- Mismatched timing of meals and insulin (e.g., postmeal hyperglycemia may occur if short-acting insulin is given immediately before or even after a meal)

Nursing actions to correct some of these factors are important for avoiding hyperglycemia. Assessment of the patient’s usual home routine is important. The nurse should try to approximate as much as possible the home schedule of insulin, meals, and activities. Monitoring blood glucose levels has been identified by the ADA as an additional “vital sign” essential in assessment of patients (ADA, 2020). The results of blood glucose monitoring provide information needed to obtain orders for extra doses of insulin (at times when insulin is usually taken), which is an important nursing function. Insulin doses must not be withheld when blood glucose levels are normal. It is very important to test blood glucose before a meal and administer insulin at that time, not on a rigid set time schedule as other medications are given. Insulin should be given when the meal is served to prevent hypoglycemia and elicit a physiologic response.

Short-acting insulin is usually needed to avoid postprandial hyperglycemia (even in patients with normal premeal glucose levels), and NPH insulin does not peak until many hours after the dose is given. IV antibiotics should be mixed in NS (if possible) to avoid excess infusion of dextrose (especially in patients who are eating). It is important to avoid overly vigorous treatment of hypoglycemia, which may lead to hyperglycemia.

Hypoglycemia During Hospitalization

Hypoglycemia in patients who are hospitalized is usually the result of too much insulin or delays in eating. Specific examples include:

- Overuse of sliding-scale regular insulin, particularly as a supplement to regularly scheduled, twice-daily short- and intermediate-acting insulins
- Lack of change in insulin dosage when dietary intake is changed (e.g., in the patient taking nothing by mouth [NPO])
- Overly vigorous treatment of hyperglycemia (e.g., giving too-frequent successive doses of regular insulin before the time of peak

- insulin activity is reached), resulting in a cumulative effect
- Delayed meal after administration of lispro, aspart, or glulisine insulin (patient should eat within 5 to 15 minutes after insulin administration)

Treatment of hypoglycemia should be based on the established hospital protocol (ADA, 2020). If the initial treatment does not increase the glucose level adequately, the same treatment may be repeated after 15 minutes. The nurse must assess the pattern of glucose values and avoid giving doses of insulin that repeatedly lead to hypoglycemia. Successive doses of subcutaneous regular insulin should be given no more frequently than every 3 to 4 hours. For patients receiving intermediate insulin before breakfast and dinner, the nurse must use caution in administering supplemental doses of regular insulin at lunch and bedtime. Hypoglycemia may occur when two insulins peak at similar times (e.g., morning NPH peaks with lunchtime regular insulin and may lead to late-afternoon hypoglycemia; dinnertime NPH peaks with bedtime regular insulin and may lead to nocturnal hypoglycemia). To avoid hypoglycemic reactions caused by delayed food intake, the nurse should arrange for snacks to be given to the patient if meals are going to be delayed because of procedures, physical therapy, or other activities.

Common Alterations in Diet

Dietary modifications commonly prescribed during hospitalization require special consideration for patients who have diabetes (ADA, 2020).

Nothing by Mouth

For patients who must be NPO in preparation for diagnostic or surgical procedures, the nurse must ensure that the usual insulin dosage has been changed. These changes may include eliminating the rapid-acting insulin and giving a decreased amount (e.g., half the usual dose) of intermediate-acting insulin. Another approach is to use frequent (every 3 to 4 hours) dosing of rapid-acting insulin only. IV dextrose may be given to provide calories and to avoid hypoglycemia.

Even without food, glucose levels may increase as a result of hepatic glucose production, especially in patients with type 1 diabetes and lean patients with type 2 diabetes. Furthermore, in type 1 diabetes, elimination of the insulin dose may lead to the development of DKA. Administration of basal insulin to patients with type 1 diabetes who are NPO is an important nursing action.

For patients with type 2 diabetes who are taking insulin, DKA does not usually develop when insulin doses are eliminated because the patient's pancreas produces some insulin. Therefore, skipping the insulin dose

altogether (when the patient is receiving IV dextrose) may be safe; however, close monitoring of blood glucose levels is essential.

For patients who are NPO for extended periods (24 hours), glucose testing and insulin administration should be performed at regular intervals, usually four times per day. Insulin regimens for the patient who is NPO for an extended period may include NPH insulin every 12 hours, rapid-acting insulin only every 4 to 6 hours, or an IV insulin drip. These patients should receive dextrose infusions to provide some calories and limit ketosis.

To prevent the problems that result from the need to withhold food, diagnostic tests and procedures and surgery should be scheduled early in the morning when possible.

Clear Liquid Diet

When the diet is advanced to include clear liquids, patients with diabetes receive more simple carbohydrate foods, such as juice and gelatin desserts, than are usually included in the diabetic diet. Because patients who are hospitalized should maintain their nutritional status as much as possible to promote healing, the use of reduced-calorie substitutes such as diet soda or diet gelatin desserts would not be appropriate when the only source of calories is clear liquids. Simple carbohydrates, if eaten alone, cause a rapid rise in blood glucose levels; therefore, it is important to try to match peak times of insulin effect with peaks in the blood glucose concentration. If the patient receives insulin at regular intervals while NPO, the scheduled times for glucose tests and insulin injections should match mealtimes.

Enteral Tube Feedings

Tube feeding formulas contain more simple carbohydrates and less protein and fat than the typical meal plan for diabetes (see [Chapter 39](#)). This results in increased levels of glucose in patients with diabetes who are receiving tube feedings. Insulin doses must be given at regular intervals (e.g., NPH every 12 hours or regular insulin every 4 to 6 hours) when continuous tube feedings are given. If insulin is administered at routine (prebreakfast and predinner) times, hypoglycemia during the day may result (because the patient receives more insulin without more calories); hyperglycemia may occur during the night if feedings continue but insulin action decreases.

A common cause of hypoglycemia in patients receiving both continuous tube feedings and insulin is inadvertent or purposeful discontinuation of the feeding. The nurse must discuss with the medical team any plans for temporarily discontinuing the tube feeding (e.g., when the patient is away from the unit). Planning ahead may allow for alterations to be made in the insulin dose or for administration of IV dextrose. In addition, if problems with the tube feeding develop unexpectedly (e.g., the patient pulls out the tube, the tube

clogs, the feeding is discontinued when residual gastric contents are found), the nurse must notify the primary provider, assess blood glucose levels more frequently, and administer IV dextrose if indicated.

Parenteral Nutrition

Patients receiving parenteral nutrition may receive both IV insulin (added to the parenteral nutrition IV bag) and subcutaneous intermediate- or short-acting insulins. If the patient is receiving continuous parenteral nutrition, the blood glucose level should be monitored and insulin given at regular intervals. If the parenteral nutrition is infused over a limited number of hours, subcutaneous insulin should be given so that peak times of insulin action coincide with times of parenteral nutrition infusion (see [Chapter 41](#) for discussion of parenteral nutrition).

Hygiene

Nurses caring for hospitalized patients with diabetes must focus attention on oral hygiene and skin care. Because these patients are at increased risk for periodontal disease, the nurse assists with at least daily dental care. The patient may also require assistance in keeping the skin clean and dry, especially in areas of contact between two skin surfaces (e.g., groin, axilla, under the breasts), where chafing and fungal infections tend to occur.

Careful assessments of the oral cavity and the skin are important. The skin is assessed for dryness, cracks, breakdown, and redness, especially at pressure points and on the lower extremities. The patient is asked about symptoms of neuropathy, such as tingling and pain or numbness of the feet. Deep tendon reflexes are assessed.

As with any patient confined to bed, nursing care must emphasize the prevention of skin breakdown at pressure points. The heels are particularly susceptible to breakdown because of loss of sensation of pain and pressure associated with sensory neuropathy.

Feet should be cleaned, dried, lubricated with lotion (but not between the toes), and inspected frequently. If the patient is in the supine position, pressure on the heels can be alleviated by elevating the lower legs on a pillow, with the heels positioned over the edge of the pillow. When the patient is seated in a chair, the feet should be positioned so that pressure is not placed on the heels. If the patient has an ulceration on one foot, the nurse provides preventive care to the unaffected foot as well as special care of the affected foot.

As always, every opportunity should be taken to educate the patient about diabetes self-management, including daily oral, skin, and foot care. Female patients should also be instructed about measures for the avoidance of vaginal infections, which occur more frequently when blood glucose levels are

elevated. Patients often take their cues from nurses and realize the importance of daily personal hygiene if this is emphasized during their hospitalization.

Stress

Physiologic stress, such as infections and surgery, contributes to hyperglycemia and may precipitate DKA or HHS. Emotional stress related to hospitalization for any reason can also have a negative impact on diabetic control. An increase in stress hormones leads to an increase in glucose levels, especially if intake of food and insulin remains unchanged. In addition, during periods of emotional stress, people with diabetes may alter their usual pattern of meals, exercise, and medication. This can contribute to hyperglycemia or even hypoglycemia (e.g., in the patient taking insulin or oral antidiabetic agents who stops eating in response to stress).

People with diabetes must be made aware of the potential deterioration in diabetic control that can accompany emotional stress. They must be encouraged to follow the diabetes treatment plan as much as possible during times of stress. In addition, learning strategies for minimizing stress and coping with stress when it does occur are important aspects of diabetes education.



Gerontologic Considerations

Because people with diabetes are living longer, both type 1 and type 2 diabetes are being seen more frequently in older patients hospitalized for various reasons. Regardless of the type or duration of diabetes, the goals of diabetes treatment may need to be altered when caring for older adults who are hospitalized. The focus is on quality-of-life issues, such as maintaining independent functioning and promoting general well-being.

Some of the barriers to learning and self-care during hospital stays and in preparing patients for discharge include decreased vision, hearing loss, memory deficits, decreased mobility and fine motor coordination, increased tremors, depression and isolation, decreased financial resources, and limitations related to disability and other medical disorders. Assessing these barriers is important in planning diabetes treatment and educational activities. Presenting brief, simplified instructions with ample opportunity for practice of skills is important. The use of special devices such as a magnifier for the insulin syringe, an insulin pen, or a mirror for foot inspection is helpful. Frequent evaluation of self-care skills (insulin administration, blood glucose monitoring, foot care, diet planning) is essential, especially in patients with deteriorating vision and memory. Providing written instructions with handouts to take home also assists with management in the home setting.

If appropriate, family members may be called on to assist with diabetes basic skills, and referral to community resources may be made. It is preferable to educate the patient or family members to test blood glucose at home; the choice of meter should be tailored to the patient's visual and cognitive status and dexterity.



Quality and Safety Nursing Alert

Careful monitoring for complications of diabetes in older adults is vital. Hypoglycemia is especially dangerous, because it may go undetected and result in falls. Dehydration is a concern in patients who have chronically elevated blood glucose levels. Assessment for long-term complications, especially eye and foot problems, is important. Avoiding blindness and amputation through early detection and treatment of retinopathy and foot ulcerations may mean the difference between placement in a long-term care facility and continued independent living for the older adult with diabetes.

Nursing Management

Monitoring and Managing Potential Complications

Assessment for hypo- and hyperglycemia involves frequent blood glucose monitoring (usually prescribed before meals and at bedtime) and monitoring for signs and symptoms of hypoglycemia or prolonged hyperglycemia (including DKA or HHS), as described previously. Inadequate control of blood glucose levels may hinder recovery from the primary health problem. Blood glucose levels are monitored, and insulin is given as prescribed. The nurse must ensure that prescribed insulin dosage is modified as needed to compensate for changes in the patient's schedule or eating pattern. Treatment is given for hypoglycemia (with oral glucose) or hyperglycemia (with supplemental regular insulin no more often than every 3 to 4 hours). Blood glucose records are assessed for patterns of hypo- and hyperglycemia at the same time of day, and findings are reported to the primary provider for modification in insulin orders. In the patient with prolonged elevations in blood glucose, laboratory values and the patient's physical condition are monitored for signs and symptoms of DKA or HHS.

Promoting Home, Community-Based, and Transitional Care



Educating Patients About Self-Care

Even if patients have had diabetes for many years, their knowledge and adherence to the plan of care must be assessed. A new plan of care may need to be devised using updated evidence. The nurse also reminds the patient and family about the importance of health promotion activities and recommended health screening.

Continuing and Transitional Care

A patient who is hospitalized may require referral for home, community-based or transitional care. The nurse can use this opportunity to assess the patient's knowledge about diabetes management and the patient's and family's ability to carry out that management. The nurse reinforces the education provided in the hospital, clinic, office, or diabetes education center and assesses the home care environment to determine its adequacy for self-care and safety.

CRITICAL THINKING EXERCISES

1 ipc An 18-year-old woman is newly diagnosed with type 1 diabetes in the clinic where you work. How will you educate this patient about this new diagnosis? Which referrals are appropriate to help this patient manage her type 1 diabetes? What steps will the interdisciplinary team take to address the patient's health care needs?

2 ebp A 45-year-old patient has had type 2 diabetes for 6 years. During his annual examination at the clinic where you work, a small ulceration is identified on the bottom of his foot. What is the evidence for management of a foot ulceration in this patient? What criteria would you use to assess the strength of the evidence? What is the evidence for the timing of future foot assessments?

3 pq A 65-year-old patient is admitted to the emergency department with possible HHS. Identify the pathophysiology and signs and symptoms of HHS. What are your priorities for the assessment, medical management, and nursing care for this patient with HHS? What are your priorities for discharge planning to prevent another episode?

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*Asterisk indicates nursing research.

**Double asterisk indicates classic reference.

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Resources

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- American Association of Diabetes Educators (AADE), www.diabeteseducator.org
- American Diabetes Association, www.diabetes.org
- American Foundation for the Blind (AFB), www.afb.org
- Go4Life, www.healthinaging.org/tools-and-tips/go4life-national-institute-aging
- JDRF (formerly the Juvenile Diabetes Research Foundation), www.jdrf.org
- MedicAlert Foundation, www.medicalert.org
- National Diabetes Information Clearinghouse, www.niddk.nih.gov
- National Library Services for the Blind and Physically Handicapped (NLS), www.loc.gov/nls/