13th Edition

ABRAMS' Clinical Drug Therapy

Rationales for Nursing Practice

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I dedicate this edition to my family, my husband Gary, our daughter Claire, son-in-law John, son Joseph, daughter-in-law Allyson, and grandson Elliott.

Geralyn Frandsen

To my family, my daughter Jennifer, and granddaughter Liliana, my constant source of strength, inspiration, hope, and gratitude.

Sandra Smith Pennington

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Preface

Abrams' Clinical Drug Therapy: Rationales for Nursing Practice has a long tradition of guiding students and instructors through the practice of safe and effective medication administration. The 13th edition includes a Clinical Judgment case study for each section to assist in preparing you for the Next Generation NCLEX Examination. Quality and Safety Alerts for patient-centered care, patient safety, and evidence-based practice have been included in each chapter. To allow a quick review, significant information on the etiology and pathophysiology of disease has been placed on thePoint®. Drug therapy pertinent to the prevention and treatment of disease is provided in each chapter.

GOALS AND RESPONSIBILITIES OF NURSING CARE RELATED TO DRUG THERAPY

Varied goals and responsibilities inherent in safe medication administration are identified in each chapter. The following information will guide you in developing your own goals and responsibilities inherent to safe and effective nursing practice.

- Preventing the need for drug therapy, when possible, by promoting health and preventing conditions that require drug therapy.
- Using nonpharmacologic interventions alone or in conjunction with drug therapy. When used with drugs, such interventions may promote lower drug dosage, less frequent administration, and fewer adverse effects.
- Administering drugs accurately and taking into consideration patient characteristics such as age, weight, and hepatic and kidney function, which can influence drug response.
- Preventing or minimizing adverse effects by knowing the major adverse effects associated with particular drugs. It is important to assess patients with impaired hepatic and kidney function closely for adverse effects. The early recognition of adverse effects allows for the implementation of interventions to minimize their severity. All drugs cause adverse effects, and nurses must maintain a high index of suspicion that the development of new signs and symptoms may be drug induced.
- Understanding the effect produced if medications are combined with other medications, herbs, or foods.
- Teaching patients and families about the effects of medications. The nurse must instruct patients and families about the role and importance of their medications in treating particular illnesses, accurate administration of medications, nonpharmacologic treatments to use with or instead of pharmacologic treatments, and when to contact their healthcare provider.

ORGANIZATIONAL FRAMEWORK

The 10 sections of the textbook provide the reader with basic information about drug therapy as well as the administration of medications for the prevention and treatment of disease. The first section introduces the safeguards in place to promote drug safety, the Institute of Medicine Core Competencies, and medication administration. It also describes the nursing process and explains the application of the nursing process in the care of patients receiving drug therapy. The use of concept maps addresses priority considerations and nursing actions related to drug therapy. The second section addresses the effect medications have throughout the lifespan. The text introduces the effects of drugs on infants, children, older adults, pregnant and lactating patients, and drugs affecting male and female health. The remaining sections provide information on drug therapy related to systems, infections, and disease processes.

Each chapter opens with a case study, and its use throughout the chapter helps readers integrate information about a particular disease and its drug therapy so they can apply it. The chapters also have NCLEX-style questions distributed throughout to test knowledge of the content and its application to patient care. This approach will help the reader prepare for class examinations as well as the NCLEX itself.

At the end of each section, a clinical judgment case study is presented. The clinical reasoning case studies are on thePoint® to increase your knowledge of patient care and related drug therapy.

The chapters that focus on drug treatment for specific diseases use the prototype approach, allowing the reader to see the similarity in medications within each broad drug classification. Introduction and Overview sections provide the basis for understanding the drug therapy that prevents or treats the disease. Drug therapy sections summarize the medications, identifying the pharmacokinetics, action, use, adverse effects, contraindications, and nursing implications—including administration, assessment, and patient teaching. Many chapters address the effect of herbal supplements on prescribed medications. This information has become crucial for the maintenance of patient safety. Boxes containing patient teaching guidelines for a drug or class of drugs highlight crucial information the nurse should teach the patient and family.

RECURRING FEATURES

This updated edition includes new and revised features to enhance learning.

Chapter-Opening Features

- **Learning Objectives** summarize what the student should learn while reading the chapter and answering both the Clinical Application Case Study Questions and NCLEX Success questions, described below.
- A **Clinical Application Case Study** opens each chapter with a patient-focused clinical scenario. Throughout the chapter, the reader is asked **critical thinking questions** to apply chapter content, emphasizing a patient-centered and interdisciplinary approach to pharmacology.
- **Key Terms** with definitions help the reader understand the chapter's content.

Special Features

- **Quality and Safety Alerts**, presented in the context of the chapter discussion, alert the reader to important considerations regarding patient safety, patient-centered care, and evidence-based practice that are pertinent to drug therapy. These considerations emphasize safety as a primary objective in patient care.
- **Boxed Warnings** highlight serious or life-threatening adverse effects identified by the FDA as being associated with a drug.
- **Drugs at a Glance Tables**—which include **Canadian drug names**—summarize the routes and dosage ranges (for adults and for children) for each drug in the class. The prototype drug is indicated with an icon.
- **Drug Interactions** boxes highlight the risk of interactions as well as increased or decreased drug effects when drugs are combined with other medications.
- **Specific herbal and dietary interactions** with medications are included in the chapter sections that discuss preventing drug interactions.
- **Patient Teaching Guidelines** list specific information for the education of the patient and family.
- **NCLEX Success** sections interspersed throughout the chapter ask the student to answer NCLEX-style questions that pertain to the learning objectives and the information just presented. This feature helps students check and apply their knowledge as they read and assists them to prepare for patient care and for the NCLEX. The questions align to the terminology used on the NCLEX. The NCLEX Success questions exclusively use generic names for medications, which is consistent with the RN licensure examination.
- **Nursing Process Concept Maps** provide a succinct overview of drug therapy in terms of assessment, planning/goals, nursing interventions, and evaluation. Located at the end of each chapter, the nursing process concept maps provide the guidelines for drug therapy specifically related to nursing care. (Nursing diagnoses do not appear in these concept maps because nursing diagnoses are not tested on the NCLEX.)
- **Unfolding Patient Stories**, written by the National League for Nursing, are an engaging way to begin meaningful conversations in the classroom. These vignettes, which appear in relevant chapters, feature patients from Wolters Kluwer's *vSim for Nursing | Pharmacology* (codeveloped with Laerdal Medical) and DocuCare products; however, each Unfolding Patient Story in the book stands alone, not requiring purchase of these products.
- **Concept Mastery Alerts** clarify common misconceptions as identified by Lippincott's Adaptive Learning Powered by PrepU.

Chapter-Ending Features

- **References and Resources** provide sources on which content is based and direction for further reading.

Content on thePoint®

- **Etiology and Pathophysiology** information for select diseases is available on thePoint®. This allows students to make connections between the etiology and pathophysiology and pharmacologic management of a disease.
- **Clinical Reasoning Case Studies** focus on the action of the medication prescribed and the associated assessment of medication outcomes. These case studies are found on thePoint®.
- **Key Concepts** available on thePoint® summarize the most salient content that appears in each chapter.

A COMPREHENSIVE PACKAGE FOR TEACHING AND LEARNING

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

Resources for Instructors

Tools to assist with teaching this text are available upon its adoption on thePoint® at http://thePoint.lww.com/Frandsen13e.

- The **Test Generator** lets you put together exclusive new tests from a bank containing more than 1,000 questions to help assess students' understanding of the material. Test questions are mapped to chapter learning objectives and page numbers.
- An extensive collection of materials is provided for each book chapter:
  - **PowerPoint Presentations** provide an easy way for you to integrate the textbook with your students' classroom experience, either via slide shows or handouts. Multiple-choice and true/false questions are integrated into the presentations to promote class participation and allow you to use i-clicker technology.
  - **Guided Lecture Notes** walk you through the chapters, objective by objective, and provide you with corresponding PowerPoint slide numbers.
  - **Discussion Topics** (and suggested answers) can be used as conversation starters or in online discussion boards.
  - **Assignments** (and suggested answers) include group, written, clinical, and Web assignments.
  - **Case Studies** with related questions (and suggested answers) give students an opportunity to apply their knowledge to a patient case similar to one they might encounter in practice.
  - **Learning Objectives** from the book.
- An **Image Bank** lets you use the photographs and illustrations from this textbook in your PowerPoint slides or as you see fit in your course.

Contact your sales representative or check out LWW.com/Nursing for more details and ordering information.

Resources for Students

An exciting set of free resources is available to help students review material and become even more familiar with vital concepts. Students can access all these resources on thePoint® at http://thePoint.lww.com/Frandsen13e, using the codes printed in the front of their textbooks.

- **Concepts in Action Animations** bring physiologic and pathophysiologic concepts to life and enhance student comprehension.

- **Watch & Learn Video Clips** demonstrate nursing skills and appeal to visual and auditory learners.
- The following **online appendices**:
  - Appendix A: Answers for NCLEX Success
  - Appendix B: Answers for the Clinical Application Case Studies
  - Appendix C: Critical Thinking Questions and Answers
  - Appendix D: The International System of Units
  - Appendix E: Serum Drug Concentrations
- **Journal Articles** for each book chapter offer access to current research available in Wolters Kluwer journals.
- Plus **Etiology and Pathophysiology information for select diseases, Clinical Reasoning Case Studies, Key Concepts**, and **Heart and Breath Sounds**.

VSIM FOR NURSING

vSim for Nursing, jointly developed by Laerdal Medical and Wolters Kluwer Health, offers innovative scenario-based learning modules consisting of web-based virtual simulations, course learning materials, and curriculum tools designed to develop critical thinking skills and promote clinical confidence and competence. vSim for Nursing | Pharmacology includes 10 cases from the Simulation in Nursing Education—Pharmacology Scenarios. Students can progress through suggested readings, pre- and postsimulation assessments, documentation assignments, and guided reflection questions, and will receive an individualized feedback log immediately upon completion of the simulation. Throughout the student learning experience, the product offers remediation back to trusted Lippincott resources, including Lippincott Nursing Advisor and Lippincott Nursing Procedures—two online, evidence-based, clinical information solutions used in healthcare facilities throughout the United States. This innovative product provides a comprehensive patient-focused solution for learning and integrating simulation into the classroom.

Contact your Wolters Kluwer sales representative or visit http://thepoint.lww.com/vsim for options to enhance your pharmacology nursing course with vSim for Nursing.

LIPPINCOTT DOCUCARE

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

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A COMPREHENSIVE, DIGITAL, INTEGRATED COURSE SOLUTION: *LIPPINCOTT® COURSEPOINT+*

The same trusted solution, innovation, and unmatched support that you have come to expect from *Lippincott CoursePoint+* is now enhanced with more engaging learning tools and deeper analytics to help prepare students for practice. This powerfully integrated digital learning solution combines learning tools, case studies, virtual simulation, real-time data, and the most trusted nursing education content on the market to make curriculum-wide learning more efficient and to meet students where they are at in their learning. And now, it is easier than ever for instructors and students to use, giving them everything they need for course and curriculum success!

Lippincott CoursePoint+ includes the following:

- Engaging course content provides a variety of learning tools to engage students of all learning styles.
- A more personalized learning approach, including adaptive learning powered by PrepU, gives students the content and tools they need at the moment they need it, giving them data for more focused remediation and helping to boost their confidence.
- Varying levels of case studies, virtual simulation, and access to Lippincott Advisor help students learn the critical thinking and clinical judgment skills to help them become practice-ready nurses.
- Unparalleled reporting provides in-depth dashboards with several data points to track student progress and help identify strengths and weaknesses.
- Unmatched support includes training coaches, product trainers, and nursing education consultants to help educators and students implement CoursePoint+ with ease.

Acknowledgments

The expertise of our contributors and reviewers is a great example of how the power of collaboration improves outcomes, and we thank them. We also wish to thank the team at Wolters Kluwer who provided their expertise to this edition.

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CHAPTER 45

Drug Therapy for Adrenal Cortex Disorders

LEARNING OBJECTIVES

After studying this chapter, you should be able to:

1. Understand the clinical considerations and manifestations of Addison disease.
2. Understand the clinical considerations and manifestations of Cushing disease.
3. Explain how corticotropin (ACTH) is used in the diagnosis of adrenocortical insufficiency.
4. Explain how cosyntropin (Cortrosyn) is used in the diagnosis of adrenocortical insufficiency.
5. Identify the prototypes and describe the action, use, adverse effects, contraindications, and nursing implications for the drugs used in the treatment of Addison disease.
6. Identify the prototypes and describe the action, use, adverse effects, contraindications, and nursing implications for the drugs used in the treatment of Cushing disease.
7. Implement the nursing process in the care of the patient with Addison disease or Cushing disease.

CLINICAL APPLICATION CASE STUDY

Rosa James is a 68-year-old woman who is being seen by her nurse practitioner with symptoms of muscle weakness and fatigue. She states that she has felt depressed. Physical assessment reveals dark pigmentation of the mucous membranes and skin on the knuckles, knees, and elbows. She appears dehydrated with poor skin turgor. Her blood pressure is 84/50 mm Hg. Blood chemistry reveals sodium level of 132 mEq/L and a potassium level of 5.5 mEq/L. Mrs. James is admitted to the hospital with suspected Addison disease, and an endocrine consult is ordered.

KEY TERMS

Acute adrenal crisis (or Addisonian crisis): acute adrenocortical insufficiency

Adrenocortical excess: increase in adrenocortical function

Adrenocortical insufficiency: decrease in adrenocortical function

INTRODUCTION

This chapter introduces the pharmacologic care of the patient with adrenocortical insufficiency and the patient with adrenocortical excess. The adrenal glands are attached to the upper portion of each kidney. The adrenal cortex of each adrenal gland secretes steroid hormones. The hypothalamic–pituitary–adrenal (HPA) axis regulates hormone secretion. The hypothalamus secretes corticotropin-releasing hormone (CRH), which in turn stimulates the pituitary gland to secrete adrenocorticotropic hormone (ACTH) (Fig. 45.1). The ACTH then stimulates the adrenal cortex to secrete glucocorticoid hormone (cortisol). As the levels of adrenal or steroid hormones increase, the levels of CRH and ACTH decrease through a negative feedback mechanism. For discussion of corticosteroids, see Chapter 17, and for discussion about the types of hormones secreted by the hypothalamus, including CRH, see Chapter 43.

OVERVIEW OF ADDISON DISEASE

Clinical Considerations and Manifestations

Adrenocortical insufficiency is a decrease in adrenocortical function. There are two forms of adrenocortical insufficiency. Primary adrenal insufficiency, or Addison disease, occurs when adrenal cortical hormones are deficient. ACTH levels are elevated because the feedback mechanism is not working. Secondary adrenal insufficiency occurs when there is a disorder in the HPA system.

Primary adrenal insufficiency most commonly results from an autoimmune disorder that has destroyed the layers of the adrenal cortex. Other causes of adrenal cortex destruction include metastatic carcinoma, fungal infections such as histoplasmosis, cytomegalovirus, amyloid disease, and hemochromatosis. Hemorrhage of the adrenal cortex related to anticoagulant therapy, coronary artery bypass graft surgery, giving birth, or trauma also leads to primary adrenal insufficiency.

Secondary adrenal cortical insufficiency results from hypopituitarism or surgical removal of pituitary gland. The abrupt withdrawal of oral glucocorticoids also causes secondary adrenal insufficiency. Patients who have endogenous steroid production from a nonendocrine tumor have adrenocortical insufficiency.

Primary adrenocortical insufficiency is associated with the destruction of the adrenal cortex. The resulting deficiency in the mineralocorticoids causes an increase in the loss of urinary sodium, chloride, and water. The patient becomes hyponatremic, and the cardiac output decreases. This progression of the disease is known as **acute adrenal (or Addisonian) crisis**, which is acute adrenocortical insufficiency. The loss of sodium leads to retention of potassium, resulting in symptoms of hyperkalemia.

The clinical manifestations of adrenocortical insufficiency are evident when approximately 90% of the adrenal cortex has been destroyed. These signs and symptoms reflect loss of sodium, water, and chloride. Findings include decreased cardiac output, dehydration, weakness, and fatigue. Excessive sodium loss results in cardiovascular collapse and shock. Other symptoms include lethargy, weakness, fever, anorexia, nausea, vomiting, and weight loss. Hyperkalemia and hypoglycemia are present. (Any patient with unexplained severe hypoglycemia requires assessment for adrenal insufficiency.) Hyperpigmentation of the gums and mucous membranes is also present;

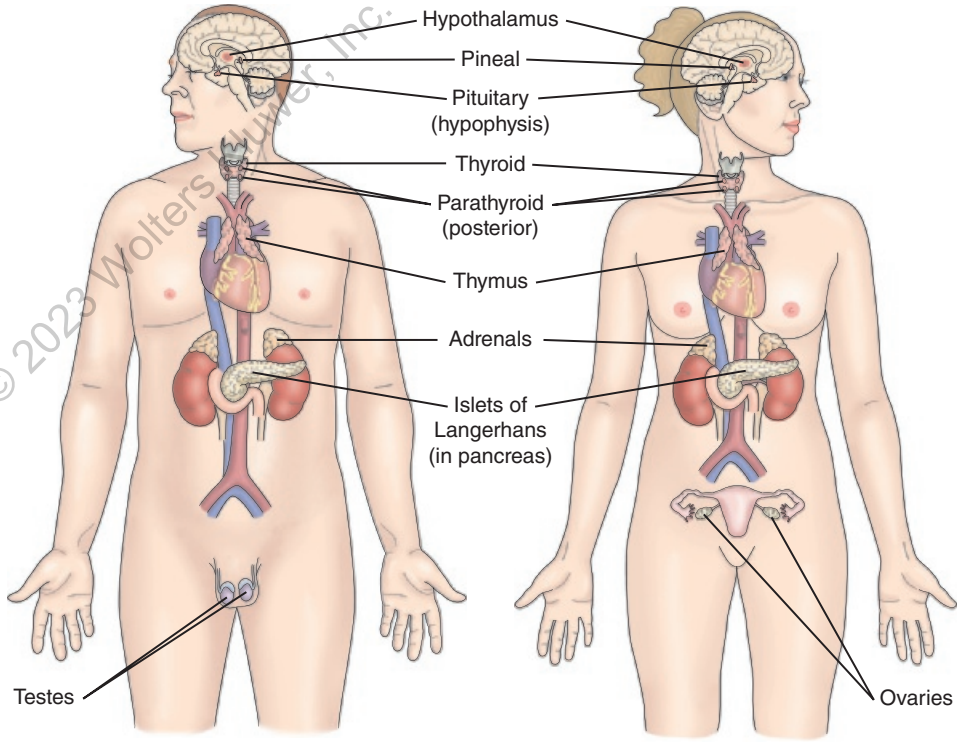

Figure 45.1. The adrenal glands are located on the top of each kidney. Adrenocorticotropic hormone is secreted by the pituitary gland.

they may be bluish black. Females have diminished axillary and pubic hair, but males have few effects from the lack of androgens due to the production of hormones by the testes.

Acute adrenal crisis, or Addisonian crisis, is a life-threatening condition that occurs when Addison disease is the underlying problem and the patient is exposed to minor illness or increased stress. Nausea, vomiting, hypotension, muscle weakness, and vascular collapse are present. There may be a craving for salt.

Clinical Application 45.1

- Mrs. James is hyponatremic. When assessing her cardiac status, what findings would the nurse expect?
- On the second day of Mrs. James' hospital admission, her sodium level is 125 mEq/L. What condition does the nurse suspect?

Quality and Safety Alert: Evidence-Based Practice

Bala et al. [2022] conducted a retrospective study collecting clinical and laboratory data from adrenal insufficiency due to autoimmune adrenalitis in pediatric patients from 2015 to 2020. Primary adrenal insufficiency is rare in children and potentially life-threatening. Pediatric patients present with non-specific symptoms, and it is imperative to establish a diagnosis of adrenal insufficiency. The eight patients identified in the study presented with chronic fatigue, weight loss, altered mental status, and seizures. The median duration of symptoms was 4.5 months. The diagnosis of adrenal insufficiency was confirmed with the serum cortisol and plasma ACTH measurement. The researchers also concluded that appropriate treatment should be instituted and that signs and symptoms of other autoimmune diseases should be investigated.

Diagnosis

Patients commonly present to their primary healthcare provider with vague symptoms of adrenocortical insufficiency. However, as the adrenocortical insufficiency progresses, acute hypotension results and acute adrenal crisis may develop. Making the diagnosis of adrenocortical insufficiency involves laboratory work. This includes early morning serum cortisol and plasma ACTH levels. A serum cortisol level less than 3 mcg/dL, or 80 nmol/L, is indicative of adrenocortical insufficiency. (The normal morning level of serum cortisol is greater—10 to 20 mcg/dL, or 275 to 555 nmol/L.) An ACTH level greater than 22.0 nmol/L is indicative of (primary) adrenocortical insufficiency. (The normal morning level of ACTH is less than 18 nmol/L.)

Confirming the diagnosis of adrenocortical insufficiency requires a short plasma corticotropin stimulation test. The examiner administers corticotropin in the morning, and a subnormal blood cortisol level in the morning and afternoon confirms the diagnosis. A higher cortisol level in the morning is a sign that a person does not have adrenal insufficiency. In a patient with adrenal insufficiency, the response to the corticotropin, or ACTH, is the same both morning and afternoon.

Another test that confirms the diagnosis of adrenocortical insufficiency is the standard high-dose test. It is a three-step process:

1. Measurement of baseline serum cortisol
2. Intravenous (IV) administration of 250 mcg of ACTH 30 minutes later
3. Measurement of serum cortisol 30 to 60 minutes later

An increase of *at least* 18 to 20 mcg/dL is considered normal. No increase in serum cortisol indicates the presence of adrenocortical insufficiency.

Diagnosis of secondary adrenocortical insufficiency in its early stages requires the low-dose test. It is also used for the diagnosis of chronic partial pituitary ACTH deficiency. This test involves the administration of 1 mcg of cosyntropin (Cortrosyn) as an IV bolus. Normally, an increase in cortisol occurs in 20 minutes. In patients with adrenocortical insufficiency, there is no increase in the serum cortisol level.

OVERVIEW OF CUSHING DISEASE

Clinical Considerations and Manifestations

Adrenocortical excess, an increase in adrenocortical function, is the cause of Cushing disease. In most patients, increased adrenocortical function results from excessive corticotropin, leading to hyperplasia of the adrenal cortex. In a smaller percentage of patients, adrenocortical excess is the result of a cortisol-secreting adrenal tumor, whether from too much corticotropin (ACTH) or a primary tumor of the adrenal gland. A malignant tumor of the adrenal gland can produce many corticosteroids, whereas the benign adrenal tumor only produces one corticosteroid that is secreted by the adrenal gland. Other, much less common causes are hyperplasia of the adrenal gland or ectopic production of ACTH by malignancies such as bronchogenic carcinoma. Long-term treatment with pharmacologic glucocorticoids leads to iatrogenic Cushing syndrome.

Patients with Cushing disease often present with classic signs and symptoms. These include obesity, with a heavy trunk and thin extremities, a fatty "buffalo hump" at the neck and supraclavicular region, and a moon-faced appearance. The skin becomes fragile and tears easily, and broad purple striae and bruises may develop. Wound healing may be impaired. The hair is thin. Females have virilization with the appearance of masculine traits such as increased facial hair, breast atrophy, enlarged clitoris, disrupted menses, and voice deepening. Libido is diminished or absent in males and females. Depression, weakness, and lassitude may also occur.

The excessive secretion of corticotropin leads to osteoporosis and fractures, which are caused by the increase in calcium reabsorption from the bone. Blood glucose levels are also increased, and glucose intolerance may occur as a result of increased hepatic gluconeogenesis and resistance to insulin. Peptic ulcers may develop because of increased secretion of gastric acid and pepsin.

Diagnosis

The diagnosis of Cushing disease requires an overnight dexamethasone suppression test. The patient takes dexamethasone,

TABLE 45.1

Drugs Administered for Addison Disease

Drug Class	Prototypes	Other Drugs in the Class
Adrenocorticoid/ mineralocorticoids	Hydrocortisone (Alkindi Sprinkle, Cortef, Solu-CORTEF)	None
Mineralocorticoids	Fludrocortisone	None

a synthetic glucocorticoid, 1 mg orally at 11 p.m. A serum cortisol level is drawn at 8 a.m. A cortisol level of less than 5 mcg/dL indicates that the HPA axis is functioning normally. Cortisol levels are higher in patients with adrenal or ectopic tumors.

DRUGS USED TO TREAT ADDISON DISEASE

The goal of treatment for Addison disease is to replace the adrenocorticoids to correct adrenal insufficiency. It is important to replace both the mineralocorticoid and adrenocorticoid. Lifetime hormone replacement is necessary. Table 45.1 summarizes the adrenocorticoid and mineralocorticoids Administered for the treatment of adrenocortical insufficiency.

ADRENOCORTICOIDS/ MINERALOCORTICOIDS

The prototype (P) **hydrocortisone** (Alkindi Sprinkle, Cortef, Solu-CORTEF), a combination of a mineralocorticoid and adrenocorticoid, is useful in acute and chronic adrenal insufficiency.

Pharmacokinetics

The oral preparation of hydrocortisone has a 1- to 2-hour onset of action, a peak of action in 1 to 2 hours, and a duration of action of 1 to 1.5 days. The parenteral preparation of the drug has an immediate onset of action, an unknown peak of action, and a duration of action of 1 to 1.5 days. Metabolism occurs in the liver. Excretion is in the kidneys.

Action

Hydrocortisone enters the cells and binds to the receptors in the cytoplasm to decrease inflammation; it suppresses the migration of polymorphonuclear lymphocytes and decreases capillary permeability. The mineralocorticoid in the drug increases the retention of sodium.

Use

Healthcare providers use hydrocortisone to replace adrenocorticoids and mineralocorticoids in patients with Addison disease. The drug is also useful in congenital adrenal hyperplasia. Table 45.2 gives route and dosage information for adrenocorticoids and mineralocorticoids.

Patient-related variables specific to the use of hydrocortisone include the following:

- Age:
  - In infants and children, the dose is individualized depending on the severity of the adrenal insufficiency and the response to the medication. Because the drug may affect growth velocity, it is important to carefully assess growth and development. Also, the neonate's respiratory status is assessed closely after administration of parenteral hydrocortisone. Some preparations contain benzyl alcohol, which may cause gasping syndrome in neonates.
  - In premature neonates, the use of high-dose dexamethasone for the treatment of bronchopulmonary dysplasia

TABLE 45.2

DRUGS AT A GLANCE: Adrenocorticoids/Mineralocorticoids and Mineralocorticoids

Drug	Routes and Dosage Ranges	
	Adults	Children
(P) **Hydrocortisone** (Alkindi Sprinkle, Cortef, Solu-CORTEF; Cortef, Solu-Cortef)	100–500 mg IV and every 2, 4, or 6 h based on condition and response 15–25 mg PO initially and individualized to patient response	Infants, 2–3 mg/kg IV (preferred) IM, PO initially; maximum dose: 100 mg/dose; then 1–5 mg/kg/dose every 6 h individualized to patient response <5 y, 25–50 mg once followed by 25–50 mg/d in divided doses every 6 h for 24 h then individualized to patient response ≥5 y, 50–100 mg once followed by 50 mg/d in divided doses every 6 h for 24 h then individualized to patient response Adolescents, 100 mg once followed by 100 mg/d in divided doses every 6 h for 24 h then individualized to patient response
(P) **Fludrocortisone** (Florinef)	0.05–0.1 mg PO daily in combination with glucocorticoid therapy; usual maintenance dose 0.05–0.2 mg once daily	0.05–0.2 mg PO daily

(approximately >0.5 mg/kg/day) has been associated with adverse neurodevelopmental outcomes.
    - Use caution in older adults due to the increased risk of adverse effects with systemic corticosteroids. It is imperative to use the smallest possible effective dose for the shortest duration of time.
- Reproduction, pregnancy, and lactation:
    - The development of oral clefts or decreased birth weight has been associated with first trimester systemic corticosteroid use.
    - For treatment of adrenal insufficiency in pregnant patients, hydrocortisone is the preferred corticosteroid.
    - Pregnant patients with adrenal insufficiency should be monitored at least once per trimester.
    - Corticosteroids are present in breast milk and are generally considered acceptable in breast-feeding patients.
- Abnormal kidney function and hepatic impairment:
    - No dosage adjustments are necessary with abnormal kidney function or hepatic impairment.

Adverse Effects

Hydrocortisone has significant adverse effects, including the following:

- Cardiac effects: fluctuations in blood pressure, shock, dysrhythmias, myocardial infarction, embolism, circulatory collapse, heart failure, and cardiac arrest
- Central nervous system (CNS) effects: vertigo, headache, and depression
- Dermatologic effects: fragile skin that tears easily, petechiae, ecchymoses
- Gastrointestinal (GI) effects: peptic or esophageal ulcers, pancreatitis, increased appetite, and weight gain
- Hematologic effects: sodium and fluid retention
- Metabolic effects: hyperglycemia and Cushing syndrome
- Musculoskeletal effects: osteoporosis and spontaneous fractures (long-term administration)
- Reproductive (female) effects: amenorrhea and irregular menses
- Other effects: immunosuppression, muscle weakness, impaired wound healing, and anaphylaxis

Contraindications

Contraindications to hydrocortisone include a known hypersensitivity to the drug or any component of the formulation as well as a serious infection. The U.S. Food and Drug Administration (FDA) has issued a **BOXED WARNING** ◆ stating that patients who are being treated with hydrocortisone should not receive live virus vaccines.

Nursing Implications

Preventing Interactions

Several medications interact with hydrocortisone, increasing or decreasing its effects (Box 45.1). Echinacea may increase the effects of hydrocortisone, whereas St. John's wort may decrease the drug's effects. Hydrocortisone has numerous other interactions. Combination of hydrocortisone with certain drugs may result in the following effects:

- Salicylates: increased serum salicylate levels
- Acetylcholinesterase drugs: diminished therapeutic effect
- Anticoagulants: increased bleeding; it is necessary to monitor the prothrombin time and the international normalized ratio closely
- Food: interference with calcium absorption
- Alcohol: increased risk of gastric mucosal irritation and development of gastric ulcers

BOX 45.1 Drug Interactions: Hydrocortisone

Drugs That Increase the Effects of Hydrocortisone

- Estrogen, hormonal contraceptives, ketoconazole, troleandomycin

Increase steroid blood levels

Drugs That Decrease the Effects of Hydrocortisone

- Cholestyramine, phenobarbital, phenytoin, rifampin

Decrease steroid blood levels

Administering the Medication

People should take the oral preparation with food to decrease gastric irritation.

! Quality and Safety Alert: Safety

Administration of hydrocortisone should take place every morning before 9 a.m. This minimizes HPA suppression.

It is necessary to dilute hydrocortisone sodium succinate to 50 mg/mL and administer the drug as an IV bolus over 30 seconds or over 10 minutes for doses ≥500 mg. For intermittent IV infusion, dilution to 1 mg/mL and administration over 20 to 30 minutes are appropriate.

Assessing for Therapeutic Effects

The nurse assesses the patient's blood pressure, pulse, and respirations for improved cardiac function. The sodium level and fluid volume status are assessed for return to normal range with retention of sodium and water. It is important to assess for increased strength and energy as well as for improved mood and ability to cope with stress.

Assessing for Adverse Effects

The nurse assesses for hypertension, heart failure, or alterations in cardiac output. It is important to assess for normal menses and diminished virilization in females. Also, the nurse checks the patient's ability to fight infection (e.g., normal white blood cell count). In addition, serum blood sugar is assessed to rule out hypoglycemia or hyperglycemia.

BOX 45.2 Patient Teaching Guidelines for Hydrocortisone

- Take the oral preparation of hydrocortisone every day at 9:00 a.m.
- Space other doses of hydrocortisone evenly throughout the day.
- Do not stop the medication abruptly or without notifying the prescriber.
- Inform the healthcare provider of increased stress, as the medication dosage may need to be increased.
- Increase calcium intake if hydrocortisone is administered for a prolonged period.
- Administer antacids between doses of hydrocortisone to prevent gastric irritation.
- Monitor blood sugar daily.
- Wear a medic alert bracelet.
- Report swelling, weight gain, muscle weakness, tarry stools, moon face, fever, infection, inability of wounds to heal, and fatigue.

Patient Teaching

Box 45.2 presents patient teaching guidelines for hydrocortisone.

Clinical Application 45.2

- Mrs. James' endocrinologist orders hydrocortisone 200 mg intravenously every 4 hours. How does the nurse dilute the drug, and over what period does the nurse administer it?
- One hour following the administration of the medication, the endocrinologist orders blood glucose testing. What is the rationale for blood glucose testing?
- What is the action of hydrocortisone?

MINERALOCORTICOIDS

If a patient with Addison disease requires additional mineralocorticoid supplementation, then **fludrocortisone**, a synthetic steroid, is useful. A patient usually takes it in combination with a glucocorticoid. It is important to note that fludrocortisone has also proved effective for the treatment of orthostatic hypotension in older adults.

Pharmacokinetics and Action

Fludrocortisone is absorbed rapidly and completely and is 42% protein-bound. The drug reaches a peak serum level in approximately 1.7 hours, and it has a 3.5-hour serum half-life. Metabolism occurs in the liver. The site of excretion is unknown.

Fludrocortisone has strong mineralocorticoid action. This drug produces sodium retention and potassium excretion to increase blood pressure.

Use

Healthcare providers use fludrocortisone for partial replacement of mineralocorticoids in the treatment of primary and secondary adrenocortical insufficiency resulting from Addison disease.

Patient-related variables specific to the use of fludrocortisone include the following:

- Age:
  - In young children who have taken high doses of fludrocortisone for long periods, the drug may cause hypercortisolism suppression of the HPA axis. This suppression can lead to adrenal crisis.
  - Because fludrocortisone may affect growth velocity, pediatric patients require routine assessment of growth and development.
  - In older adults, the lowest dose possible for the shortest duration is used to prevent the risk of adverse effects.
- Reproduction, pregnancy, and lactation:
  - Use of systemic corticosteroids during pregnancy requires administration of the lowest possible dosage.
  - It is not known if fludrocortisone enters breast milk. The manufacturer recommends cautious use of fludrocortisone during breast-feeding.
- Abnormal kidney function and hepatic impairment:
  - No dosage adjustments are recommended with abnormal kidney function or hepatic impairment.
  - Patients with hepatic impairment or cirrhosis who take fludrocortisone for long periods require close monitoring for fluid retention.

Adverse Effects and Contraindications

The endocrine and metabolic effects of fludrocortisone are HPA axis suppression, growth suppression, hyperglycemia, and hypokalemia alkalosis. The most commonly reported cardiopulmonary adverse effects include heart failure, edema, and hypertension. Other adverse effects include peptic ulcer, acne, bruising, rash, cataracts, muscle weakness, and diaphoresis.

Contraindications include known hypersensitivity to the medication or any component of its formulation as well as a systemic fungal infection.

Nursing Implications

Preventing Interactions

Many medications interact with fludrocortisone, increasing or decreasing its effects (Box 45.3). Amphotericin B, indacaterol, loop diuretics, and thiazide diuretics in combination with fludrocortisone enhance the hypokalemic effect of fludrocortisone. Warfarin in combination with fludrocortisone enhances the anticoagulant effect, placing the patient at risk for hemorrhage.

Administering the Medication

People should take fludrocortisone concomitantly with a glucocorticoid to enhance effectiveness and produce a more normal adrenal response. It is necessary to store the drug in an airtight, light-protected container at a temperature of 59°F to 86°F.

Assessing for Therapeutic Effects

The nurse assesses sodium levels for increased values and potassium levels for decreased values. It is also important to assess

BOX 45.3 Drug Interactions: Fludrocortisone

Drugs That Decrease the Effects of Fludrocortisone

- Acetylcholinesterase inhibitors, neuromuscular agents
  Increase muscle weakness
- Aminoglutethimide
  Increases metabolism of fludrocortisone
- Antacids
  Decrease bioavailability of fludrocortisone
- Barbiturates, primidone
  Decrease serum concentration of fludrocortisone

Drugs That Increase the Effects of Fludrocortisone

- Antifungal agents, calcium channel blockers, macrolide antibiotics
  Decrease the metabolism of corticosteroids
- Aprepitant, estrogens, fosaprepitant, mifepristone, mitotane, telaprevir
  Increase serum concentration of fludrocortisone

intake, output, and blood pressure. The nurse monitors the patient's weight and assesses fluid volume status. If the weight increases by 5 lb in 1 week, it is necessary to notify the prescriber.

Assessing for Adverse Effects

In children, it is essential to assess growth patterns. In all patients, the nurse assesses the patient's fluid and electrolyte status for hypokalemia and alkalosis. Patients are assessed for pedal edema, hypertension, crackles in the lungs, and an audible S_3 that is indicative of heart failure. It is important to assess the GI system for burning, epigastric pain, and bleeding, which are signs of peptic ulcer disease. The nurse assesses the skin for bruising and rash. Muscle weakness is also assessed.

BOX 45.4 Patient Teaching Guidelines for Fludrocortisone

- Eat foods high in potassium such as bananas, potatoes, and orange juice.
- Consume foods high in calcium and vitamins A and D such as dairy products.
- Take supplements containing vitamins B_6 and C, folate, zinc, and phosphorous.
- Decrease sodium in the diet and limit salt intake.
- Report muscle weakness, numbness, fatigue, depression, increased urination, changes in heart rhythm, epigastric pain, and tarry stools.
- Monitor weight and report an increase of 5 lb to the primary healthcare provider.
- See an ophthalmologist every 6 months to determine if cataracts have formed.
- Have periodic laboratory tests as ordered by the prescriber.
- Report swelling of feet, hands, and shortness of breath to the primary healthcare provider.
- Report any infections or injuries to the primary healthcare provider.
- Wear medical alert identification.

Patient Teaching

Box 45.4 contains patient teaching guidelines for fludrocortisone.

NCLEX Success

1. A patient is taking fludrocortisone acetate for adrenal insufficiency. Which of the following symptoms indicates that the patient is hypokalemic?
   A. tetany
   B. irregular pulse rate
   C. decreased pulse rate
   D. muscle weakness

2. The administration of fludrocortisone is necessary in which of the following conditions?
   A. hypoglycemia
   B. hypernatremia
   C. hypercalcemia
   D. hyperphosphatemia

3. A patient receives a prescription of hydrocortisone for adrenal insufficiency. It is necessary to report which of the following conditions to the primary healthcare provider?
   A. fever
   B. headache
   C. insomnia
   D. neuropathic pain

DRUGS USED TO TREAT CUSHING DISEASE

The treatment of Cushing disease depends on the cause of the medical condition. The most common treatment of hypercortisolism is transsphenoidal surgery. Drug therapy is indicated in several situations: when surgery is contraindicated, in preparation for surgery, in occult ectopic ACTH syndrome, with a recurrence of hypercortisolism following surgery, and with treatment using radiation therapy to the pituitary.

GLUCOCORTICOID RECEPTOR ANTAGONISTS

Glucocorticoid receptor antagonists are administered when surgery to treat corticotroph tumors is delayed or contraindicated. The two medications used to normalize the 24-hour urinary cortisol are cabergoline and pasireotide. Cabergoline is an ergot derivative. Its off-label use is to normalize urinary free cortisol levels in the treatment of Cushing syndrome. Pasireotide is a somatostatin analog to maximize the reduction of urinary free cortisol. The maximum reduction of cortisol is usually noted in approximately 2 months.

Cabergoline is a long-acting dopamine receptor agonist. It has a high affinity for D_2 receptors in the anterior pituitary. It inhibits lactation and hyperprolactinemia. Cabergoline is administered orally with food. Cabergoline is extensively

distributed to the pituitary gland. It is 40% to 42% protein-bound, with a half-life of 63 to 69 hours. Metabolism of the drug takes place in the liver by hydrolysis. The peak of action is 2 to 3 hours, with 60% excretion in the feces and approximately 20% in the urine. Patients with impaired liver function will see an increase in cabergoline levels. Hot flashes, edema, and orthostatic hypotension are the most commonly reported adverse effects. Patients who have a history of cardiac valvular disease should not be administered cabergoline. It is also contraindicated in patients with a known ergot hypersensitivity.

Pasireotide (Signifor, Signifor LAR) binds to somatostatin receptor with high affinity for sst_1, sst_2, sst_3, and sst_5. It inhibits ACTH secretion and causes decreased cortisol secretion. Patients with severe hepatic impairment should avoid this medication. If symptoms of hepatic impairment develop, the medication should be discontinued. Gloves should be worn when unpacking, administering, or disposing of pasireotide. Assess the patient for peripheral edema, hyperglycemia, diarrhea, and prolonged partial thromboplastin time. Assess the serum alanine aminotransferase and serum aspartate aminotransferase; if there is an elevation of ALT and AST, the medication dosage should be reduced or discontinued. In addition, assess the blood glucose for elevation.

11-DEOXYCORTISOL INHIBITORS

In Cushing disease, the goal of drug therapy is to inhibit one or more enzymes contained in cortisol synthesis. The antifungal drug (see Chapter 24) **ketoconazole** can inhibit these enzymes. It also prevents the conversion of 11-deoxycortisol to cortisol. Table 45.3 identifies the drugs administered for the treatment of Cushing disease.

Pharmacokinetics and Action

Ketoconazole is absorbed rapidly in the GI tract. The drug is protein-bound. It is metabolized in the liver and excreted by the kidneys.

TABLE 45.3

Drugs Administered for Cushing Disease

Drug Class	Prototype	Other Drugs in the Class
Glucocorticoid receptor antagonists	N/A	Ergot derivative: Cabergoline Somatostatin analog: Pasireotide (Signifor, Signifor LAR
11-Deoxycortisol inhibitors	Ketoconazole	Metyrapone (Metopirone) Etomidate (Amidate)
Antineoplastics	Mitotane (Lysodren)	None

Ketoconazole acts by inhibiting the first step in cortisol biosynthesis and the conversion of deoxycortisol to cortisol.

Use

Healthcare providers use ketoconazole to control cortisol secretion in Cushing disease. Table 45.4 presents route and dosage information for the drugs used in Cushing disease.

Patient-related variables specific to the use of ketoconazole include the following:

- Age:
  - There are no specific variables based on age.
- Reproduction, pregnancy, and lactation:
  - Patients treated with ketoconazole may experience a decrease in ovulatory disturbances and should be informed of the potential return of fertility.
  - Ketoconazole may cause fetal harm.
  - Ketoconazole is present in breast milk.
- Abnormal kidney function and hepatic impairment:
  - No dosage adjustments are necessary with abnormal kidney function.
  - Use of ketoconazole is contraindicated in acute or chronic liver disease.

Adverse Effects and Contraindications

The most commonly reported adverse effects associated with ketoconazole include pruritus, headache, sedation, nausea, vomiting, and abdominal pain. Gynecomastia, impotence, and decreased libido may occur and are related to the decrease in testosterone production. The FDA has issued a **BOXED WARNING** ◆ stating that ketoconazole can cause hepatotoxicity. Therefore, it is contraindicated in acute or chronic liver disease.

Contraindications include a known hypersensitivity to the medication.

Nursing Implications

Preventing Interactions

Some medications interact with ketoconazole, decreasing its effects (Box 45.5). Ketoconazole combined with echinacea puts the patient at risk for hepatotoxicity. In addition, a **BOXED WARNING** ◆ alerts that ketoconazole increases the plasma concentration of methadone, pimozide, cisapride, disopyramide, dronedarone, and ranolazine, and it may prolong QT intervals on an electrocardiogram. Coadministration increases the risk of life-threatening ventricular dysrhythmias, such as torsades de pointes.

Administering the Medication

People should take ketoconazole with water, coffee, tea, or fruit juice. The presence of stomach acid enhances absorption.

Assessing for Therapeutic Effects and Adverse Effects

The nurse assesses for a decrease in blood pressure and checks the blood glucose for normal levels. It is also important to assess

TABLE 45.4
DRUGS AT A GLANCE: Glucocorticoid Receptor Antagonists, 11-Deoxycortisol Inhibitors, and Antineoplastics

Drug	Routes and Dosage Ranges	
	Adults	**Children**
Glucocorticoid Receptor Antagonists		
Cabergoline	0.5 mg PO daily	Not administered to children
Pasireotide (Signifor, Signifor LAR)	0.6 or 0.9 mg subcutaneous two times per day Signifor LAR: Initial: 10 mg once every 28 d; may increase dose to a maximum of 40 mg once every 28 d	Not administered to children
11-Deoxycortisol Inhibitors		
P **Ketoconazole** (Apo-Ketoconazole, Teva-Ketoconazole)	200–400 mg PO 2–3 times per day	Cushing syndrome: second line therapy: Children >12 y and adolescents: Initial PO 400–600 mg/d in 2–3 divided doses; doses can be increased by 200 mg/d every 7–28 d based on patient response. Peripheral precocious puberty: Children > 2 y and adolescents: PO 10–20 mg/kg/d in 3 divided doses
Metyrapone (Metopirone)	250 mg PO four times daily (max dose 6,000 mg) Diagnostic test for hypothalamic–pituitary adrenocorticotropic hormone (ACTH) function: single dose, 30 mg/kg (max 3 g) at midnight; multiple dose, 750 mg/kg every 4 h for 6 doses	Diagnostic test for ACTH function: single dose, 30 mg/kg (min 250 mg; max 750 mg) at midnight; multiple dose, 15 mg/kg every 4 h for 6 doses
Etomidate (Amidate)	0.04–0.05 mg/kg IV per hour titrated to serum cortisol level	Same as adults
Antineoplastics		
P **Mitotane** (Lysodren; Lysodren)	Adrenocortical carcinoma: 2–6 g/d in 3–4 divided doses and then increase incrementally to 9–10 g/d in 3–4 divided doses; max tolerated range is 2–16 g/d, usually 9–10 g/d; max studied dose is 18–19 g/d Cushing syndrome: 500 mg PO three times per day; max dose 4,000–8,000 mg in three divided doses per day	Safety and efficacy not established

for increased muscle strength and cardiopulmonary status without edema or crackles in the lower lobes and audible S_3.

Assessing for adverse effects is also necessary. The nurse assesses for skin irritation and pruritus; nausea, vomiting, and headache; and diminished libido, gynecomastia, and impotence.

BOX 45.5 Drug Interactions: Ketoconazole

Drugs That Decrease the Effects of Ketoconazole

- Antacids, anticholinergics
  Decrease absorption, thus decreasing serum levels
- Isoniazid, rifampin
  Increase metabolism

Patient Teaching

Box 45.6 presents patient teaching guidelines for ketoconazole.

! Quality and Safety Alert: Patient-Centered Care

Stomach acid increases the absorption of some medications, like ketoconazole. Coffee, tea, and fruit juice increase the acidity in the stomach, enhancing the absorption of these medications.

BOX 45.6 Patient Teaching Guidelines for Ketoconazole

- Take the medication with water, coffee, tea, or fruit juice. Acidic drinks enhance absorption.
- Take the drug with food to prevent gastrointestinal upset.
- Do not take antacids.
- Maintain serum liver enzyme laboratory tests as ordered by the prescriber.
- Report clay-colored stools, extreme thirst, and yellowing of skin or eyes. These signs and symptoms indicate elevated liver enzymes.

Other Drugs in the Class

Metyrapone and etomidate are also administered for their inhibition of 11-deoxycortisol. Metyrapone (Metopirone) blocks the final step in cortisol biosynthesis and increases adrenal androgen production. The drug also decreases cortisol production. It is administered as an adjunctive agent to prevent further release of ACTH in patients with mild Cushing disease or following radiation therapy of the pituitary gland.

Etomidate (Amidate) also blocks the production of cortisol. Normally administered to produce sedation, it is a local anesthetic and is discussed in Chapter 50. Prescribers order it for patients with ectopic secretion of ACTH (Cushing disease).

Quality and Safety Alert: Safety

Etomidate has not demonstrated sedation at recommended doses for Cushing disease. However, patients should be managed in an intensive care unit with sedation scoring every 2 hours for the first 24 hours, then every 12 hours, to assess for sedation.

Etomidate lowers serum cortisol to normal in approximately 10 hours. Cortisol levels should be measured every 4 to 6 hours.

ANTINEOPLASTICS

Healthcare providers use the antineoplastic drug (P) **mitotane** (Lysodren) for the treatment of adrenocortical carcinoma. The drug may also be useful for therapy of Cushing disease caused by such carcinoma.

Pharmacokinetics and Action

Mitotane is absorbed rapidly, with approximately 40% of the drug absorbed in the GI tract. The onset of action is 2 to 4 weeks. The half-life of mitotane is 18 to 159 days. The drug is metabolized in the liver and deposited in the adipose tissues. It is eliminated in the urine and feces.

Mitotane is an adrenolytic agent that causes the adrenal cortex to atrophy. The drug affects the mitochondrial adrenal cortical cells, resulting in decreased production of cortisol. It also alters the peripheral metabolism of steroids.

Use

Mitotane is used for the treatment of an inoperable adrenocortical carcinoma. Its unlabeled use is for treatment of Cushing syndrome. Table 45.4 gives route and dosing information for mitotane.

Patient-related variables specific to the use of mitotane include the following:

- Age:
  - Mitotane is associated with moderate emetic potential, so antiemetic use is recommended with pediatric patients.
- Reproduction, pregnancy, and lactation:
  - Use of effective birth control is recommended in patients receiving mitotane.
  - Mitotane crosses the placenta and has the potential to cause fetal harm.
  - Mitotane is present in breast milk, and breast-feeding should be discontinued with its use.
- Abnormal kidney function and hepatic impairment:
  - No dosage adjustments are recommended in patients with abnormal kidney function or mild-to-moderate hepatic impairment.
  - Mitotane is not recommended in severe hepatic impairment.

Adverse Effects and Contraindications

The CNS effects of mitotane include depression, lethargy, and dizziness. GI effects are anorexia, nausea, vomiting, and diarrhea. Neuromuscular effects include weakness and muscle tremors.

Contraindications include known hypersensitivity to the drug.

Nursing Implications

Preventing Interactions

Mitotane increases the metabolism of phenytoin, phenobarbital, and warfarin. The antineoplastic drug decreases the effect of potassium-sparing diuretics. Alcohol increases the CNS depression associated with mitotane.

Administering the Medication

The FDA has issued a **BOXED WARNING** ◆ stating that it is necessary to withhold mitotane in the event the patient develops shock or with trauma, because the primary action of the drug is adrenal suppression, and adrenal crisis can occur. The onset of shock or trauma should lead to a temporary discontinuation of mitotane followed by the administration of steroids.

Quality and Safety Alert: Safety

The Institute for Safe Medication Practices considers mitotane to be a drug that has a heightened risk of causing significant patient harm when used in error.

Assessing for Therapeutic Effects

The nurse assesses for a decrease in blood pressure. It is necessary to check the blood glucose for normal levels. The nurse also

assesses for increased muscle strength and cardiopulmonary status without edema or crackles in the lower lobes and audible S_3.

Assessing for Adverse Effects

The nurse assesses for CNS depression, which may place the patient at risk for injury. It is also important to assess the gait for safety with walking. The nurse assesses for decreased weight related to anorexia or fluid and electrolyte balance related to nausea and vomiting, as well as for muscle weakness and tremors.

Patient Teaching

Box 45.7 presents patient teaching guidelines for mitotane.

NCLEX Success

4. **A patient has received a diagnosis of Cushing disease and is taking ketoconazole. Which of the following conditions affects the treatment plan?**
   A. hypertension with administration of hydrochlorothiazide
   B. type 2 diabetes with the administration of metformin
   C. migraine headaches with the administration of ergotamine
   D. heart failure with the administration of digoxin

5. **A patient receiving mitotane for an inoperable adrenocortical carcinoma is admitted to the emergency department following an automobile accident. The patient is diaphoretic and unresponsive. The patient's blood pressure is 80/30 mm Hg. What medication should the nurse anticipate administering?**
   A. a steroid
   B. an anticoagulant
   C. a beta-adrenergic blocker
   D. a sulfonylurea

BOX 45.7 Patient Teaching Guidelines for Mitotane

- Understand that this drug will decrease the tumor mass but will not cure the disease.
- Report signs and symptoms of adrenal insufficiency, including weakness, fatigue, orthostatic hypotension, nausea, anorexia, vomiting, increase skin pigmentation, and weight loss.
- Do not operate machinery due to diminished alertness and central nervous system depression.

THE NURSING PROCESS

A concept map outlines the nursing process related to drug therapy considerations in this chapter. Additional nursing implications related to the disease process should also be considered in care decisions.

Assessment

- For the patient with Addison's disease, the major focus of the assessment should be on the severity of symptoms and the effectiveness of drug therapy for the treatment of the disease. Assess for dehydration, hypovolemia, hypoglycemia, hyperkalemia, weight loss, hyperpigmentation of gums and mucous membranes, and depression.
- For the patient with Cushing's disease, the major focus of the assessment should be on the severity of symptoms, body image, and drug therapy of the treatment of the disease. Assess for glucose intolerance, fragile skin tissues, truncal obesity, peripheral edema, osteoporosis, moon face, buffalo hump, irritability, and decreased wound healing.

Outcomes of Therapy

The patient will

- Take the drug correctly.
- Practice measures to decrease the need for corticosteroids and minimize adverse effects.
- Be monitored regularly for adverse drug effects.
- Keep appointments for follow-up care.
- Be assisted to cope with body image changes.
- Verbalize or demonstrate essential drug information.

Nursing Interventions

- For the patient taking long-term, systemic mineralocorticoid/glucocorticoid therapy, use supplementary drugs as ordered and nondrug measures to decrease dosage and adverse effects of corticosteroid drugs. Specific measures include the following:
  - Help set reasonable goals of drug therapy. For example, partial relief of symptoms may be better than complete relief if the latter requires larger doses or longer periods of treatment with systemic drugs.
  - Dietary changes may be beneficial. Salt restriction may help prevent hypernatremia, fluid retention, and edema. Foods high in potassium may help prevent hypokalemia. A diet high in protein, calcium, and vitamin D may help prevent osteoporosis. Increased intake of vitamin C may help decrease bleeding in the skin and soft tissues.
  - Avoid exposing the patient to potential sources of infection by washing hands frequently, using aseptic technique when changing dressings, keeping healthcare personnel and visitors with colds or other infections away from the patient, and following other appropriate measures.
  - Handle tissues very gently during any procedures (e.g., bathing, assisting out of bed, venipunctures).

Evaluation

- Interview and observe for relief of symptoms for which mineralocorticoids, adrenocorticoids, cortisol, 11-deoxycortisol inhibitors, and antineoplastic agents have been prescribed.
- Interview and observe for accurate drug administration.
- Interview and observe for use of nondrug measures indicated for the condition being treated.
- Interview and observe for adverse drug effects on a regular basis.
- Interview regarding drug knowledge and effects to be reported to healthcare providers.

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Clinical Judgment in Practice: Section 8: Drugs Affecting the Endocrine System

A 15-year-old patient is seen by the pediatric nurse practitioner (PNP) for the required school physical. The patient's body mass index is greater than 30. The abdominal area is noted with excessive fat tissue in and around the abdomen. The PNP notes that the patient has developed metabolic syndrome. Metabolic syndrome is a clustering of risk factors for type 2 diabetes. The patient has hyperglycemia, dyslipidemia, and hypertension. The patient's hemoglobin A1C is 7.0%. The low-density lipoprotein is 225 mg/dL. The patient's blood pressure is 140/88. The patient's mother reports that the patient plays video games approximately 2 hours per day. The patient has good grades, but with virtual learning, the patient's computer use has increased significantly.

At the initial visit, the PNP prescribes metformin 500 mg extended release with the evening meal. The patient is placed on a calorie reduction diet. The patient and the patient's mother are referred to the dietician and certified diabetic educator. After 3 months on metformin, the patient has only lost 7 lb. The hemoglobin A1C is 6.8%.

Step 1: Recognize Cues

Identify the relevant and important information from different sources, such as the medical history or subjective and objective data.

Answer: *At the initial visit, the patient's body mass index is greater than 30, the hemoglobin A1C is 7.0%, the low-density lipoprotein is 225 mg/dL, and the blood pressure is 140/88. The patient's mother reports the patient has a sedentary lifestyle with the increased use of virtual learning and playing video games. After 3 months on metformin, the patient has only lost 7 lb and the hemoglobin A1C is 6.8%.*

Step 2: Analyze Cues

Organize and link the recognized cues to the patient's clinical presentation.

Answer: *As the PNP, you link the patient's loss of 7 lb and the patient's hemoglobin A1C of 6.8%, a slight improvement from the initial visit. You determine that an improvement in the patient's weight loss and improvement of hemoglobin A1C will be enhanced with the addition of semaglutide as follows:*

Weeks 1 to 4: 0.25 mg once weekly

Weeks 5 to 8: 0.5 mg once weekly

Weeks 9 to 12: 1 mg once weekly

Weeks 13 to 16: 1.7 mg once weekly

≥Week 17: Maintenance dose 2.4 mg once weekly

Step 3: Prioritize Hypotheses

Evaluate hypotheses and rank them according to priority, such as urgency, likelihood, risk, difficulty, and/or time. Cluster your findings to generate a list of problems (actual or potential) you believe the patient is experiencing or may experience and determine the level of urgency. Which problem is of the greatest concern?

Answer: *You prioritize the needs of the patient, which include the need to lose weight and to decrease the hemoglobin A1C. The patient and family are instructed on the action, use, administration, adverse effects, and therapeutic effects of semaglutide. The patient will engage in a support group for adolescents diagnosed with type 2 diabetes. The school nurse meets with the patient weekly to review the patient's diet and exercise.*

Step 4: Generate Solutions

Identify expected outcomes and use hypotheses to define a set of interventions for the expected outcomes.

Answer: *The patient will have a decrease in hemoglobin A1C with the administration of semaglutide and metformin. The patient will reduce carbohydrate intake to aid in weight loss and decrease hemoglobin A1C. The patient will increase physical activity.*

Step 5: Take Actions

Implement the solutions that address the highest priorities.

Answer: *You arrange for the patient to attend biweekly support group meetings for adolescents diagnosed with type 2 diabetes. You instruct the patient and family on a diet with an increased intake of fruits and vegetables. You also instruct the patient on the need to reduce carbohydrate intake, and you and the patient set goals to reduce the intake of fast food. In addition, you and the patient set the goal for them to swim 3 times per week for 30 minutes.*

Step 6: Evaluate Outcomes

Compare observed outcomes against expected outcomes.

Answer: *By week 9 of semaglutide administration, the patient has lost 14 lb and the hemoglobin A1C is 5.8%. In addition, the patient's blood pressure is 126/74. The patient's low-density lipoprotein is 150.*