Corticosteroids

OBJECTIVES

After studying this chapter, you will be able to:

1. Review physiologic effects of endogenous corticosteroids.
2. Discuss clinical indications for use of exogenous corticosteroids.
3. Differentiate between physiologic and pharmacologic doses of corticosteroids.
4. Differentiate between short-term and long-term corticosteroid therapy.
5. List at least 10 adverse effects of long-term corticosteroid therapy.
6. Explain the pathophysiologic basis of adverse effects.
7. State the rationale for giving corticosteroids topically when possible rather than systemically.
8. Use other drugs and interventions to decrease the need for corticosteroids.
9. Discuss the use of corticosteroids in selected populations and conditions.
10. Apply the nursing process with a client receiving long-term systemic corticosteroid therapy, including teaching needs.

APPLYING YOUR KNOWLEDGE

Sue Hubble is a 70-year-old African-American female. She is 5 feet 9 inches tall and weighs 275 pounds. She has type 2 diabetes mellitus, and, as a result of lifelong smoking, has been diagnosed with chronic obstructive pulmonary disease (COPD). In her home, she uses oxygen. In addition to her respiratory drugs and her drugs for diabetes, she has been taking prednisone 20 mg PO daily for the past month. You are Ms. Hubble’s home care nurse.

INTRODUCTION

Corticosteroids, also called glucocorticoids or steroids, are hormones produced by the adrenal cortex, part of the adrenal glands. These hormones affect almost all body organs and are extremely important in maintaining homeostasis when secreted in normal amounts. Disease results from inadequate or excessive secretion. Exogenous corticosteroids are used as drugs in a variety of disorders. Their use must be closely monitored, because they have profound therapeutic and adverse effects. To understand the effects of corticosteroids used as drugs (exogenous corticosteroids), it is necessary to understand the physiologic effects and other characteristics of the endogenous hormones.

Secretion of Endogenous Corticosteroids

Corticosteroid secretion is controlled by the hypothalamus, the anterior pituitary, and adrenal cortex (the hypothalamic–pituitary–adrenal, or HPA, axis). Various stimuli (eg, low plasma levels of corticosteroids; pain; anxiety; trauma; illness; anesthesia) activate the system. These stimuli cause the hypothalamus of the brain to secrete corticotropin-releasing hormone or factor (CRH or CRF), which stimulates the anterior pituitary gland to secrete corticotropin, and corticotropin then stimulates the adrenal cortex to secrete corticosteroids.

The rate of corticosteroid secretion is usually maintained within relatively narrow limits but changes according to need. When plasma corticosteroid levels rise to an adequate level, secretion of corticosteroids slows or stops. The mechanism by which the hypothalamus and anterior pituitary “learn” that no more corticosteroids are needed is called a negative feedback mechanism.

This negative feedback mechanism is normally very important, but it does not work during stress responses. The stress response activates the sympathetic nervous system (SNS) to produce more epinephrine and norepinephrine and the adrenal cortex to produce as much as 10 times the normal amount of cortisol. The synergistic interaction of these hormones increases the person’s ability to respond to stress. However, the increased
Glucocorticoids are secreted cyclically, with the largest amount accounts for little activity and is secreted in minute quantities. To 4 milligrams of corticosterone are secreted daily. Cortisone accounts for a small amount of activity, and approximately 1.5 milligrams of glucocorticoids are secreted daily. Corticosterone of glucocorticoid activity, and approximately 15 to 20 milligrams, and cortisone. Cortisol accounts for at least 95% and immune processes. Glucocorticoids include cortisol, corticosteroids, which are important in metabolic, inflammatory, adrenal cortex, but it is most often used to designate the glucocorticoids and more uniform distribution to the tissues.

Types of Endogenous Corticosteroids

The adrenal cortex produces approximately 30 steroid hormones, which are divided into glucocorticoids, mineralocorticoids, and adrenal sex hormones. Chemically, all corticosteroids are derived from cholesterol and have similar chemical structures. Despite their similarities, however, slight differences cause them to have different functions.

Glucocorticoids

The term corticosteroids actually refers to all secretions of the adrenal cortex, but it is most often used to designate the glucocorticoids, which are important in metabolic, inflammatory, and immune processes. Glucocorticoids include cortisol, corticosterone, and cortisone. Cortisol accounts for at least 95% of glucocorticoid activity, and approximately 15 to 20 milligrams of glucocorticoids are secreted daily. Corticosterone accounts for a small amount of activity, and approximately 1.5 to 4 milligrams of corticosterone are secreted daily. Cortisone accounts for little activity and is secreted in minute quantities. Glucocorticoids are secreted cyclically, with the largest amount being produced in the early morning and the smallest amount during the evening hours (in people with a normal day–night schedule). At the cellular level, glucocorticoids account for most of the characteristics and physiologic effects of the corticosteroids (Box 23-1).

Mineralocorticoids

Mineralocorticoids play a vital role in the maintenance of fluid and electrolyte balance. Aldosterone is the main mineralocorticoid and is responsible for approximately 90% of mineralocorticoid activity. Characteristics and physiologic effects of mineralocorticoids are summarized in Box 23-2.

Adrenal Sex Hormones

The adrenal cortex secretes male (androgens) and female (estrogens and progesterone) sex hormones. Compared with the effect of hormones produced by the testes and ovaries, the adrenal sex hormones have an insignificant effect on normal body function. Adrenal androgens, secreted continuously in small quantities by both sexes, are responsible for most of the physiologic effects exerted by the adrenal sex hormones. They increase protein synthesis (anabolism), which increases the mass and strength of muscle and bone tissue; they affect development of male secondary sex characteristics; and they increase hair growth and libido in women. Excessive secretion of adrenal androgens in women causes masculinizing effects (eg, hirsutism, acne, breast atrophy, deepening of the voice, amenorrhea). Female sex hormones are secreted in small amounts and normally exert few physiologic effects. Excessive secretion may produce feminizing effects in men (eg, breast enlargement, decreased hair growth, voice changes).

Disorders of the Adrenal Cortex

Disorders of the adrenal cortex involve increased or decreased production of corticosteroids, especially cortisol as the primary mineralocorticoid and aldosterone as the primary mineralocorticoid. These disorders include the following:

- **Primary adrenocortical insufficiency (Addison’s disease)** is associated with destruction of the adrenal cortex by disorders such as tuberculosis, cancer, or hemorrhage; with atrophy of the adrenal cortex caused by autoimmune disease or prolonged administration of exogenous corticosteroids; and with surgical excision of the adrenal glands. In primary adrenocortical insufficiency, there is inadequate production of both cortisol and aldosterone.

- **Secondary adrenocortical insufficiency**, produced by inadequate secretion of corticotropin, is most often caused by prolonged administration of corticosteroids. This condition is largely a glucocorticoid deficiency; mineralocorticoid secretion is not significantly impaired.

- **Congenital adrenogenital syndromes and adrenal hyperplasia** result from deficiencies in one or more enzymes required for cortisol production. Low plasma levels of cortisol lead to excessive corticotropin secretion, which then leads to excessive adrenal secretion of androgens and hyperplasia (abnormal increase in number of cells).

- **Androgen-producing tumors** of the adrenal cortex, which are usually benign, produce masculinizing effects.

- **Adrenocortical hyperfunction** (Cushing’s disease) may result from excessive corticotropin or a primary adrenal tumor. Adrenal tumors may be benign or malignant. Benign tumors often produce one corticosteroid normally secreted by the adrenal cortex, but malignant tumors often secrete several corticosteroids.

- **Hyperaldosteronism** is a rare disorder caused by adenoma (a benign tissue from glandular tissue) or hyperplasia of the adrenal cortex cells that produce aldosterone. It is characterized by hypokalemia, hypernatremia, hypertension, thirst, and polyuria.
Drugs Affecting the Endocrine System

**Box 23-1 Effects of Glucocorticoids on Body Processes and Systems**

**Carbohydrate Metabolism**
- ↑Formation of glucose (gluconeogenesis) by breaking down protein into amino acids. The amino acids are then transported to the liver, where they are acted on by enzymes that convert them to glucose. The glucose is then returned to the circulation for use by body tissues or storage in the liver as glycogen.
- ↓Cellular use of glucose, especially in muscle cells. This is attributed to a ↓effect of insulin on the proteins that normally transport glucose into cells and by ↓numbers and functional capacity of insulin receptors.
- Both the ↑production and ↓use of glucose promote higher levels of glucose in the blood (hyperglycemia) and may lead to diabetes mellitus. These actions also increase the amount of glucose stored as glycogen in the liver, skeletal muscles, and other tissues.

**Protein Metabolism**
- ↑Breakdown of protein into amino acids (catabolic effect); ↑rate of amino acid transport to the liver and conversion to glucose
- ↓Rate of new protein formation from dietary and other amino acids (antianabolic effect)
- The combination of ↑breakdown of cell protein and ↓protein synthesis leads to protein depletion in virtually all body cells except those of the liver. Thus, glycogen stores in the body are ↑and protein stores are ↓.

**Lipid Metabolism**
- ↑Breakdown of adipose tissue into fatty acids; the fatty acids are transported in the plasma and used as a source of energy by body cells.
- ↑Oxidation of fatty acids within body cells

**Inflammatory and Immune Responses**
- ↓Inflammatory response. Inflammation is the normal bodily response to tissue damage and involves three stages. First, a large amount of plasma-like fluid leaks out of capillaries into the damaged area and becomes clotted. Second, leukocytes migrate into the area. Third, tissue healing occurs, largely by growth of fibrous scar tissue. Normal or physiologic amounts of glucocorticoids probably do not significantly affect inflammation and healing, but large amounts of glucocorticoids inhibit all three stages of the inflammatory process.
  
  More specifically, corticosteroids stabilize lysosomal membranes (and thereby prevent the release of inflammatory proteolytic enzymes); ↓capillary permeability (and thereby ↓leakage of fluid and proteins into the damaged tissue); ↓the accumulation of neutrophils and macrophages at sites of inflammation (and thereby impair phagocytosis of pathogenic microorganisms and waste products of cellular metabolism); and ↓production of inflammatory chemicals, such as interleukin-1, prostaglandins, and leukotrienes, by injured cells.
- ↑Immune response. The immune system normally protects the body from foreign invaders, and several immune responses overlap inflammatory responses, including phagocytosis. In addition, the immune response stimulates the production of antibodies and activated lymphocytes to destroy the foreign substance. Glucocorticoids impair protein synthesis, including the production of antibodies; ↓the numbers of circulating lymphocytes, eosinophils, and macrophages; and ↓amounts of lymphoid tissue. These effects help to account for the immunosuppressive and antiallergic actions of the glucocorticoids.

**Cardiovascular System**
- Help to regulate arterial blood pressure by modifying vascular smooth muscle tone, by modifying myocardial contractility, and by stimulating renal mineralocorticoid and glucocorticoid receptors
- ↑The response of vascular smooth muscle to the pressor effects of catecholamines and other vasoconstrictive agents

**Nervous System**
- Physiologic amounts help to maintain normal nerve excitability; pharmacologic amounts ↓nerve excitability, slow activity in the cerebral cortex, and alter brain wave patterns.
- ↓Secretion of corticotropin-releasing hormone by the hypothalamus and of corticotropin by the anterior pituitary gland. This results in suppression of further glucocorticoid secretion by the adrenal cortex (negative feedback system).

**Musculoskeletal System**
- Maintain muscle strength when present in physiologic amounts but cause muscle atrophy (from protein breakdown) when present in excessive amounts
- ↓Bone formation and growth and ↑bone breakdown. Glucocorticoids also ↓intestinal absorption and ↑renal excretion of calcium. These effects contribute to bone demineralization (osteoporosis) in adults and to ↓linear growth in children.

**Respiratory System**
- Maintain open airways. Glucocorticoids do not have direct bronchodilating effects, but help to maintain and restore responsiveness to the bronchodilating effects of endogenous catecholamines, such as epinephrine.
- Stabilize mast cells and other cells to inhibit the release of bronchoconstrictive and inflammatory substances, such as histamine.

**Gastrointestinal System**
- ↓Viscosity of gastric mucus. This effect may ↓protective properties of the mucus and contribute to the development of peptic ulcer disease.

↑, increase/increased; ↓, decrease/decreased;
Mechanisms of Action
Like endogenous glucocorticoids, exogenous drug molecules act at the cellular level by binding to glucocorticoid receptors in target tissues. The drugs are lipid soluble and easily diffuse through the cell membranes of target cells. Inside the cell, they bind with receptors in intracellular cytoplasm. The drug–receptor complex then moves to the cell nucleus, where it interacts with DNA to stimulate or suppress gene transcription.

Glucocorticoids increase or decrease transcription of many genes to alter the synthesis of proteins that regulate their many physiologic effects (e.g., enzymes, transport proteins, structural proteins). Metabolic effects do not occur for at least 45 to 60 minutes because of the time required for protein synthesis. Several hours or days may be needed for full production of proteins.

Because the genes vary in different types of body cells, glucocorticoid effects also vary, depending on the specific cells being targeted. For example, supraphysiologic concentrations of glucocorticoids induce the synthesis of lipolytic and proteolytic enzymes and other specific proteins in various tissues. Overall, corticosteroids have multiple mechanisms of action and effects (Fig. 23-1), including the following:

- **Inhibiting arachidonic acid metabolism.** Normally, when a body cell is injured or activated by various stimuli, the enzyme phospholipase A₂ causes the phospholipids in cell membranes to release arachidonic acid. Free arachidonic acid is then metabolized to produce proinflammatory prostaglandins (see Chap. 7) and leukotrienes. At sites of tissue injury or inflammation, corticosteroids induce the synthesis of proteins that suppress the activation of phospholipase A₂. This action, in turn, decreases the release of arachidonic acid and the formation of prostaglandins and leukotrienes.

- **Strengthening or stabilizing biologic membranes.** Two biologic membranes are especially important in inflammatory processes. Stabilization of cell membranes inhibits the release of arachidonic acid and production of prostaglandins and leukotrienes, as described above. Stabilization of lysosomal membranes inhibits release of bradykinin, histamine, enzymes, and perhaps other substances from lysosomes. (Lysosomes are intracellular structures that contain inflammatory chemical mediators and enzymes that destroy cellular debris and phagocytized pathogens.) This reduces capillary permeability and thus prevents leakage of fluid into the injured area and development of edema. It also reduces the chemicals that normally cause vasodilation and tissue irritation.

- **Inhibiting the production of interleukin-1, tumor necrosis factor, and other cytokines.** This action also contributes to the anti-inflammatory and immunosuppressant effects of glucocorticoids.

- **Impairing phagocytes.** The drugs inhibit the ability of phagocytic cells to leave the bloodstream and move into the injured or inflamed tissue.

- **Impairing lymphocytes.** The drugs inhibit the ability of these immune cells to increase in number and perform their functions.

- **Inhibiting tissue repair.** The drugs inhibit the growth of new capillaries, fibroblasts, and collagen needed for tissue repair.

**Indications for Use**
Corticosteroids are extensively used to treat many different disorders. Except for replacement therapy in deficiency states, the use of corticosteroids is largely empiric. Because the drugs affect virtually every aspect of inflammatory and immune responses, they are used in the treatment of a broad spectrum of diseases with an inflammatory or immunologic component.

Corticosteroid preparations applied topically in ophthalmic and dermatologic disorders are discussed in Chapters 63 and effects (Fig. 23-1), including the following:

- Overall, corticosteroids have multiple mechanisms of action and effects (Fig. 23-1), including the following:

  - Secretion of aldosterone is controlled by several factors, most of which are related to kidney function. In general, secretion is increased when the potassium level of extracellular fluid is high, the sodium level of extracellular fluid is low, the renin–angiotensin system of the kidneys is activated, or the anterior pituitary gland secretes corticotropin.

  - Inadequate secretion of aldosterone causes hyperkalemia, hyponatremia, and extracellular fluid volume deficit (dehydration). Hypotension and shock may result from decreased cardiac output. Absence of mineralocorticoids causes death.

  - Excessive secretion of aldosterone produces hypokalemia, hypernatremia, and extracellular fluid volume excess (water intoxication). Edema and hypertension may result.

- **Inhibiting arachidonic acid metabolism.** Normally, when a body cell is injured or activated by various stimuli, the enzyme phospholipase A₂ causes the phospholipids in cell membranes to release arachidonic acid. Free arachidonic acid is then metabolized to produce proinflammatory prostaglandins (see Chap. 7) and leukotrienes. At sites of tissue injury or inflammation, corticosteroids induce the synthesis of proteins that suppress the activation of phospholipase A₂. This action, in turn, decreases the release of arachidonic acid and the formation of prostaglandins and leukotrienes.

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**General Characteristics of Exogenous Corticosteroids**

Mechanisms of Action
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Corticosteroids are extensively used to treat many different disorders. Except for replacement therapy in deficiency states, the use of corticosteroids is largely empiric. Because the drugs affect virtually every aspect of inflammatory and immune responses, they are used in the treatment of a broad spectrum of diseases with an inflammatory or immunologic component.

Corticosteroid preparations applied topically in ophthalmic and dermatologic disorders are discussed in Chapters 63
and 64, respectively. The corticosteroids discussed in this chapter are used to treat potentially serious or disabling disorders. These disorders include the following:

- **Allergic** or hypersensitivity disorders, such as allergic reactions to drugs, serum and blood transfusions, and dermatoses with an allergic component
- **Collagen** disorders, such as systemic lupus erythematosus, scleroderma, and periarteritis nodosa. Collagen is the basic structural protein of connective tissue, tendons, cartilage, and bone, and it is therefore present in almost all body tissues and organ systems. The collagen disorders are characterized by inflammation of various body tissues. Signs and symptoms depend on which body tissues or organs are affected and the severity of the inflammatory process.
- **Dermatologic** disorders that may be treated with systemic corticosteroids include acute contact dermatitis, erythema multiforme, herpes zoster (prophylaxis of postherpetic neuralgia), lichen planus, pemphigus, skin rashes caused by drugs, and toxic epidermal necrolysis.
- **Endocrine** disorders, such as adrenocortical insufficiency and congenital adrenal hyperplasia. Corticosteroids are given to replace or substitute for the natural hormones (both glucocorticoids and mineralocorticoids) in cases of insufficiency and to suppress corticotropin when excess secretion causes adrenal hyperplasia. These conditions are rare and account for a small percentage of corticosteroid usage.
- **Gastrointestinal** disorders, such as ulcerative colitis and regional enteritis (Crohn’s disease)
- **Hematologic** disorders, such as idiopathic thrombocytopenic purpura or acquired hemolytic anemia
- **Hepatic** disorders characterized by edema, such as cirrhosis and ascites
- **Neoplastic** disease, such as acute and chronic leukemias, Hodgkin’s disease, other lymphomas, and multiple myeloma. The effectiveness of corticosteroids in these conditions probably stems from their ability to suppress lymphocytes and other lymphoid tissue.
- **Neurologic** conditions, such as cerebral edema, brain tumor, acute spinal cord injury, and myasthenia gravis
- **Ophthalmic** disorders, such as optic neuritis, sympathetic ophthalmia, and chorioretinitis
- **Organ or tissue transplants and grafts** (eg, kidney, heart, bone marrow). Corticosteroids suppress cellular and humoral immune responses (see Chap. 41) and help prevent rejection of transplanted tissue. Drug therapy is usually continued as long as the transplanted tissue is in place.
Chapter 23  Corticosteroids

- Renal disorders characterized by edema, such as the nephrotic syndrome
- Respiratory disorders, such as asthma, status asthmaticus, chronic obstructive pulmonary disease (COPD), and inflammatory disorders of nasal mucosa (rhinitis). In asthma, corticosteroids increase the number of beta-adrenergic receptors and increase or restore responsiveness of beta receptors to beta-adrenergic bronchodilating drugs. In asthma, COPD, and rhinitis, the drugs decrease mucus secretion and inflammation.
- Rheumatic disorders, such as ankylosing spondylitis, acute and chronic bursitis, acute gouty arthritis, rheumatoid arthritis, and osteoarthritis
- Shock. Corticosteroids are clearly indicated only for shock resulting from adrenocortical insufficiency (Addisonian or adrenal crisis), which may mimic hypovolemic or septic shock. The use of corticosteroids in septic shock has been highly controversial, and randomized studies and meta-analyses have indicated that corticosteroids are not beneficial in treating septic shock. However, more recent small studies indicate possible clinical usefulness in septic shock, because this form of shock may be associated with relative adrenal insufficiency. In anaphylactic shock resulting from an allergic reaction, corticosteroids may increase or restore cardiovascular responsiveness to adrenergic drugs.

Indications for use, routes and dosage ranges are given in Table 23-1.

Contraindications to Use

Corticosteroids are contraindicated in systemic fungal infections and in people who are hypersensitive to drug formulations. They should be used with caution in clients at risk for infections (they may decrease resistance), clients with infections (they may mask signs and symptoms so that infections become more severe before they are recognized and treated), diabetes mellitus (they cause or increase hyperglycemia), peptic ulcer disease, inflammatory bowel disorders, hypertension, congestive heart failure, and renal insufficiency.

**APPLYING YOUR KNOWLEDGE 23-1**

You arrive at Ms. Hubble’s home and begin your assessment of the client. You notice the appearance of white patches on Ms. Hubble’s mouth. What action do you take?

All adrenal corticosteroids are available as drug preparations, as are many synthetic derivatives developed by altering the basic steroid molecule in efforts to increase therapeutic effects while minimizing adverse effects. When corticosteroids are administered from sources outside the body, they are given mainly for replacement or therapeutic purposes. Replacement involves small doses to correct a deficiency state and restore normal function (physiologic effects). Therapeutic purposes require relatively large doses to exert pharmacologic effects. Drug effects involve extension of the physiologic effects of endogenous corticosteroids and new effects that do not occur with small, physiologic doses. The most frequently desired pharmacologic effects are anti-inflammatory, immunosuppressive, antiallergic, and antistress. These are glucocorticoid effects. Mineralocorticoid and androgenic effects are usually considered adverse reactions.

- The drugs are palliative; they control many symptoms but do not cure underlying disease processes. In chronic disorders, they may enable clients to continue the usual activities of daily living and delay disability. However, the disease may continue to progress, and long-term use of systemic corticosteroids inevitably produces serious adverse effects.
- Drug effects vary, so a specific effect may be considered therapeutic in one client but adverse in another. For example, an increased blood sugar level is therapeutic for the client with adrenocortical insufficiency or an islet-cell adenoma of the pancreas, but is an adverse reaction for most clients, especially those with diabetes mellitus. In addition, some clients respond more favorably or experience adverse reactions more readily than others taking equivalent doses. This is partly caused by individual differences in the rate at which corticosteroids are metabolized.
- Administration of exogenous corticosteroids suppresses the HPA axis. This decreases secretion of corticotropin, which, in turn, causes atrophy of the adrenal cortex and decreased production of endogenous adrenal corticosteroids.
- Hydrocortisone, the exogenous equivalent of endogenous cortisol, is the prototype of corticosteroid drugs. When a new corticosteroid is developed, it is compared with hydrocortisone to determine its potency in producing anti-inflammatory and antiallergic responses, increasing deposition of liver glycogen, and suppressing secretion of corticotropin. Daily administration of physiologic doses (15–20 mg of hydrocortisone or its equivalent) or administration of pharmacologic doses (more than 15–20 mg of hydrocortisone or its equivalent) for approximately 2 weeks suppresses the HPA axis. HPA recovery usually occurs within a few weeks or months after corticosteroids are discontinued, but may take 9 to 12 months. During that time, supplemental corticosteroids are usually needed during stressful situations (eg, fever, illness, surgical procedures) to improve the client’s ability to respond to stress and prevent acute adrenocortical insufficiency.
- Anti-inflammatory activity of glucocorticoids is approximately equal when the drugs are given in equivalent
## Table 23-1 Drugs at a Glance: Corticosteroids

<table>
<thead>
<tr>
<th>GENERIC/TRADE NAME</th>
<th>ROUTES AND DOSAGE RANGES</th>
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<tbody>
<tr>
<td><strong>Glucocorticoids</strong></td>
<td></td>
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<tr>
<td>Beclamethasone oral inhalation (QVAR)</td>
<td>1–2 inhalations (40–80 mcg) 2 times daily (maximum daily dose 320 mcg)</td>
</tr>
<tr>
<td>Nasal inhalation (Beconase AQ)</td>
<td>1–2 inhalations (42–84 mcg in each nostril) 2 times daily; as maintenance, 1 inhalation each nostril</td>
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<tr>
<td><strong>Betamethasone</strong> (Celestone)</td>
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<tr>
<td>Betamethasone acetate and sodium phosphate (Celestone Soluspan)</td>
<td>PO 0.6–7.2 mg daily initially, gradually reduced to lowest effective dose IM 0.5–9 mg daily Intra-articular injection 0.25–2 mL</td>
</tr>
<tr>
<td>Budesonide oral inhalation (Pulmicort Terbuhaler, Pulmicort Respules)</td>
<td>Turbuhaler, 200–400 mcg twice daily</td>
</tr>
<tr>
<td>Nasal inhalation (Rhinocort)</td>
<td>256 mcg daily initially (2 sprays each nostril morning and evening or 4 sprays each nostril every morning). When symptoms are controlled, reduce dosage to lowest effective maintenance dose.</td>
</tr>
<tr>
<td>Oral capsule (Entocort EC)</td>
<td>Crohn’s disease, PO 9 mg once daily in the morning, for up to 8 wk</td>
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<tr>
<td>Cortisone (Cortone)</td>
<td>PO 25–300 mg daily, individualized for condition and response</td>
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<tr>
<td>Dexamethasone (Decadron)</td>
<td>PO 0.75–9 mg daily in 2–4 doses; higher ranges for serious diseases</td>
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<tr>
<td>Dexamethasone acetate</td>
<td>IM 8–16 mg (1–2 mL) in single dose, repeated every 1–3 wk if necessary</td>
</tr>
<tr>
<td>Dexamethasone sodium phosphate</td>
<td>IM, IV 0.5–9 mg, depending on severity of disease</td>
</tr>
<tr>
<td>Flunisolide oral inhalation (AeroBid)</td>
<td>2 inhalations (500 mcg) twice daily</td>
</tr>
<tr>
<td>Nasal inhalation (Nasarel)</td>
<td>2 sprays in each nostril twice daily; maximal daily dose 8 sprays in each nostril</td>
</tr>
<tr>
<td>Fluticasone (Flovent) oral inhalation (Fionase) nasal inhalation</td>
<td>2 inhalations (88 mcg) 2 times daily (maximum daily dose 440 mcg inhaled 2 times daily)</td>
</tr>
<tr>
<td>Fluticasone (Flovent) oral inhalation (Fionase) nasal inhalation</td>
<td>200 mcg daily initially (2 sprays each nostril once daily or 1 spray each nostril twice daily). After a few days, reduce dosage to 100 mcg daily (1 spray each nostril once daily) for maintenance therapy.</td>
</tr>
<tr>
<td>Hydrocortisone (Hydrocortone, Cortef)</td>
<td>PO 20–240 mg daily, depending on condition and response</td>
</tr>
<tr>
<td>Hydrocortisone sodium phosphate</td>
<td>IV, IM, Sub-Q 15–240 mg daily in 2 divided doses</td>
</tr>
<tr>
<td>Hydrocortisone sodium succinate</td>
<td>IV, IM 100–400 mg initially, repeated at 2, 4, or 6 hour intervals if necessary</td>
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**Table 23-1 Drugs at a Glance: Corticosteroids**

<table>
<thead>
<tr>
<th>GENERIC/TRADE NAME</th>
<th>ROUTES AND DOSAGE RANGES</th>
<th>Adults</th>
<th>Children</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hydrocortisone retention enema (Cortenema)</td>
<td>Rectally, one enema (100 mg) nightly for 21 d or until optimal response</td>
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<tr>
<td>Hydrocortisone acetate intrarectal foam (Cortifoam)</td>
<td>1 applicatorful 1–2 times daily for 2–3 wk, then once every 2–3 d if needed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Methylprednisolone (Medrol)</td>
<td>PO 4–48 mg daily initially, gradually reduced to lowest effective level</td>
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<tr>
<td>Methylprednisolone sodium succinate (Solu-Medrol)</td>
<td>IV, IM 10–40 mg initially, adjusted to condition and response</td>
<td>Infants and children: IV, IM not less than 0.5 mg/kg/24 hours</td>
<td></td>
</tr>
<tr>
<td>Methylprednisolone acetate (Depo-Medrol)</td>
<td>IM 40–120 mg once daily</td>
<td></td>
<td></td>
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<tr>
<td>Mometasone (Nasonex)</td>
<td>2 sprays (50 mcg/spray) in each nostril once daily (200 mcg/d)</td>
<td>&gt;12 y: Same as adults</td>
<td>3–11 y: 1 spray (50 mcg) in each nostril once daily (100 mcg/d)</td>
</tr>
<tr>
<td>Prednisolone (Delta-Cortef)</td>
<td>PO 5–60 mg daily initially, adjusted for maintenance</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prednisolone acetate</td>
<td>IM 4–60 mg daily initially, adjusted for maintenance</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prednisone (Deltasone)</td>
<td>PO 5–60 mg daily initially, reduced for maintenance</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triamcinolone (Aristocort, Kenacort)</td>
<td>PO 4–48 mg daily initially, reduced for maintenance</td>
<td></td>
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</tr>
<tr>
<td>Triamcinolone acetonide (Kenalog-40)</td>
<td>IM 2.5–60 mg daily, depending on the disease. Reduce dosage and start oral therapy when feasible.</td>
<td>6–12 y: 1–2 inhalations (100–200 mcg) 3–4 times daily or 2–4 inhalations (200–400 mcg) 2 times daily. Maximum daily dose, 12 inhalations (1200 mcg)</td>
<td></td>
</tr>
<tr>
<td>Oral inhalation (Azmacort)</td>
<td>2 inhalations (200 mcg) 3–4 times daily or 4 inhalations (400 mcg) 2 times daily</td>
<td>≥6 y: 2 sprays (110 mcg) in each nostril once daily (total dose 220 mcg/d). May increase to maximal daily dose of 440 mcg if indicated</td>
<td></td>
</tr>
<tr>
<td>Nasal inhalation (Nasacort)</td>
<td>2 sprays (110 mcg) in each nostril once daily (total dose 220 mcg/d). May increase to maximal daily dose of 440 mcg if indicated</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triamcinolone diacetate (Aristocort Forte)</td>
<td>IM 20–80 mg initially</td>
<td>≥6 y: 2 sprays (110 mcg) in each nostril once daily (220 mcg/d) initially; reduce to 1 spray per nostril once daily (110 mcg/d)</td>
<td></td>
</tr>
<tr>
<td>Mineralocorticoid Fiudroccitison (Florinef)</td>
<td>Chronic adrenocortical insufficiency, PO 0.1 mg daily</td>
<td>PO 0.05–0.1 mg daily</td>
<td></td>
</tr>
<tr>
<td>Salt-losing adrenogenital syndromes, PO 0.1–0.2 mg daily</td>
<td></td>
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</table>

*Ophthalmic and dermatologic preparations are discussed in Chapters 63 and 64, respectively.
IM, intramuscular; IV, intravenous; PO, oral; Sub-Q, subcutaneous.
doses (hydrocortisone 20 mg; prednisone and prednisolone 5 mg; methylprednisolone and triamcinolone 4 mg; dexamethasone 0.75 mg; and betamethasone 0.6 mg). Mineralocorticoid activity is high in cortisone (which is rarely used); intermediate in hydrocortisone, prednisolone, and prednisone; and low in newer agents.

- Duration of action also varies and is only known for oral drugs. Betamethasone and dexamethasone last 48 hours; methylprednisolone, prednisolone, prednisone, and triamcinolone last 18 to 36 hours; and hydrocortisone lasts 18 hours.

- Corticosteroids are metabolized in the liver (mainly by cytochrome P450 3A4 enzymes) and conjugated to inactive metabolites. About 25% of the metabolites are excreted in the bile, then in the feces. The other 75% enter the circulation and are excreted by the kidneys. Metabolism is slowed by hepatic disease, and excretion is slowed by renal disease. In these conditions, corticosteroids may accumulate and cause signs and symptoms of hypercorticism.

APPLYING YOUR KNOWLEDGE 23–2

Ms. Hubble asks you why she has to take the prednisone because it has not made her COPD go away. How should you respond?

NURSING PROCESS

Assessment

Initiation of Corticosteroid Therapy

- For a client expected to receive short-term corticosteroid therapy, the major focus of assessment is the extent and severity of symptoms. Such data can then be used to evaluate the effectiveness of drug therapy.

- For a client expected to receive long-term, systemic corticosteroid therapy, a thorough assessment is needed. This may include diagnostic tests for diabetes mellitus, tuberculosis, and peptic ulcer disease, because these conditions may develop from or be exacerbated by administration of corticosteroid drugs. If one of these conditions is present, corticosteroid therapy must be altered and other drugs given concomitantly.

- If acute infection is found on initial assessment, it should be treated with appropriate antibiotics either before corticosteroid drugs are started or concomitantly with corticosteroid therapy. This is necessary because corticosteroids may mask symptoms of infection and impair healing. Thus, even minor infections can become serious if left untreated during corticosteroid therapy. If infection occurs during long-term corticosteroid therapy, appropriate antibiotic therapy (as determined by culture of the causative microorganism and antibiotic sensitivity studies) is again indicated. Also, increased doses of corticosteroids are usually indicated to cope with the added stress of the infection.

Previous or Current Corticosteroid Therapy

Initial assessment of every client should include information about previous or current treatment with systemic corticosteroids. This can usually be determined by questioning the client or reviewing medical records.

- If the nurse determines that the client has taken corticosteroids in the past, additional information is needed about the specific drug and dosage taken, the purpose and length of therapy, and when therapy was stopped. Such information is necessary for planning nursing care. If the client had an acute illness and received an oral or injected corticosteroid for approximately 1 week or received corticosteroids by local injection or application to skin lesions, no special nursing care is likely to be required. However, if the client took systemic corticosteroids for 2 weeks or longer during the past year, nursing observations must be especially vigilant. Such a client may be at higher risk for acute adrenocortical insufficiency during stressful situations. If the client has surgery, corticosteroid therapy is restarted either before or on the day of surgery and continued, in decreasing dosage, for a few days after surgery. In addition to anesthesia and surgery, potentially significant sources of stress include hospitalization, various diagnostic tests, concurrent infection or other illnesses, and family problems.

If the client is currently taking a systemic corticosteroid drug, again the nurse must identify the drug, the dosage and schedule of administration, the purpose for which the drug is being taken, and the length of time involved. After this basic information is obtained, the nurse can further assess client status and plan nursing care. Some specific factors include the following:

- If the client undergoes anesthesia and surgery, expect that higher doses of corticosteroids will be given for several days. This may be done by changing the drug, the route of administration, and the dosage. Specific regimens vary according to type of anesthesia, surgical procedure, client condition, prescriber preference, and other variables. A client having major abdominal surgery may be given 300 to 400 mg of hydrocortisone (or the equivalent dosage of other drugs) on the day of surgery and then be tapered back to maintenance dosage within a few days.

- Note that additional corticosteroids may be given in other situations as well. One extra dose may be adequate for a short-term stress situation, such as an angiogram or other invasive diagnostic test.

- Using all available data, assess the likelihood of the client’s having acute adrenal insufficiency.
● Assess for signs and symptoms of adrenocortical excess and adverse drug effects.
● Assess for signs and symptoms of the disease for which long-term corticosteroid therapy is being given.

**Nursing Diagnoses**

- Disturbed Body Image related to cushingoid changes in appearance
- Imbalanced Nutrition: Less Than Body Requirements related to protein and potassium losses
- Imbalanced Nutrition: More Than Body Requirements related to sodium and water retention and hyperglycemia
- Excess Fluid Volume related to sodium and water retention
- Risk for Injury related to adverse drug effects of impaired wound healing, increased susceptibility to infection, weakening of skin and muscles, osteoporosis, gastrointestinal ulceration, diabetes mellitus, hypertension, and acute adrenocortical insufficiency
- Ineffective Coping related to chronic illness; long-term drug therapy and drug-induced mood changes, irritability and insomnia
- Deficient Knowledge related to disease process and corticosteroid drug therapy

**Planning/Goals**

**The client 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

**Interventions**

For clients on long-term, systemic corticosteroid therapy, use supplementary drugs as ordered and nondrug measures to decrease dosage and adverse effects of corticosteroid drugs. Specific measures include the following:

- Help clients 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.
- In clients with bronchial asthma and COPD, other treatment measures should be continued during corticosteroid therapy. With asthma, the corticosteroid needs to be given on a regular schedule; inhaled bronchodilators can usually be taken as needed.
- In clients with rheumatoid arthritis, rest, physical therapy, and salicylates or other nonsteroid anti-inflammatory drugs are continued. Systemic corticosteroid therapy is reserved for severe, acute exacerbations when possible.
- Help clients identify stressors and find ways to modify or avoid stressful situations when possible. For example, most clients probably do not think of extreme heat or cold or minor infections as significant stressors. However, they can be for people taking corticosteroids. This assessment of potential stressors must be individualized because a situation viewed as stressful by one client may not be stressful to another.
- Encourage activity, if not contraindicated, to slow demineralization of bone (osteoporosis). This is especially important in postmenopausal women who are not taking replacement estrogens, because they are very susceptible to osteoporosis. Walking is preferred if the client is able. Range-of-motion exercises are indicated in immobilized or bedridden people. Also, bedridden clients taking corticosteroids should have their positions changed frequently because these drugs thin the skin and increase the risk of pressure ulcers. This risk is further increased if edema also is present.
- Dietary changes may be beneficial in some clients. Salt restriction may help prevent hyponatremia, 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 client to potential sources of infection by washing hands frequently; using aseptic technique when changing dressings; keeping health care personnel and visitors with colds or other infections away from the client; and following other appropriate measures. Reverse or protective isolation is sometimes indicated, commonly for those clients who have had organ transplantation and are receiving corticosteroids to help prevent rejection of the transplanted organ.
- Handle tissues very gently during any procedures (eg, bathing, assisting out of bed, venipunctures). Because long-term corticosteroid therapy weakens the skin and bones, there are risks of skin damage and fractures with even minor trauma.
- This assessment of potential stressors must be individualized because a situation viewed as stressful by one person may not be stressful to another.

Client Teaching Guidelines for Long-Term Corticosteroid Therapy are presented in the accompanying display.

**Evaluation**

- Interview and observe for relief of symptoms for which corticosteroids were 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 health care providers.
In most instances, corticosteroids are used to relieve symptoms; they do not cure the underlying disease process. However, they can improve comfort and quality of life.

When taking an oral corticosteroid (eg, prednisone) for longer than 2 weeks, it is extremely important to take the drug as directed. Missing a dose or two, stopping the drug, changing the amount or time of administration, taking extra drug (except as specifically directed during stress situations), or any other alterations may result in complications. Some complications are relatively minor; several are serious, even life threatening. When these drugs are being discontinued, the dosage is gradually reduced over several weeks. They must not be stopped abruptly.

Weigh frequently when starting corticosteroid therapy and at least weekly during long-term maintenance. An initial weight gain is likely to occur and is usually attributed to increased appetite. Later weight gains may be caused by fluid retention.

Do not object when your prescriber reduces your dose of oral corticosteroid for local application must be applied correctly and specifically directed during stress situations), or any other alterations may result in complications. Some complications are relatively minor; several are serious, even life threatening. When these drugs are being discontinued, the dosage is gradually reduced over several weeks. They must not be stopped abruptly.?
Corticosteroids are not the same as the steroids often abused by athletes and body builders. Those are anabolic steroids derived from testosterone, the male sex hormone.

Self- or Caregiver Administration
- Take an oral corticosteroid with a meal or snack to decrease gastrointestinal upset.
- If taking the medication once a day or every other day, take before 9 AM; if taking multiple doses, take at evenly spaced intervals throughout the day.
- Report to the prescriber if unable to take a dose orally because of vomiting or some other problem. In some circumstances, the dose may need to be given by injection.
- If taking an oral corticosteroid in tapering doses, be sure to follow instructions exactly to avoid adverse effects.
- When applying a corticosteroid to skin lesions, do not apply more often than ordered and do not cover with an occlusive dressing unless specifically instructed to do so.

With an intranasal corticosteroid, use on a regular basis (usually once or twice daily) for the best anti-inflammatory effects.

With an oral-inhalation corticosteroid, use on a regular schedule for anti-inflammatory effects. The drugs are not effective in relieving acute asthma attacks or shortness of breath and should not be used “as needed” for that purpose. Use metered-dose inhalers as follows (unless instructed otherwise by a health care provider):
1. Shake canister thoroughly.
2. Place canister between lips (both open and pursed lips have been recommended) or outside lips.
3. Exhale completely.
4. Activate canister while taking a slow, deep breath.
5. Hold breath for 10 seconds or as long as possible.
6. Wait at least 1 minute before taking additional inhalations.
7. Rinse mouth after inhalations to decrease the incidence of oral thrush (a fungal infection).
8. Rinse mouthpiece at least once per day.

APPLYING YOUR KNOWLEDGE 23-3
You test Ms. Hubble’s blood glucose level and it is 345 mg/dL. Why is this client hyperglycemic, and what action should you take?

PRINCIPLES OF THERAPY

Goal of Therapy
The goal of corticosteroid therapy is usually to reduce symptoms to a tolerable level. Total suppression of symptoms may require excessively large doses and produce excessive adverse effects. Because systemic corticosteroids can cause serious adverse reactions, indications for their clinical use should be as clear-cut as possible.

Drug Selection
Choice of corticosteroid drug is influenced by many factors, including the purpose for use, characteristics of specific drugs, desired route of administration, characteristics of individual clients, and expected adverse effects. Some guidelines for rational drug choice include the following:

- Hydrocortisone and cortisone are usually the drugs of choice in adrenocortical insufficiency which requires replacement of both glucocorticoids and mineralocorticoids whether caused by Addison’s disease, adrenalectomy, or inadequate corticotropin. These drugs have greater mineralocorticoid activity compared with other corticosteroids. If additional mineralocorticoid activity is required, fludrocortisone can be given.
- Prednisone is often the glucocorticoid of choice in nonendocrine disorders in which anti-inflammatory, antiallergic, antistress, and immunosuppressive effects are desired. A corticosteroid drug with primarily glucocorticoid activity is necessary.
- Beclomethasone (QVAR, Beconase AQ), budesonide (Pulmicort, Rhinocort), flunisolide (AeroBid), fluticasone (Flonase, Flovent), mometasone (Nasonex), and triamcinolone (Azmacort, Nasacort) are corticosteroids formulated to be given by oral or nasal inhalation in respiratory disorders. Their use replaces, prevents, delays, or decreases use of systemic drugs and thereby decreases risks of serious adverse effects. However, high doses or frequent use may suppress adrenocortical function.
- Dexamethasone (parenterally or orally) is considered the corticosteroid of choice for cerebral edema associated with brain tumors, craniotomy, or head injury, because dexamethasone is thought to penetrate the blood–brain barrier more readily and achieve higher concentrations in cerebrospinal fluids and tissues. It also has minimal sodium- and water-retaining properties. With brain tumors, the drug is more effective in metastatic lesions and glioblastomas than astrocytomas and meningiomas.
- Hydrocortisone, dexamethasone, and methylprednisolone are among those drugs that may be given parenterally and are useful in acute, life-threatening situations that require such administration, usually intravenously (IV). This requirement limits the choice of
drugs, because not all corticosteroids are available in injectable preparations.

**Drug Dosage**

Many factors have an effect on drug dosage, such as the specific drug to be given, the desired route of administration, the reason for use, expected adverse effects, and client characteristics. In general, the smallest effective dose should be given for the shortest effective time. The dosage must be individualized according to the severity of the disorder being treated, whether the disease is acute or chronic, and the client’s response to drug therapy. (Dosage for children is calculated according to severity of disease rather than weight.) If life-threatening disease is present, high doses are usually given until acute symptoms subside. Then the dose is gradually reduced until a maintenance dose is determined or the drug is discontinued. If life-threatening disease is not present, relatively high doses may still be given initially and then lowered. Doses should be gradually reduced (tapered) over several days. With long-term corticosteroid therapy, periodic attempts to reduce dosage are desirable to decrease adverse effects. One way is to reduce the dose gradually until symptoms worsen, indicating the minimally effective dose.

Physiologic doses (approximately 15–20 mg of hydrocortisone or its equivalent daily) are given to replace or substitute for endogenous adrenocortical hormone. Pharmacologic doses (supraphysiologic amounts) are usually required for anti-inflammatory, antiallergic, antistress, and immunosuppressive effects.

Compared with hydrocortisone, newer corticosteroids are more potent on a weight basis but are equipotent in anti-inflammatory effects when given in equivalent doses. Statements of equivalency with hydrocortisone are helpful in evaluating new drugs, comparing different drugs, and changing drugs or dosages. However, dosage equivalents apply only to drugs given orally or IV.

**Drug Administration**

**Routes of Administration**

Corticosteroids can be given by several different routes, based on the clinical problem, to produce local or systemic effects. If feasible, these drugs should be given locally rather than systemically to prevent or decrease systemic toxicity. In recent years there have been several formulations developed for oral inhalation in the treatment of asthma and for nasal inhalation in the treatment of allergic rhinitis. When these drugs must be given systemically, the oral route is preferred. Parenteral administration is indicated only for clients who are seriously ill or unable to take oral medications.

For intramuscular or IV injections, sodium phosphate or sodium succinate salts are used because they have low solubility in water and provide prolonged local action.

**Frequency of Administration**

Scheduling of drug administration is more important with corticosteroids than with most other drug classes. Most adverse effects occur with long-term administration of high doses. A major adverse reaction is suppression of the HPA axis and subsequent loss of adrenocortical function. Certain schedules are often recommended to prevent or minimize HPA suppression.

Corticosteroids can be given in relatively large, divided doses for approximately 48 to 72 hours in acute situations until the condition has been brought under control. After acute symptoms subside or 48 to 72 hours have passed, the dosage is tapered so that a slightly smaller dose is given each day until the drug can be discontinued completely (total period of use: approximately 1 week). Such a regimen may be useful in allergic reactions, contact dermatitis, exacerbations of chronic conditions (eg, bronchial asthma), and stressful situations such as surgery.

Daily administration is required in cases of chronic adrenocortical insufficiency. The entire daily dose can be taken each morning, between 6 and 9 A.M. This schedule simulates normal endogenous corticosteroid secretion.

Alternate-day therapy (ADT), in which a double dose is taken every other morning, is usually preferred for other chronic conditions. This schedule allows rest periods so that adverse effects are decreased while anti-inflammatory effects continue. ADT seems to be as effective as more frequent administration in most clients with bronchial asthma, ulcerative colitis, and other conditions for which long-term corticosteroid therapy is prescribed. ADT is used only for maintenance therapy (ie, clinical signs and symptoms are controlled initially with more frequent drug administration). ADT can be started after symptoms have subsided and stabilized.

Intermediate-acting glucocorticoids (eg, prednisone, prednisolone, methylprednisolone) are the drugs of choice for ADT. Long-acting drugs (eg, betamethasone, dexamethasone) are not recommended because of their prolonged suppression of adrenocortical function.

ADT has other advantages. It probably decreases susceptibility to infection and does not retard growth in children, as do other schedules.

ADT is not usually indicated in clients who have previously received corticosteroids on a long-term basis. First, these clients already have maximal HPA suppression, so a major advantage of ADT is lost. Second, if these clients begin ADT, recurrence of symptoms and considerable discomfort may occur on days when drugs are omitted. Clients with severe disease and very painful or disabling symptoms also may experience severe discomfort with ADT.
Stress Dosage Corticosteroid Therapy

Long-term use of pharmacologic doses (eg, more than 5 mg of prednisone daily) produces adverse reactions. For this reason, long-term corticosteroid therapy should be reserved for life-threatening conditions or severe, disabling symptoms that do not respond to treatment with more benign drugs or other measures. For people receiving chronic corticosteroid therapy, dosage must be increased during periods of stress or illness. Some common sources of stress for most people include surgery and anesthesia, infections, anxiety, and extremes of temperature. Note that events that are stressful for one client may not be stressful for another. Some guidelines for corticosteroid dosage during stress include the following:

- During minor or relatively mild illness (eg, viral upper respiratory infection; any febrile illness; strenuous exercise; gastroenteritis with vomiting and diarrhea; minor surgery), doubling the daily maintenance dose is usually adequate. After the stress period is over, dosage may be reduced abruptly to the usual maintenance dose.
- During major stress or severe illness, even larger doses are necessary. For example, a client undergoing abdominal surgery may require 300 to 400 mg of hydrocortisone on the day of surgery. This dose can gradually be reduced to usual maintenance doses within approximately 5 days if postoperative recovery is uncomplicated. As a general rule, it is better to administer excessive doses temporarily than to risk inadequate doses and adrenal insufficiency. The client also may require sodium chloride and fluid replacement, antibiotic therapy if infection is present, and supportive measures if shock occurs.
- During acute stress situations of short duration, such as traumatic injury or invasive diagnostic tests (eg, angiography), a single dose of approximately 100 mg of hydrocortisone immediately after the injury or before the diagnostic test is usually sufficient.
- Many chronic diseases that require long-term corticosteroid therapy are characterized by exacerbations and remissions. Dosage of corticosteroids usually must be increased during acute flare-ups of disease symptoms but can then be decreased gradually to maintenance levels.

Treatment of Specific Disorders

Allergic Rhinitis

Allergic rhinitis (also called seasonal rhinitis, hay fever, and perennial rhinitis) is a common problem for which corticosteroids are given by nasal spray, once or twice daily. Therapeutic effects usually occur within a few days with regular use. Systemic adverse effects are minimal with recommended doses but may occur with higher doses, including adrenocortical insufficiency from HPA suppression.

Arthritis

Corticosteroids are the most effective drugs for rapid relief of the pain, edema, and restricted mobility associated with acute episodes of joint inflammation. They are usually given on a short-term basis. When inflammation is limited to three or fewer joints, the preferred route of drug administration is by injection directly into the joint. Intra-articular injections relieve symptoms in approximately 2 to 8 weeks, and several formulations are available for this route. However, corticosteroids do not prevent disease progression and joint destruction. As a general rule, a joint should not be injected more often than three times yearly because of risks of infection and damage to intra-articular structures from the injections and from overuse when pain is relieved.

Asthma

Corticosteroids are commonly used in the treatment of asthma because of their anti-inflammatory effects. In addition, corticosteroids increase the effects of adrenergic bronchodilators to prevent or treat bronchoconstriction and bronchospasm. The drugs work by increasing the number and responsiveness of beta-adrenergic receptors and preventing the tolerance usually associated with chronic administration of these bronchodilators. Research studies indicate that responsiveness to beta-adrenergic bronchodilators increases within 2 hours and that numbers of beta receptors increase within 4 hours.

In acute asthma or status asthmaticus unrelieved by inhaled beta-adrenergic bronchodilators, high doses of systemic corticosteroids are given orally or IV along with bronchodilators for approximately 5 to 10 days. Although these high doses suppress the HPA axis, the suppression lasts for only 1 to 3 days, and other serious adverse effects are avoided. Thus, systemic corticosteroids are used for short-term therapy, as needed, and not for long-term treatment. People who regularly use inhaled corticosteroids also require high doses of systemic drugs during acute attacks because aerosols are not effective. As soon as acute symptoms subside, the dose should be tapered; the lowest effective maintenance dose should be used, or the drug should be discontinued.

In chronic asthma, inhaled corticosteroids are the drugs of first choice. This recommendation evolved from increased knowledge about the importance of inflammation in the pathophysiology of asthma and the development of aerosol corticosteroids that are effective with minimal adverse effects. Inhaled drugs may be given alone or with systemic drugs. In general, inhaled corticosteroids can replace oral drugs when daily dosage of the oral drug has been tapered to 10 to 15 milligrams of prednisone or the equivalent. When a client is being switched from an oral to an inhaled corticosteroid, the inhaled drug should be started during tapering of the oral drug, approximately 1 or 2 weeks
before discontinuing or reaching the lowest anticipated dose of the oral drug. When a client requires a systemic corticosteroid, co-administration of an aerosol allows smaller doses of the systemic corticosteroid. Although the inhaled drugs can cause suppression of the HPA axis and adrenocortical function, especially at higher doses, they are much less likely to do so than systemic drugs.

**Cancer**

Corticosteroids are commonly used in the treatment of lymphomas, lymphocytic leukemias, and multiple myeloma. In these disorders, corticosteroids inhibit cell reproduction and are cytotoxic to lymphocytes. In addition to their anti-cancer effects in hematologic malignancies, corticosteroids are beneficial in treatment of several signs and symptoms that often accompany cancer, although the mechanisms of action are unknown and drug/dosage regimens vary widely. Corticosteroids are used to treat anorexia; nausea and vomiting; cerebral edema and inflammation associated with brain metastases or radiation of the head; spinal cord compression; and pain and edema related to pressure on nerves or bone metastases; graft-versus-host disease after bone marrow transplantation; and other disorders that occur in clients with cancer. Clients tend to feel better when taking corticosteroids, although the basic disease process may be unchanged.

**Primary Central Nervous System Lymphomas**

Formerly considered rare tumors of older adults, central nervous system lymphomas are being diagnosed more frequently in younger clients. They are usually associated with chronic immunosuppression caused by immunosuppressant drugs or acquired immunodeficiency syndrome (AIDS). Many of these lymphomas are very sensitive to corticosteroids, and therapy is indicated when the diagnosis is established.

**Other Central Nervous System Tumors**

Corticosteroid therapy may be useful in both supportive and definitive treatment of brain and spinal cord tumors, and neurologic signs and symptoms often improve dramatically within 24 to 48 hours. Corticosteroids help relieve symptoms by controlling edema around the tumor, at operative sites, and at sites receiving radiation therapy. Some clients no longer require corticosteroids after surgical or radiation therapy, whereas others require continued therapy to manage neurologic symptoms. Adverse effects of long-term corticosteroid therapy may include mental changes ranging from mild agitation to psychosis, and steroid myopathy (muscle weakness and atrophy) that may be confused with tumor progression. Mental symptoms usually improve if drug dosage is reduced and resolve if the drug is discontinued; steroid myopathy may persist for weeks or months.

**Chemotherapy-Induced Emesis**

Corticosteroids have strong antiemetic effects; the mechanism is unknown. One effective regimen is a combination of an oral or IV dose of dexamethasone (10–20 mg) and a serotonin antagonist or metoclopramide given immediately before the chemotherapeutic drug. This regimen is the treatment of choice for chemotherapy with cisplatin, which is a strongly emetic drug.

**Chronic Obstructive Pulmonary Disease**

Corticosteroids are more helpful in acute exacerbations than in stable COPD. However, oral corticosteroids may improve pulmonary function and symptoms in some clients. For example, for a client with inadequate relief from a bronchodilator, a trial of a corticosteroid (eg, prednisone 20–40 mg each morning for 5–7 days) may be justified. Treatment should be continued only if there is significant improvement. As in other conditions, the lowest effective dose is needed to minimize adverse drug effects.

Inhaled corticosteroids can also be tried. They produce minimal adverse effects, but their effectiveness in COPD has not been clearly demonstrated.

**Inflammatory Bowel Disease**

Crohn’s disease and ulcerative colitis often require periodic corticosteroid therapy.

In moderate Crohn’s disease, oral prednisone, 40 milligrams daily, is usually given until symptoms subside. With severe disease, clients often require hospitalization, IV fluids for hydration, and parenteral corticosteroids until symptoms subside. An oral form of budesonide (Entocort EC) may be used for Crohn’s disease. The capsule dissolves in the small intestine and acts locally before being absorbed into the bloodstream and transported to the liver for metabolism. It has fewer adverse effects than systemic corticosteroids, but is also less effective and more expensive.

In ulcerative colitis, corticosteroids are usually used when aminosalicylates (eg, mesalamine) are not effective or when symptoms are more severe. Initially, hydrocortisone enemas may be effective. If not effective, oral prednisone (20–60 mg daily) may be given until symptoms subside. In severe disease, oral prednisone may be required initially. After remission of symptoms is achieved, the dose can be tapered by 2.5 to 5 milligrams per day each week to a dose of 20 milligrams. Then, tapering may be slowed to 2.5 to 5 milligrams per day every other week. As in Crohn’s disease, clients with severe ulcerative colitis often require hospitalization and parenteral corticosteroids. One regimen uses IV hydrocortisone 300 milligrams per day or the equivalent dose of another drug. When the client’s condition improves, oral prednisone can replace the IV corticosteroid.
Spinal Cord Injury

High-dose corticosteroid therapy to treat spinal cord injury is a common practice in clinical settings, although controversy exists regarding its use. Data suggest that methylprednisolone may be effective in acute spinal cord injury when given in high doses within 8 hours of the injury. Methylprednisolone improves neurologic recovery, although it does not improve mortality, and its use is unlikely to result in normal neurologic function. In addition, severe adverse outcomes including wound and systemic infections, gastrointestinal hemorrhage, and pneumonia have been reported.

Prevention of Acute Adrenocortical Insufficiency

Suppression of the HPA axis may occur with corticosteroid therapy and may lead to life-threatening inability to increase cortisol secretion when needed to cope with stress. It is most likely to occur with abrupt withdrawal of systemic corticosteroid drugs. The risk of HPA suppression is high with systemic drugs given for more than a few days, although clients vary in degree and duration of suppression with comparable doses, and the minimum dose and duration of therapy that cause suppression are unknown.

When corticosteroids are given for replacement therapy, adrenal insufficiency is lifelong, and drug administration must be continued. When the drugs are given for purposes other than replacement and then discontinued, the HPA axis usually recovers within several weeks to months, but recovery may take a year. Several strategies have been developed to minimize HPA suppression and risks of acute adrenal insufficiency, including:

- Administering a systemic corticosteroid during high-stress situations (eg, moderate or severe illness, trauma, surgery) to clients who have received pharmacologic doses for 2 weeks within the previous year or who receive long-term systemic therapy (ie, are steroid dependent)
- Giving short courses of systemic therapy for acute disorders, such as asthma attacks, then decreasing the dose or stopping the drug within a few days
- Gradually tapering the dose of any systemic corticosteroid. Although specific guidelines for tapering dosage have not been developed, higher doses and longer durations of administration in general require slower tapering, possibly over several weeks. The goal of tapering may be to stop the drug or to decrease the dosage to the lowest effective amount.
- Using local rather than systemic therapy when possible, alone or in combination with low doses of systemic drugs. Numerous preparations are available for local application, including aerosols for oral or nasal inhalation; formulations for topical application to the skin, eyes, and ears; and drugs for intra-articular injections.
- Using ADT, which involves titrating the daily dose to the lowest effective maintenance level, then giving a double dose every other day

Use in Special Populations

Use in Children

Corticosteroids are used for the same conditions in children as in adults; a common indication is for treatment of asthma. With severe asthma, continual corticosteroid therapy may be required. A major concern with children is growth retardation, which can occur with small doses and administration by inhalation. Many children have a growth spurt when the corticosteroid is discontinued. Drug effects on adult stature are unknown.

Parents and prescribers can monitor drug effects by recording height and weight weekly. ADT is less likely to impair normal growth and development than daily administration. In addition, for both systemic and inhaled corticosteroids, each child’s dose should be titrated to the lowest effective amount.

Use in Older Adults

Corticosteroids are used for the same conditions in older adults as in younger ones. Older adults are especially likely to have conditions that are aggravated by the drugs (eg, congestive heart failure, hypertension, diabetes mellitus, arthritis, osteoporosis, increased susceptibility to infection, concomitant drug therapy that increases risks of gastrointestinal ulceration and bleeding). Consequently, risk—benefit ratios of systemic corticosteroid therapy should be carefully considered, especially for long-term therapy.

When used, lower doses are usually indicated because of decreased muscle mass, plasma volume, hepatic metabolism, and renal excretion in older adults. In addition, therapeutic and adverse responses should be monitored regularly by a health care provider (eg, blood pressure, serum electrolytes, and blood glucose levels at least every 6 months). As in other populations, adverse effects are less likely to occur with oral or nasal inhalations than with oral drugs.

Use in Clients With Renal Impairment

Systemic corticosteroids should be used with caution because of slowed excretion, with possible accumulation and signs and symptoms of hypercorticism. In renal transplantation, corticosteroids are extensively used, along with other immunosuppressive drugs, to prevent or treat rejection reactions. In these clients, as in others, adverse effects of systemic corticosteroids may include infections, hypertension, glucose...
intolerance, obesity, cosmetic changes, bone loss, growth retardation in children, cataracts, pancreatitis, peptic ulcerations, and psychiatric disturbances. Doses should be minimized, and eventually the drugs can be withdrawn in some clients.

Use in Clients With Hepatic Impairment
Metabolism of corticosteroids is slowed by severe hepatic disease, and corticosteroids may accumulate and cause signs and symptoms of hypercorticism. In addition, clients with liver disease should be given prednisolone rather than prednisone. Liver metabolism of prednisone is required to convert it to its active form, prednisolone.

Use in Clients With Critical Illness
Corticosteroids have been extensively used in the treatment of serious illness, with much empiric usage.

Adrenal Insufficiency
Adrenal insufficiency is the most clear-cut indication for use of a corticosteroid, and even a slight impairment of the adrenal response during severe illness can be lethal if corticosteroid therapy is not instituted. For example, hypotension is a common symptom in critically ill clients, and hypotension caused by adrenal insufficiency may mimic either hypovolemic or septic shock. If adrenal insufficiency is the cause of the hypotension, administration of corticosteroids can eliminate the need for vasopressor drugs to maintain adequate tissue perfusion.

However, adrenal insufficiency may not be recognized because hypotension and other symptoms also occur with many illnesses. The normal response to critical illness (eg, pain, hypovolemia) is an increased and prolonged secretion of cortisol. If this does not occur, or if too little cortisol is produced, a state of adrenal insufficiency exists. One way to evaluate a client for adrenal insufficiency is a test in which a baseline serum cortisol level is measured, after which corticotropin is given IV to stimulate cortisol production, and the serum cortisol level is measured again in approximately 30 to 60 minutes. Test results are hard to interpret in seriously ill clients, though, because serum cortisol concentrations that would be normal in normal subjects may be low in this population. In addition, a lower-than-expected rise in serum cortisol levels may indicate a normal HPA axis that is already maximally stimulated, or interference with the ability of the adrenal cortex to synthesize cortisol. Thus, a critically ill client may have a limited ability to increase cortisol production in response to stress.

In any client suspected of having adrenal insufficiency, a single IV dose of corticosteroid seems justified. If the client does have adrenal insufficiency, the corticosteroid may prevent immediate death and allow time for other diagnostic and therapeutic measures. If the client does not have adrenal insufficiency, the single dose is not harmful.

Acute Respiratory Failure in Chronic Obstructive Pulmonary Disease
Some studies support the use of IV methylprednisolone. Thus, if other medications do not produce adequate bronchodilation, it seems reasonable to try an IV corticosteroid during the first 72 hours of the illness. However, corticosteroid therapy increases the risks of pulmonary infection.

Adult Respiratory Distress Syndrome
Although corticosteroids have been widely used, several well-controlled studies demonstrate that the drugs are not beneficial in early treatment or in prevention of adult respiratory distress syndrome (ARDS). Thus, corticosteroids should be used in these clients only if there are other specific indications.

Sepsis
Large, well-controlled, multicenter studies have shown that the use of corticosteroids in gram-negative bacteremia, sepsis, or septic shock has no beneficial effect. In addition, the drugs do not prevent development of ARDS or multiple organ dysfunction syndrome or decrease mortality in clients with sepsis. In addition, clients receiving corticosteroids for other conditions are at risk of sepsis, because the drugs impair the ability of white blood cells to leave the bloodstream and reach a site of infection.

Acquired Immunodeficiency Syndrome
Adrenal insufficiency is being increasingly recognized in clients with AIDS, who should be assessed and treated for it, if indicated. In addition, corticosteroids improve survival and decrease risks of respiratory failure with pneumocystosis, a common cause of death in clients with AIDS. The recommended regimen is prednisone 40 milligrams twice daily for 5 days, then 40 milligrams once daily for 5 days, then 20 milligrams daily until completion of treatment for pneumocystosis. The effect of corticosteroids on risks of other opportunistic infections or neoplasms is unknown.

Use in Home Care
Corticosteroids are extensively used in the home setting, by all age groups, for a wide variety of disorders, and by most routes of administration. Because of potentially serious adverse effects, especially with oral drugs, it is extremely important that these drugs be used as prescribed. A major responsibility of home care nurses is to teach, demonstrate, supervise, monitor, or do whatever is needed to facilitate correct use. In addition, home care nurses must teach clients and caregivers interventions to minimize adverse effects of these drugs.

Applying Your Knowledge 23-4
Long-term therapy with corticosteroids involves multiple teaching opportunities for the nurse. What should you include as priorities when providing a teaching plan for Ms. Hubble?
### Nursing Actions

#### Corticosteroids

<table>
<thead>
<tr>
<th>Nursing Actions</th>
<th>Rationale/Explanation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Administer accurately</strong></td>
<td></td>
</tr>
<tr>
<td>a. Read the drug label carefully to be certain of having the correct preparation for the intended route of administration.</td>
<td>Many corticosteroid drugs are available in several different preparations. For example, hydrocortisone is available in formulations for intravenous (IV) or intramuscular (IM) administration, for intra-articular injection, and for topical application in creams and ointments of several different strengths. These preparations cannot be used interchangeably without causing potentially serious adverse reactions and decreasing therapeutic effects. Some drugs are available for only one use. For example, several preparations are for topical use only; beclomethasone is prepared only for oral and nasal inhalation.</td>
</tr>
<tr>
<td>b. With oral corticosteroids:</td>
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<tr>
<td>(1) Give single daily doses or alternate day doses between 6 and 9 A.M.</td>
<td>Early-morning administration causes less suppression of hypothalamic–pituitary–adrenal (HPA) function.</td>
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<tr>
<td>(2) Give multiple doses at evenly spaced intervals.</td>
<td>To avoid adverse effects</td>
</tr>
<tr>
<td>(3) If dosage is being tapered, follow the exact schedule.</td>
<td>To decrease gastrointestinal (GI) upset</td>
</tr>
<tr>
<td>(4) Give with meals or snacks.</td>
<td>This drug is formulated to dissolve in the intestine and have local anti-inflammatory effects. Biting or chewing allows it to dissolve in the stomach.</td>
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<tr>
<td>(5) With oral budesonide (Entocort EC), ask the client to swallow the drug whole, without biting or chewing.</td>
<td>The antacids decrease absorption of corticosteroids, with possible reduction of therapeutic effects.</td>
</tr>
<tr>
<td>(6) Do not give these drugs with an antacid containing aluminum or magnesium (eg, Maalox, Mylanta).</td>
<td>Most of the injectable formulations are suspensions, which need to be mixed well for accurate dosage.</td>
</tr>
<tr>
<td>c. For IV or IM administration:</td>
<td></td>
</tr>
<tr>
<td>(1) Shake the medication vial well before withdrawing medication.</td>
<td>To increase safety of administration</td>
</tr>
<tr>
<td>(2) Give a direct IV injection over at least 1 minute.</td>
<td>These drugs are given by metered-dose inhalers or nasal sprays, and correct usage of the devices is essential to drug administration and therapeutic effects.</td>
</tr>
<tr>
<td>d. For oral or nasal inhalation of a corticosteroid, check the instruction leaflet that accompanies the inhaler.</td>
<td>The primary objective of corticosteroid therapy is to relieve signs and symptoms, because the drugs are not curative. Therefore, therapeutic effects depend largely on the reason for use. These signs and symptoms of impaired metabolism do not occur with adequate replacement of corticosteroids.</td>
</tr>
<tr>
<td><strong>2. Observe for therapeutic effects</strong></td>
<td></td>
</tr>
<tr>
<td>a. With adrenocortical insufficiency, observe for absence or decrease of weakness, weight loss, anorexia, nausea, vomiting, hyperpigmentation, hypotension, hypoglycemia, hyponatremia, and hyperkalemia.</td>
<td></td>
</tr>
<tr>
<td>b. With rheumatoid arthritis, observe for decreased pain and edema in joints, greater capacity for movement, and increased ability to perform usual activities of daily living.</td>
<td></td>
</tr>
<tr>
<td>c. With asthma and chronic obstructive pulmonary disease, observe for decrease in respiratory distress and increased tolerance of activity.</td>
<td></td>
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<tr>
<td>d. With skin lesions, observe for decreasing inflammation.</td>
<td>(continued)</td>
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</tbody>
</table>
**NURSING ACTIONS**

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<tr>
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<tr>
<td>e. When the drug is given to suppress the immune response to organ transplants, therapeutic effect is the absence of signs and symptoms indicating rejection of the transplanted tissue.</td>
<td>These are uncommon with replacement therapy but common with long-term administration of the pharmacologic doses used for many disease processes. Adverse reactions may affect every body tissue and organ.</td>
</tr>
<tr>
<td>3. Observe for adverse effects</td>
<td>This reaction is likely to occur in clients receiving daily corticosteroid drugs who encounter stressful situations. It is caused by drug-induced suppression of the HPA axis, which makes the client unable to respond to stress by increasing adrenocortical hormone secretion.</td>
</tr>
<tr>
<td>a. Adrenocortical insufficiency—fainting, weakness, anorexia, nausea, vomiting, hypotension, shock, and if untreated, death</td>
<td>Most adverse effects result from excessive corticosteroids.</td>
</tr>
<tr>
<td>b. Adrenocortical excess (hypercorticism or Cushing’s disease)</td>
<td>This appearance is caused by abnormal fat deposits in cheeks, shoulders, breasts, abdomen, and buttocks. These changes are more cosmetic than physiologically significant. However, the alterations in self-image can lead to psychological problems. These changes cannot be prevented, but they may be partially reversed if corticosteroid therapy is discontinued or reduced in dosage.</td>
</tr>
<tr>
<td>(1) “Moon face,” “buffalo hump” contour of shoulders, obese trunk, thin extremities</td>
<td>Corticosteroid drugs can cause hyperglycemia and diabetes mellitus or aggravate pre-existing diabetes mellitus by their effects on carbohydrate metabolism.</td>
</tr>
<tr>
<td>(2) Diabetes mellitus—glycosuria, hyperglycemia, polyuria, polydipsia, polyphagia, impaired healing, and other signs and symptoms</td>
<td>Some clients enjoy the drug-induced euphoria so much that they resist attempts to withdraw the drug or decrease its dosage.</td>
</tr>
<tr>
<td>(3) Central nervous system effects—euphoria, psychological dependence, nervousness, insomnia, depression, personality and behavioral changes, aggravation of pre-existing psychiatric disorders</td>
<td>Demineralization of bone produces thin, weak bones that fracture easily. Fractures of vertebrae, long bones, and ribs are relatively common, especially in postmenopausal women and immobilized clients. Myopathy results from abnormal protein metabolism. Decreased growth in children results from impaired bone formation and protein metabolism.</td>
</tr>
<tr>
<td>(4) Musculoskeletal effects—osteoporosis, pathologic fractures, muscle weakness and atrophy, decreased linear growth in children</td>
<td>These effects result largely from mineralocorticoid activity, which causes retention of sodium and water. They are more likely to occur with older corticosteroids, such as hydrocortisone and prednisone.</td>
</tr>
<tr>
<td>(5) Cardiovascular, fluid, and electrolyte effects—fluid retention, edema, hypertension, congestive heart failure, hypernatremia, hypokalemia, metabolic alkalosis</td>
<td>Caused by suppression of normal inflammatory and immune processes and impaired protein metabolism.</td>
</tr>
<tr>
<td>(6) Gastrointestinal effects—nausea, vomiting, possible peptic ulcer disease, increased appetite, obesity</td>
<td>Caused by excessive sex hormones, primarily androgens.</td>
</tr>
<tr>
<td>(7) Increased susceptibility to infection and delayed wound healing</td>
<td></td>
</tr>
<tr>
<td>(8) Menstrual irregularities, acne, excessive facial hair</td>
<td></td>
</tr>
<tr>
<td>(9) Ocular effects—increased intraocular pressure, glaucoma, cataracts</td>
<td></td>
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<tr>
<td>(10) Integumentary effects—skin becomes reddened, thinner, has stretch marks, and is easily injured</td>
<td></td>
</tr>
<tr>
<td>4. Observe for drug interactions</td>
<td></td>
</tr>
<tr>
<td>a. Drugs that increase effects of corticosteroids:</td>
<td></td>
</tr>
<tr>
<td>(1) Estrogens, oral contraceptives, ketoconazole, macrolide antibiotics (eg, erythromycin)</td>
<td>These drugs apparently inhibit the enzymes that normally metabolize corticosteroids in the liver.</td>
</tr>
<tr>
<td>(2) Diuretics (eg, furosemide, thiazides)</td>
<td>Increase hypokalemia</td>
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</tbody>
</table>
**Chapter 23 ● Corticosteroids**

**NURSING ACTIONS**

<table>
<thead>
<tr>
<th>b. Drugs that decrease effects of corticosteroids:</th>
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</thead>
<tbody>
<tr>
<td>(1) Antacids, cholestyramine</td>
</tr>
<tr>
<td>(2) Carbamazepine, phenytoin, rifampin</td>
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</table>

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<tr>
<td>Decrease absorption</td>
</tr>
<tr>
<td>These drugs induce microsomal enzymes in the liver and increase the rate at which corticosteroids are metabolized or deactivated.</td>
</tr>
</tbody>
</table>

**APPLYING YOUR KNOWLEDGE: ANSWERS**

**23-1** Notify Ms. Hubble’s physician. This is a sign of thrush or oral candidiasis, a fungal infection. The client requires immediate treatment and may not be able to continue on the prednisone if the infection becomes systemic. Systemic fungal infections are a contraindication to the administration of corticosteroids.

**23-2** Prednisone is a drug that will treat the symptoms of COPD, but it is not designed to cure the underlying disease process. Because the drug reduces respiratory inflammation, Ms. Hubble will have less difficulty with breathing.

**23-3** Prednisone causes hyperglycemia in many clients and especially in clients with diabetes mellitus. The physician should be notified for a change in Ms. Hubble’s diabetic medication. Also, the nurse should review Ms. Hubble’s diet.

**23-4** The teaching plan should include taking the drug as ordered, carrying medical alert identification that includes the drugs taken, taking measures to avoid infection, and observing for any signs of infection or delayed wound healing. The nurse should encourage activity as tolerated to prevent osteoporosis, and teach the client when to seek medical attention.

**6.** What adverse effects are associated with chronic use of systemic corticosteroids?

**7.** What are the main differences between administering corticosteroids in adrenal insufficiency versus in other disorders?

**8.** When a corticosteroid is given by inhalation to clients with asthma, what is the expected effect?

**NCLEX-Style Questions**

**9.** It is important to taper the dose in long-term systemic corticosteroid therapy rather than stopping the drug abruptly because tapering results in which of the following?
   a. less suppression of hypothalamic–pituitary–adrenal function
   b. increased client compliance with drug therapy
   c. greater tolerance of adverse effects
   d. significantly increased anti-inflammatory effect

**10.** A nurse is instructing a client regarding the correct way follow the order, “prednisone 10 mg PO once daily.” The nurse should tell the client to take
   a. the entire dose once a day at bedtime
   b. the entire dose once a day on arising
   c. half of the dose in the morning and half at bedtime
   d. one third of the dose in the morning and two thirds in the afternoon

**11.** A client who has been on long-term corticosteroid therapy begins to gain weight and complains that her rings no longer fit on her hands. She asks the nurse why she is gaining weight. The nurse tells the client that the most likely cause of later weight gain when undergoing corticosteroid therapy is
   a. increased appetite
   b. hyperglycemia
   c. muscle hypertrophy
   d. fluid retention
12. A client taking an inhaled steroid should be instructed to rinse her mouth after administration of the drug to avoid development of which of the following conditions?
   a. anorexia
   b. nausea and vomiting
   c. thrush
   d. gingival hyperplasia

13. Clients taking glucocorticoids have an increased risk for which of the following conditions?
   a. allergic reaction
   b. hypotension
   c. hypoglycemia
   d. infection

Selected References


