**BOX 24–2 EFFECTS OF MINERALOCORTICOIDS ON BODY PROCESSES AND SYSTEMS**

- The overall physiologic effects are to conserve sodium and water and eliminate potassium. Aldosterone increases sodium reabsorption from kidney tubules, and water is reabsorbed along with the sodium. When sodium is conserved, another cation must be excreted to maintain electrical neutrality of body fluids; thus, potassium is excreted. This is the only potent mechanism for controlling the concentration of potassium ions in extracellular fluids.
- 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.

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 or hyperplasia of the adrenal cortex cells that produce aldosterone. It is characterized by hypokalemia, hypernatremia, hypertension, thirst, and polyuria.

## EXOGENOUS CORTICOSTEROIDS (GLUCOCORTICOID DRUGS)

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. Therapeutic purposes involve 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 effects are anti-inflammatory, immunosuppressive, antiallergic, and antistress. These are glucocorticoid effects. Mineralocorticoid and androgenic effects are usually considered adverse reactions. Additional characteristics of therapeutic corticosteroids include the following:

- 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. These efforts have been most successful in decreasing mineralocorticoid activity.
- The drugs are palliative; they control many symptoms but do not cure underlying disease processes. In chronic disorders, they may enable a client 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 an adverse reaction for most clients, especially for 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. Daily administration of physiologic doses (15 to 20 mg of hydrocortisone or its equivalent) or administration of pharmacologic doses (more than 15 to 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.
- **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.
- Anti-inflammatory activity of glucocorticoids is approximately equal when the drugs are given in equivalent 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.
- Many glucocorticoids are available for use in different clinical problems, and routes of administration vary. Several of these drugs can be given by more than one route; others can be given only orally or topically. For example, in recent years there have been several formulations developed for oral inhalation in the treatment