Endocrine System

PHYSIOLOGY OF THE PITUITARY GLAND
A. Often referred to as the “master gland” because it secretes hormones that control hormone secretion of other endocrine glands (Figure 13-1).
B. Located in the skull beneath the hypothalamus of the brain.
C. Two lobes.
   1. Anterior pituitary: secretes hormones affecting other glands (Table 13-1).
   2. Posterior pituitary: hormones (see Table 13-1) are produced in the hypothalamus and subsequently secreted by the posterior pituitary.

System Assessment
A. Assess for growth imbalance.
   1. Evaluate overall growth pattern of child.
   2. Assess for excessive or retarded growth.
      a. In adults, assess for excessive growth of small bones and soft tissue.
      b. In children, assess for excessive or retarded growth in height.
   3. Evaluate excessive weight gain or loss.
B. Evaluate familial tendencies.
   1. Parents who displayed slower growth patterns.
   2. Compare rate of growth of siblings at age comparable to that of client.
   3. Assess for specific characteristics and/or genetic traits in the adults of the immediate family.
C. Assess for secondary sexual characteristics appropriate to age.
D. Assess intellectual development and mental changes appropriate to physical development and age.
   1. Intelligence.
   2. Increased excitability.
   3. Mental confusion, apathy.

DISORDERS OF THE PITUITARY GLAND

Hyperpituitary: Acromegaly
Acromegaly is most often the result of a benign slow-growing tumor (pituitary adenoma) that secretes growth hormones. It occurs after the closure of epiphyses of the long bones.

Assessment
A. Clinical manifestation.
   1. Enlargement of the hands and feet and hypertrophy of the skin.
   2. Changes in facial features: protruding jaw, slanting forehead, and an increase in the size of the nose.
   3. Severe enlargement of the pituitary gland may cause pressure on the optic nerve, resulting in changes in vision and headaches.
B. Diagnostics (see Appendix 13-1).
C. Complications.
   1. Headache and visual field disturbances.
   2. Diabetes mellitus/glucose intolerance.
   3. Obstructive sleep apnea.

Treatment
Surgical intervention is primary method of correcting problem; hypophysectomy may be accomplished by the transsphenoidal approach.

Nursing Interventions
Goal: To provide supportive preoperative care (see Chapter 3).
Goal: To ensure that the client will not experience complications after hypophysectomy.
A. Elevate the head 30 degrees.
B. Discourage coughing, sneezing, or straining at stool to prevent cerebrospinal fluid leak.
C. Assess for symptoms of increasing intracranial pressure (see Chapter 20).
D. Evaluate urine for excessive increase in volume (>200 mL/hr) or specific gravity <1.005 (i.e., development of diabetes insipidus).
E. Frequent oral hygiene with nonirritating solutions.
Goal: To assist client to reestablish hormone balance after hypophysectomy (adrenal insufficiency and hypothyroidism are most common complications).
A. Administer corticosteroids.
B. If output becomes excessive (because of decrease in antidiuretic hormone [ADH]), anticipate administration of ADH-regulating medications (see Appendix 13-2).
C. Evaluate serum glucose levels for significant changes.
D. Monitor for problems related to hypothyroidism.

**Goal:** To cope with altered body image.
A. Infertility will frequently be experienced as a result of hormone changes.
B. Skeletal changes before surgery are not reversible.
C. Medication.
   1. Cortisone and thyroid hormone replacement throughout lifetime.
   2. ADH-regulating medications.
   3. Medication to support target organs involved (pancreas, thyroid, gonads).

**Disorders of the Posterior Pituitary**

Diabetes insipidus (DI) is characterized by a deficiency of antidiuretic hormone (ADH) or kidney’s inability to respond to ADH. When it occurs, it is most often associated with neurologic conditions, surgery, tumors, head injury, or inflammatory problems.

The syndrome of inappropriate antidiuretic hormone (SIADH) is a condition in which there is continued release of ADH, regardless of the level of plasma osmolarity.
A. When there is a decrease in the serum osmolarity, the normal body response is to decrease the secretion of ADH.
B. When the normal feedback mechanism for ADH fails and the level of ADH is sustained, there is excessive water retention in the body.

### Table 13-1 SUMMARY OF PITUITARY GLAND HORMONE PRODUCTION

<table>
<thead>
<tr>
<th>Hormone Produced</th>
<th>Site of Action</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anterior Lobe</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adrenocorticotropic hormone (ACTH)</td>
<td>Adrenal cortex</td>
<td>Growth and secretion activity of adrenal cortex (stimulates secretion of corticosteroids)</td>
</tr>
<tr>
<td>Thyroid-stimulating hormone (TSH)</td>
<td>Thyroid gland</td>
<td>Controls synthesis and secretion of thyroid hormone</td>
</tr>
<tr>
<td>Follicle-stimulating hormone (FSH)</td>
<td>Testes</td>
<td>Male: stimulates sperm production</td>
</tr>
<tr>
<td>Follicle-stimulating hormone (FSH)</td>
<td>Ovaries</td>
<td>Female: stimulates development of ovarian follicles and ovulation</td>
</tr>
<tr>
<td>Luteinizing hormone (LH)</td>
<td>Testes</td>
<td>Male: stimulates spermatogenesis</td>
</tr>
<tr>
<td>Luteotrophic hormone (LTH), Prolactin (PRL)</td>
<td>Mammary glands</td>
<td>Secretion of milk, maintenance of corpus luteum</td>
</tr>
<tr>
<td>Growth hormone (GH, somatotropin)</td>
<td>All body cells</td>
<td>Growth and maturation of bones, muscles, and other organs</td>
</tr>
<tr>
<td><strong>Posterior Lobe</strong></td>
<td>Renal tubules</td>
<td>Increases reabsorption of water, thereby decreasing urinary output</td>
</tr>
<tr>
<td>Antidiuretic hormone (ADH)</td>
<td>Smooth muscle (particularly wall of uterus and mammary glands)</td>
<td>Contraction of uterus at end of gestation Initiates let-down response of milk during lactation</td>
</tr>
</tbody>
</table>
C. When there is a decreased or inadequate amount of ADH, the body is unable to concentrate urine, and excessive water loss occurs.

**Assessment**

A. Risk factors/etiology: both conditions frequently have predisposing pathology.

B. Clinical manifestations of DI.
   1. Polyuria: excretion of excessive amounts urine (greater than 200 mL/hr).
   2. Polydipsia.
   3. Low urine specific gravity (1.001 to 1.005).
   4. Severe dehydration (tachycardia, poor skin turgor, dry mucous membranes).
   5. Increase in serum sodium level (greater than 147 mEq/L).

C. Clinical manifestations of SIADH.
   1. Low urinary output with weight gain and no obvious edema.
   2. Decreased (dilutional) serum sodium level (less than 120 mEq/L).
   3. Gastrointestinal (GI) disturbances (anorexia, nausea, abdominal cramps).
   5. High specific gravity (greater than 1.030).
   6. Fatigue and muscle aches.

D. Diagnosis based on characteristic clinical manifestations.

**Treatment**

A. Diabetes insipidus: administration of ADH-regulating medications (see Appendix 13-2).

B. SIADH.
   1. Intravenous (IV) administration of normal saline solution (if level is 126–134 mEq/L).
   2. Fluid restriction (limit fluids to 500–1000 mL/24 hr).

**Nursing Interventions**

**Goal:** To maintain fluid and electrolyte balances (see Chapter 6).

A. Encourage intake of fluids containing electrolytes for clients with DI; restrict fluids for clients with SIADH.

B. Monitor intake and output carefully. Weigh daily.

C. Evaluate urine specific gravity for changes.

D. Assess hydration status.

E. Correlate hydration status with weight gain or loss.

F. Closely monitor sodium and potassium levels with fluid shifts (low sodium in SIADH and increased sodium in DI).

**Goal:** To understand the implications of the condition.

A. DI caused by other problems is usually self-limiting.

B. Correction of SIADH is based on identification and correction of the predisposing condition.

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**PHYSIOLOGY OF THE THYROID GLAND**

A. Located in the anterior portion of the neck behind the trachea.

B. Anterior pituitary gland controls secretions of the thyroid-stimulating hormone (TSH).

C. Release of TSH is controlled by the level of thyroid hormone in the blood.

D. Primary function of thyroid hormone is to control the level of cellular metabolism by secreting thyroxin (T4) and triiodothyronine (T3) (Table 13-2).

E. An inadequate secretion of hormone during fetal life and neonatal development will depress metabolic activity and result in stunted physical and mental growth.

F. Disorders of the thyroid gland result in hyperfunction and hypofunction or simple enlargement of the gland.

**System Assessment**

A. Assess for changes in metabolism.
   1. Significant increase weight or decrease in weight.
   2. Diarrhea or constipation.
   3. Increase or decrease in appetite.
   4. Changes in vital signs.
   5. Changes in texture and appearance of skin.

B. Assess for intellectual development and mental changes.
   1. Intellectual level appropriate for age.
   2. Increased irritability, excitability, nervousness.
   3. Mental confusion, lethargy.

**DISORDERS OF THE THYROID GLAND**

**Hyperthyroidism**

Hyperthyroidism (also called Graves’ disease) or thyrotoxicosis (the signs and symptoms caused by hypermetabolism) is characterized by excessive output of thyroid hormones.

**Assessment**

A. Risk factors.
   1. More prevalent in women and older adults.
   2. Peak incidence in third or fourth decade of life.
   3. Probably an autoimmune condition.

**Table 13-2**

<table>
<thead>
<tr>
<th>Hormone Produced</th>
<th>Site of Action</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thyroxine (T4)</td>
<td>General</td>
<td>Controls metabolic rate and growth and development</td>
</tr>
<tr>
<td>Triiodothyronine (T3)</td>
<td>Bone</td>
<td>Inhibits bone resorption, lowers serum calcium levels</td>
</tr>
</tbody>
</table>

Note: Thyroid gland hormone secretion is controlled by the secretion of thyroid-stimulating hormone by the pituitary gland.
B. Clinical manifestations (Figure 13-2).
   1. Increased rate of body metabolism.
      a. Intolerance to heat.
      b. Significant weight loss, despite increased appetite and food intake.
      c. Tachycardia, increase in systolic blood pressure.
      d. Increased peristalsis, leading to diarrhea.
      e. Hand tremors at rest.
      a. Exophthalmos (bulging eyeballs).
      b. Changes in vision.
      c. Eyelid retraction (lid lag).
   3. Changes in menstrual cycle.
   4. Enlarged, palpable thyroid gland.
   5. Labile emotional state.
C. Diagnostics (see Appendix 13-1).
   1. Increase in T₃, T₄, and free T₄ serum levels.
   2. Decrease in TSH.
   3. Radioactive iodine uptake test (I¹³¹) greater than 50%.
   4. Client’s physical appearance and symptoms.

Complications
A. Thyroid storm or crisis: may occur after surgery or treatment with radioactive iodine.
   1. Systolic hypertension.
   2. Tachycardia.
   3. Increased temperature (>102° F).
   4. Increased agitation and anxiety.
B. Calcium deficit may occur as a result of trauma to the parathyroid (see Hypoparathyroidism, later in this chapter).

Treatment
A. Surgical: thyroidectomy.
B. Medical.

1. Reduce thyroid tissue: irradiation of thyroid gland with radioactive iodine (I¹³¹), eventually resulting in hypothyroid state.
2. Decrease thyroid synthesis and release (see Appendix 13-2).

Nursing Interventions
Goal: To decrease effects of excess thyroid hormone.
A. Decrease environmental stress (lights, visitors, noise, etc.).
B. Cool environment.
C. Sedatives, if appropriate.
D. Well-balanced meals (high in calories and high in vitamins); small meals served 4 to 6 times per day.

Goal: To protect eyes of client experiencing complications caused by eye changes.
A. Eye drops or ointment.
B. Assess for excess tearing, a sign of dry cornea.
C. Eye patches or mask may be necessary at night.

Goal: To prevent complications of thyroid storm (or crisis) (thyrotoxicosis).
A. Identify risk factors precipitating thyroid crisis.
   1. Inadequate preoperative preparation.
   2. Infection, increased emotional lability.
   4. After treatment of radioactive I¹³¹ ablation.
B. Assess for increase in hyperthyroid state.
   1. Increase in temperature, dehydration.
   2. Tachycardia (heart rate above 130 beats/min).
   3. Pulmonary edema.
   5. Increase in agitation.

Goal: To maintain homeostasis in client experiencing thyroid storm (or crisis).
A. Decrease body temperature and heart rate.
   1. Hypothermia blanket.
   2. Acetaminophen to decrease fever.
   3. Propranolol (Inderal) to treat cardiac issues.
B. Oxygen to meet increased metabolic demands.
C. IV fluids.
D. Hydrocortisone for shock and adrenal insufficiency.
E. Iodine preparations to decrease T₄ output.
F. Assess for cardiac tachydysrhythmias.
G. Propylthiouracil or methimazole (Tapazole) given to inhibit formation of thyroid hormone.

Goal: To provide preoperative nursing measures if surgery is indicated.
A. Demonstrate to client how to provide neck support after surgery.
B. Administer iodine preparations to decrease vascularity of the thyroid gland.
C. Establish baseline data for postoperative comparison.

Goal: To maintain homeostasis after thyroidectomy.
A. Maintain semi-Fowler’s position to avoid tension on the suture line.
B. Administer analgesics for pain.
Hypothyroidism is characterized by a slow deterioration of thyroid function. It occurs primarily in older adults and five times more frequently in women (ages 30 to 60) than in men. Myxedema coma is a life-threatening form of hypothyroidism.

**Assessment**

A. Early clinical manifestations.
1. Extreme fatigue, menstrual disturbances.
2. Hair loss, brittle nails, and dry skin.
3. Intolerance to cold, anorexia.

B. Late clinical manifestations.
1. Subnormal temperature.
2. Cardiac complications (bradycardia, congestive heart failure, hypotension).
3. Weight gain and edema, thickened skin.
4. Change or decrease in level of consciousness.

C. Diagnostics.
1. Decrease in serum T₃ and T₄ levels.
2. Increase in TSH level.

**Treatment**

A. Medical management.
1. Replacement of thyroid hormone.
2. Low-calorie diet to promote weight loss.
3. Decrease in cholesterol intake.

**Complications**

A. Thyroid hormone replacement will increase the workload of the heart and increase myocardial oxygen requirements.

B. Observe client for development of cardiac failure.

**Nursing Interventions**

**Goal:** To assist the client to return to hormone balance.

A. Begin thyroid replacement and evaluate client’s response; advise client that it will be about 7 days before he or she begins to feel better.

B. Provide a warm environment.

**Home Care**

A. Thyroid levels checked annually.
B. Lifelong thyroid replacement.
C. If excessive fatigue or tachycardia and tremors become consistent problems, notify health care provider.

**Hypothyroidism**

C. Prevent and/or treat constipation.
D. Assess progress.
1. Decrease in body weight.
2. Intake and output balance.
3. Decrease in visible edema.
4. Energy level and mental alertness should increase in 7 to 14 days and continue to rise until normal.
E. Evaluate cardiovascular response to medication.

**Goal:** To assist client to understand implications of disease and requirements for health maintenance.

A. Need for lifelong drug therapy.
B. Client with diabetes needs to evaluate blood glucose levels more frequently; thyroid preparations may alter effects of hypoglycemic agents.
C. Continue to reinforce teaching as client begins to make progress; early in the disease, the client may not comprehend importance of information.

**C. Administer IV fluids until nausea and swelling difficulty subside.**
D. Check dressings on the side and back of the neck for bleeding.
E. Assess for hematoma around the wound.
F. Apply ice collar to decrease edema.
G. Evaluate calcium levels; parathyroid may have been damaged or accidentally removed.
H. Evaluate temperature elevations; temperature increase may be early indication of thyroid storm.

### Nursing Priority

Monitor status of client who has undergone surgery (hemorrhage, airway, wound).

**Goal:** To prevent complication of respiratory distress after thyroidectomy.

A. Assess client frequently for noisy breathing and increased restlessness.
B. Evaluate voice changes; increasing hoarseness may be indicative of laryngeal edema.
C. Keep tracheotomy set readily available.

**Goal:** To decrease radiation exposure in client being treated as an in-patient with radioactive iodine (I¹³¹).

A. All body secretions are contaminated because this is a systemic type of radiation.
B. Advise family members to avoid oral contact because saliva is contaminated.
C. For any body fluid spills (urine, vomitus, etc.), contact the radiation safety officer for the facility. Do not clean up the spill until directed to do so.
D. General guideline is to maintain a distance of 1 meter (a little more than 3 feet) from the client unless direct contact is necessary.
E. Infants and pregnant women should avoid contact with the client for approximately 2 days.
F. All health care personnel providing direct care to the client should wear a radiation badge.
G. Monitor client for a transient period of several days to weeks, when the symptoms of hyperthyroidism may actually worsen after radioactive iodine therapy.

**Alert** Observe client for side effects of chemotherapy or radiation.

**Home Care**

A. Thyroid levels checked annually.
B. Lifelong thyroid replacement.
C. If excessive fatigue or tachycardia and tremors become consistent problems, notify health care provider.

**Nursing Priority** Administer sedatives and hypnotics with caution because of increased susceptibility. These medications tend to precipitate respiratory depression in the client with hypothyroidism.
Congenital Hypothyroidism (Cretinism)

Congenital hypothyroidism is a deficiency of thyroid hormones present at birth. Symptoms depend on the amount of thyroid hormones present at birth.

**Assessment**

A. Condition is not generally evident in the newborn because of thyroid hormones received through the maternal circulation; may become evident at 3 to 6 months of age.

B. Early clinical manifestations.
   1. Thick, dry, mottled skin.
   2. Bradycardia.
   3. Hypotonia, hyporeflexia.
   4. Poor feeding.
   5. Hypotonic abdominal musculature.
      a. Constipation.
      b. Protruding abdomen (umbilical hernia).

C. Diagnostics.
   1. Filter-paper blood-spot thyroxine (T₄) test; if result is low, then a TSH test is done.
   2. Mandatory test in all states; should be done within 24 to 48 hours after birth.

**Treatment**

Medical management includes replacement of thyroid hormones. (If replacement is accomplished shortly after birth, it is possible that the child will have normal physical growth and intellectual development.)

**Nursing Interventions**

**Goal:** To identify neonates experiencing congenital hypothyroidism.

**Goal:** To assist parents to understand the implications of the disease and requirements for continued health maintenance.

A. Child will require lifelong medication.

B. Continue medical care to evaluate changes in thyroid replacement as the child grows.

**PHYSIOLOGY OF THE PARATHYROID**

Four small parathyroid glands are located near, or embedded in, the thyroid gland. The hormone secreted is parathyroid hormone (PTH), which is primarily involved in the control of serum calcium levels.

A. Function of PTH:
   1. Facilitates the mobilization of calcium and phosphorous from bone.
   2. Promotes the resorption of calcium from bone to maintain normal serum calcium levels.
   3. Promotes absorption of calcium in the GI tract (by stimulating kidneys to convert vitamin D to its active form).

B. Function of calcium:
   1. Maintains normal muscle and neuromuscular responses.
   2. Necessary component of blood coagulation mechanisms.
   3. Control of PTH occurs by way of the negative feedback system involving the level of serum calcium. When serum calcium levels are low, there is increased mobilization of calcium from bone, resulting in an increase in serum calcium levels.

**System Assessment**

A. History of problems of calcium metabolism and/or thyroid surgery.

B. Assess for changes in mental or emotional status.

C. Evaluate reflexes and neuromuscular response to stimuli.

D. Evaluate serum and urine calcium levels.

**DISORDERS OF THE PARATHYROID**

**Hyperparathyroidism**

Hyperparathyroidism is characterized by excessive secretion of PTH, resulting in hypercalcemia.

A. Normal function of PTH is to maintain serum calcium and phosphate levels.

B. Excessive PTH leads to bone damage, hypercalcemia, and kidney damage.

**Assessment**

A. Risk factors: more common in women after menopause.

B. Clinical manifestations.
   1. Primary hyperparathyroidism is due to adenomas, hyperplasia, or carcinomas. Bone cysts and pathologic fractures occur because of bone decalcification. Calcium is released into the blood, causing hypercalcemia.
   2. Secondary hyperparathyroidism is frequently due to chronic renal failure.
      a. Renal calculi, azotemia.
      b. Hypertension caused by renal failure.
      c. Repeated urinary tract and renal infections.
   3. Central nervous system problems of lethargy, stupor, and psychosis.
   4. GI problems.
      a. Anorexia, nausea, and vomiting.
      b. Constipation, development of peptic ulcer.

C. Diagnostics.
   1. Increased level of serum total calcium; decreased level of serum phosphorous; increased PTH.
   2. CT scan and/or x-ray film show demineralized cystic areas in bone.
   3. Increased urine calcium and phosphorous levels.

**Treatment**

A. Decrease level of circulating calcium.

B. Parathyroidectomy.
**Nursing Interventions**

**Goal:** To decrease the level of serum calcium.
A. High fluid intake to dilute serum calcium and urine calcium levels.
B. If IV is necessary, generally administer normal saline solution.
C. Furosemide (Lasix), a loop diuretic, may be used to increase excretion of calcium.
D. Encourage mobility, because immobility increases demineralization of bones.
E. Limit foods high in calcium.
F. Phosphate replacement.

**Goal:** To assess client’s tolerance of and response to increased PTH level.
A. Assess for skeletal involvement.
B. Assess for renal involvement.
   1. Strain urine for stones.
   2. Evaluate for low back pain (renal).
   3. Check for hematuria.
   4. Assess intake and output carefully.
C. Assess for presence of bone pain.
D. Assess cardiac response to increased level of calcium.

**Goal:** To provide appropriate preoperative measures if surgery is indicated (see Chapter 3).

**Goal:** To prevent postoperative complications of parathyroidectomy.
A. Care of client who has undergone parathyroidectomy is same as that for client who has undergone thyroidectomy.
B. Bone pain is relieved shortly after surgery; bone lesions frequently heal; serious renal disease may not be reversible.

**Hypoparathyroidism**

Hypoparathyroidism is characterized by a decrease in the PTH level, resulting in hypocalcemia and elevated serum phosphate levels. Severe hypocalcemia results in tetany.

**Assessment**

A. Risk factors or precipitating causes.
   1. Adults: inadvertent removal of parathyroid gland during thyroidectomy or radical neck dissection.
   2. Children: primary cause is idiopathic (unknown).
B. Clinical manifestations.
   1. Insidious onset.
      a. Muscle weakness/spasms.
      b. Loss of hair; dry skin.
   2. Overt/acute tetany (potentially fatal).
      a. Bronchospasm, laryngospasm.
      b. Seizures, cardiac dysrhythmias.
      c. Circumoral paresthesia.
      d. Abdominal cramps, nausea, vomiting, diarrhea, anorexia.
      e. Positive Chvostek’s sign (sign is positive when sharp tapping over the facial nerve elicits mouth, nose, and eye twitching).
      f. Trousseau’s phenomenon (present when carpopedal spasm is precipitated by occluding blood flow for 3 minutes to the upper portion of the upper extremity with a blood pressure cuff).

   a. Carpopedal spasms, muscle cramps, twitching.
   b. Seizures: generalized or absence.
   c. Brittle hair, thin nails.

C. Diagnostics.
   1. Decreased serum calcium levels.
   2. Increased serum phosphate levels.
   3. Low PTH levels.

**Treatment**

A. Vitamin D to enhance calcium absorption.
B. Increased calcium in the diet.

**ALERT** Adjust food and fluid intake to improve fluid and electrolyte balances.
C. Acute.
   1. Replace calcium through slow IV drip (calcium gluconate, calcium chloride).
   2. Sedatives, anticonvulsants.

**Nursing Interventions**

**Goal:** To assist client to increase serum calcium levels.
A. Administer calcium preparations.
B. Evaluate for an increase in serum calcium levels and a decrease in serum phosphate levels.
C. Client will require lifelong medical care to maintain homeostasis.

**Goal:** To prevent complications of neuromuscular irritability.
A. Quiet environment.
B. Low lights.
C. Seizure precautions.

**Goal:** To help client avoid complications of respiratory distress.
A. Bronchodilators.
B. Tracheotomy set readily available.
C. Frequent assessment of respiratory status.

**Goal:** To prevent complications of cardiac problems.
A. Assess client for history of cardiac problems.
B. Assess frequently for dysrhythmias.
C. Calcium will potentiate effects of digitalis; use cautiously together.

**Physiology of the Pancreas**

A. The pancreas is located in the upper left aspect of the abdominal cavity.
B. The pancreas produces the enzymes trypsin, amylase, and lipase, which are secreted into the duodenum and are necessary for the digestion and absorption of nutrients.
C. Located within the pancreas are the islets of Langerhans, which contain beta cells that are responsible for the production of insulin.
D. Insulin is necessary for maintaining normal carbohydrate metabolism and glucose utilization.
System Assessment

A. Evaluate changes in weight, particularly weight gain in an adult and weight loss in a child.
B. Evaluate alterations in fluid balance.
C. Evaluate changes in mental status.
D. Evaluate serum glucose levels.
E. Evaluate results of pancreatic enzyme studies.
F. Evaluate the abdomen for epigastric pain and abdominal discomfort.

DISORDERS OF THE PANCREAS

Diabetes Mellitus

Diabetes mellitus (DM) is a complex, multisystem disease characterized by the absence of, or a severe decrease in, the secretion or utilization of insulin.

A. Pathophysiology.
1. The primary function of insulin is to facilitate the movement of glucose from the blood into the cell, thus decreasing the blood glucose level.
   a. Necessary for the transport of glucose into the cells of the liver, muscles, and other tissues.
   b. Regulates the rate of carbohydrate metabolism and conversion to glucose; normally insulin is decreased during fasting and increased after eating (prandial).
2. Insulin is secreted by the beta cells in the islets of Langerhans in the pancreas.
3. Insulin allows the body to use carbohydrates more effectively for conversion of glucose for energy.
   a. Adequate intake of carbohydrates.
   b. Available insulin to facilitate the movement of glucose into cells.
   c. Adequate reserves of glucagon (released by pancreas; converts glycogen to glucose in the liver).
4. If carbohydrates are not available to be used for energy, cells will begin to oxidize the fats and protein stores.
   a. Breakdown of fat results in the production of ketone bodies.
   b. Protein is wasted during insulin deficiency. Protein is broken down and converted to glucose by the liver, thus contributing to the increase in circulating glucose.
   c. When fats are used as the primary energy source, the serum lipid level rises and contributes to the accelerated development of atherosclerosis.
5. When circulating glucose cannot be used for energy, the level of serum glucose will increase (hyperglycemia).
   a. Hyperglycemia will cause an increase in the osmotic gradient; water moves out of the cells into the circulating volume to decrease the osmolarity. This results in an increase in urinary output.
   b. The increase in circulating glucose exceeds the renal threshold, and glucose spills into the urine.
6. Pathophysiologic bases for symptoms.
   a. Polyuria: because of the increased serum osmolarity, there is more circulating volume; water is not reabsorbed from the renal tubules, and there is a significant increase in urine output.
   b. Polydipsia: increased loss of fluids precipitates dehydration, causing thirst.
   c. Polyphagia: tissue breakdown and wasting cause hunger.
   d. Weight loss (with type 1 DM): glucose is not available to the cells; body begins to break down fat and protein stores for energy.

B. Classification.
1. Type 1: absolute lack of insulin secretion.
   a. Absence of insulin production; client is dependent on insulin to prevent ketoacidosis and maintain life.
   b. Onset is frequently in childhood; most often diagnosed before the age of 18 years. Most common age range is 10 to 15 years.
   c. Previously called juvenile diabetes or insulin-dependent diabetes mellitus.
   d. Familial tendencies in transmission.
   e. Client will have type 1 diabetes for the rest of his or her life.
2. Type 2: combination of insulin resistance and inadequate insulin secretion to compensate.
   a. Insulin deficiency caused by defects in insulin production or by excessive demands for insulin; client is not dependent on insulin.
   b. Ketoacidosis is generally not a problem because of limited insulin production.
   c. Onset is predominately in adulthood, generally after the age of 40 years, but it may occur at any age.
   d. Previously called adult onset diabetes (AODM) or noninsulin-dependent diabetes mellitus (NIDDM).
   e. Associated with obesity; overweight people require more insulin.
   f. If not associated with obesity, there is usually a strong family history.
   g. Blood sugar often controlled by diet and oral hypoglycemics but during episodes of stress may require insulin for control.
   a. Develops during pregnancy; usually detected at 24 to 28 weeks’ gestation by oral glucose tolerance test.
   b. Glucose tolerance usually returns to normal soon after delivery.
   c. Infant may be large for gestational age and may experience hypoglycemia shortly after birth.
   d. Commonly occurs again in future pregnancies; client is at increased risk for development of glucose intolerance and type 2 diabetes later in life.
Assessment

A. Clinical manifestations.
   1. Types 1 and 2.
      a. Three Ps: polyphagia, polydipsia, polyuria.
      b. Fatigue.
      c. Increased frequency of infections.
   2. Type 1.
      a. Weight loss, excessive thirst.
      b. Bed-wetting, blurred vision
      c. Complaints of abdominal pain.
      d. Rapid onset, generally over days to weeks.
   3. Type 2 (most clients asymptomatic first 5 to 10 years).
      a. Weight gain (obese), visual disturbances.
      b. Slow onset; may occur over months.
      c. Onset usually after the age of 40 years; peaks around 45 to 50 years.
      d. Fatigue and malaise.
      e. Recurrent vaginal yeast or monilia infections—frequently, this is initial symptom in women.
      f. Older adult assessment considerations (Box 13-1).

B. Diagnostics (the criteria for diagnosis are two or more abnormal test results with two or more values outside the normal range) (see Appendix 13-1).
   1. Fasting blood glucose level: above 126 mg/dL (normal glucose below 100 mg/dL).
   2. Glucose tolerance test: 2-hour glucose values are greater than 200 mg/dL.
   3. Random glucose >200 mg/dL with symptoms (three Ps, weight loss).
      a. Impaired glucose tolerance (IGT): 2 hours after a meal, plasma glucose is >140 mg/dL; 200 mg/dL or higher during an oral glucose tolerance test.
      b. Impaired fasting glucose (IFG): fasting blood glucose is greater than 100 mg/dL, but less than 126 mg/dL.
   5. Glycosylated hemoglobin (HbA1c) is increased (less than 7% is considered good control; HbA1c is not a test to diagnose diabetes.)

Box 13-1 OLDER ADULT CARE FOCUS

Diabetic Assessment Considerations and Care

- Determine mental status and manual dexterity to handle injections.
- Determine whether client can access the injection sites.
- Is client alert and mentally capable of making judgments on medications?
- Determine whether the client can pay for supplies.
- What is the client’s attitude about needles and injections?
- Assess how many other medications the client is taking; problems with “polypharmacy” (too many medications).
- Determine family’s or client’s ability to accurately perform serum glucose testing.
- What is the client’s support system?

1 NURSING PRIORITY The glycosylated hemoglobin level will indicate overall glucose control for approximately the past 120 days. This allows evaluation of control of the blood glucose level, regardless of increases or decreases in the blood glucose level immediately before the sample was obtained.

Treatment

A. Factors in diabetes management.
   1. Regular physical activity.
   2. Diet.
   3. Pharmacologic intervention.

B. Hypoglycemic agents.
   1. Insulin: may be used in both types of diabetes.
      Primary function of insulin is to transport glucose into muscle and fat cells (Table 13-3).
      a. Combination premixed insulin therapy eliminates problem of mixing different types (example: NPH/regular 70/30—number refers to percentage of each type of insulin).
      b. Do not give mixtures intravenously.
      c. Response to insulin mixtures varies with individuals.
      d. Insulin will be used for glucose control during pregnancy.
   2. Oral hypoglycemic agents for noninsulin-dependent clients (see Appendix 13-2).
   3. Insulin requirement increases when:
      a. Client is seriously ill.
      b. An infection develops.
      c. Client experiences physical trauma (surgery or accidents).
      d. Growth spurts occur during adolescence and puberty.
   4. Diabetic diet.
      a. Decrease calories for weight loss.
      b. Diet to meet nutritional needs and maintain optimum glucose level. Avoid simple sugars.
      c. Decrease in cholesterol level.
      d. Meal plan.
         (1) 45% to 65% carbohydrate (low glycemic index foods and increased dietary fiber).
         (2) 25% to 30% fat (reduced saturated and trans fat foods).
         (3) 10% protein (significantly lower than for normal adult due to the stress that moderate to high protein places on the kidneys).
   5. Exercise.
      a. Planned exercise; sporadic exercise is discouraged.
      b. Glucose for exercising muscles comes from the liver.
      c. The counterregulatory hormones increase the delivery of glucose to the muscles.

ALERT Intervene to control symptoms of hypoglycemia or hyperglycemia.
**Table 13-3**  PROFILE OF INSULINS

<table>
<thead>
<tr>
<th>Insulin</th>
<th>Nursing Implications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rapid-Acting</strong></td>
<td>Should be used in combination with longer-acting insulin.</td>
</tr>
<tr>
<td><strong>NURSING PRIORITY</strong> Because of quick onset of action, client must eat immediately.</td>
<td></td>
</tr>
<tr>
<td><strong>Short-Acting</strong></td>
<td>Usually given 20-30 minutes before meals. May be given alone or in combination with longer-acting insulins. Given for sliding scale coverage.</td>
</tr>
<tr>
<td><strong>NURSING PRIORITY</strong> When administering injections:</td>
<td></td>
</tr>
<tr>
<td>• May mix regular insulin with other insulins.</td>
<td></td>
</tr>
<tr>
<td>• Only regular insulin may be given IV.</td>
<td></td>
</tr>
<tr>
<td><strong>Intermediate-Acting</strong></td>
<td>Hypoglycemia tends to occur in mid to late afternoon. Never give IV. May be mixed with regular insulin.</td>
</tr>
<tr>
<td><strong>Long-Acting</strong></td>
<td>Glargine has low pH (4); CANNOT be mixed with other insulins. Usually given once a day at bedtime, but can be administered during the day.</td>
</tr>
<tr>
<td><strong>ALERT</strong> Intervene to control hypoglycemia/hyperglycemia. Know various insulins and nursing implications. Specifically, know when to anticipate reaction and what to teach the client about his or her insulin.</td>
<td></td>
</tr>
</tbody>
</table>


**Complications of Insulin Therapy**

A. Hypoglycemia (Table 13-4).

B. Lipoatrophy and lipohypertrophy.
   1. Associated with administration of cold insulin.
   2. May result from poor rotation of injection sites.

3. Use of human insulin greatly decreases the chance of this occurring.

C. Somogyi effect (Figure 13-3).
   1. Physiologic reflex: rebound hyperglycemia from an unrecognized hypoglycemic state.
   2. Most often occurs at night.
   3. May be treated by decreasing the evening insulin dose or by increasing the calories in the bedtime snack.

D. Dawn phenomenon (Figure 13-4).
   1. Results from nighttime release of growth hormone and cortisol.
   2. Blood glucose elevates at 5:00 to 6:00 a.m. (predawn hours).
**Table 13-4** COMPARISON OF DIABETIC KETOACIDOSIS (DKA), HYPEROSMOLAR HYPERGLYCEMIA SYNDROME (HHS), AND HYPOGLYCEMIA

<table>
<thead>
<tr>
<th></th>
<th><strong>DKA</strong></th>
<th><strong>HHS</strong></th>
<th><strong>Hypoglycemia</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td>All ages, increased incidence in children.</td>
<td>Adult (usually seen in older adults with underlying chronic disease)</td>
<td>All ages</td>
</tr>
<tr>
<td><strong>GI</strong></td>
<td>Abdominal pain, anorexia, nausea, vomiting, diarrhea</td>
<td>Normal</td>
<td>Normal; may be hungry</td>
</tr>
<tr>
<td><strong>Mental state</strong></td>
<td>Dull, confusion increasing to coma</td>
<td>More severe neurologic symptoms due to increased osmolarity and high blood glucose level</td>
<td>Difficulty in concentrating, coordinating; eventually coma</td>
</tr>
<tr>
<td><strong>Skin temperature</strong></td>
<td>Warm, dry, flushed</td>
<td>Warm, dry, flushed</td>
<td>Cold, clammy</td>
</tr>
<tr>
<td><strong>Pulse</strong></td>
<td>Tachycardia, weak</td>
<td>Tachycardia</td>
<td>Tachypnea</td>
</tr>
<tr>
<td><strong>Respirations</strong></td>
<td>Initially deep and rapid; lead to Kussmaul respirations</td>
<td>Tachypnea</td>
<td>Shallow</td>
</tr>
<tr>
<td><strong>Breath odor</strong></td>
<td>Fruity, acetone</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Urine output</strong></td>
<td>Increased</td>
<td>Increased</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Lab values:</strong></td>
<td><strong>Glucose</strong></td>
<td>Greater than 600 mg/dL</td>
<td>Below 70 mg/dL</td>
</tr>
<tr>
<td></td>
<td>Greater than 300 (up to 1500 mg/dL)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Ketones</strong></td>
<td>High/large</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>pH</strong></td>
<td>Acidotic (less than 7.3)</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Hematocrit</strong></td>
<td>High due to dehydration</td>
<td>High due to dehydration</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Lab values:</strong></td>
<td><strong>Sugar</strong></td>
<td>High</td>
<td>Negative</td>
</tr>
<tr>
<td></td>
<td>High</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Ketones</strong></td>
<td>High</td>
<td>High</td>
<td>Negative</td>
</tr>
<tr>
<td><strong>Onset</strong></td>
<td>Rapid (less than 24 hr)</td>
<td>Slow (over many days)</td>
<td>Rapid</td>
</tr>
<tr>
<td><strong>Classification of diabetes</strong></td>
<td>Primarily type 1; type 2 in severe distress</td>
<td>Type 2</td>
<td>Type 1 and type 2</td>
</tr>
</tbody>
</table>

*DKA,* Diabetic ketoacidosis; *GI,* gastrointestinal; *HHS,* hyperosmolar hyperglycemia nonketotic syndrome.

3. May be treated by increasing insulin for overnight period.

4. Most severe in adolescence and young adulthood with peak growth hormone.

E. Hormones that counteract insulin.
1. Glucagon.
2. Epinephrine.
3. Cortisol.
4. Growth hormone.

**NURSING PRIORITY** Intensive control of blood glucose levels in clients with type 1 diabetes can prevent or ameliorate many of the complications.

**Complications Associated with Poorly Controlled Diabetes**

A. Diabetic ketoacidosis.

1. An extreme increase in the hyperglycemic state.
2. An increase in the mobilization of fat and protein as energy sources.
3. Metabolism of fat results in the production of fatty acids, which are converted to ketone bodies.
4. An increase in circulating ketone bodies precipitates the state of acidosis.
5. Occurs predominately in type 1 diabetes.
B. Clinical manifestations of diabetic ketoacidosis (see Table 13-4).
   1. Onset.
      a. May be acute or occur over several days.
      b. May result from stress, infection, surgery, or lack of effective insulin control.
      c. Results from poorly controlled diabetes.
   2. Severe hyperglycemia (blood glucose levels of 300-800 mg/dL).
   3. Presence of metabolic acidosis (low pH [6.8-7.3] and serum bicarbonate level less than 15 mEq/L).
   4. Hyperkalemia, hypokalemia, or normal potassium level, depending on amount of water loss.
   5. Urine ketone and sugar levels are increased.
   6. Excessive weakness, increased thirst.
   7. Nausea, vomiting.
   8. Fruity (acetone) breath.
   10. Decreased level of consciousness.
   11. Dehydration.
   12. Increased temperature caused by dehydration.
C. Hyperglycemic-hyperosmolar state (HHS); also known as nonketotic hyperosmolar coma and/or hyperglycemic hyperosmolar nonketotic syndrome (HHNS).
   1. Occurs primarily in older adult clients with type 2 diabetes.
   2. Insulin production is adequate to prevent the breakdown of fat for cellular function, but severe hyperglycemia exists.
   3. Severe electrolyte imbalance and dehydration exist in the absence of the acidic state.

4. Characterized by extreme hyperglycemia (values greater than 600 mg/dL).
5. Because of the ability of the body to maintain a low level of insulin production, the breakdown of fat and the production of ketone bodies do not occur. This prevents the client from developing acidosis.
6. Osmotic diuresis occurs as a result of the hyperglycemia; the client becomes dehydrated very rapidly.

D. Clinical manifestations of HHS (see Table 13-4).
   1. Warm, flushed skin, lethargy.
   2. Weakness, thirst, decreased level of consciousness.
   3. Increased temperature from dehydration.
   4. Tachycardia, decrease in blood pressure.
   5. Generally no GI problems.
   6. No acetone odor to breath or glycosuria.
   7. Severe hyperglycemia (greater than 600 mg/dL).
   8. Serum pH is normal; urine output is increased.
E. Electrolyte imbalance.
   1. As acidosis develops, potassium ions move out of cells; this leaves cellular potassium levels depleted, but the serum potassium level is usually normal because of excessive excretion.
   2. As osmotic diuresis occurs from the hyperglycemic/hyperosmolar state, serum potassium is excreted.
   3. As the client becomes more dehydrated, the serum potassium becomes concentrated and does not reflect the potassium loss.
   4. As the osmolality and acidosis are corrected, along with the administration of insulin, the potassium will move back into the cells and result in severe hypokalemia.

Complications of Long-Term Diabetes
A. Angiopathy: premature degenerative changes in the vascular system.
   1. May affect the large vessels (macroangiopathy, early onset of atherosclerosis, and arteriosclerotic vascular problems).
   2. May affect the small vessels (microangiopathy); problems are specific to diabetes.
B. Peripheral vascular disease: combination of both types of angiopathy.
C. Hypertension.
D. Diabetic gastroparesis: delayed gastric emptying.
E. Cerebrovascular disease.
F. Coronary artery disease.
G. Ocular complications: retinopathy, cataracts, glaucoma.

¡ PEDIATRIC PRIORITY An ophthalmologic examination should be obtained once the child is 10 years of age and has had diabetes for 3-5 years.

H. Nephropathy.
   1. Microangiopathy primarily affects the glomerular capillaries, resulting in thickening and increased permeability of the glomerular basement membrane; will progress to end-stage renal disease.
2. Recurrent pyelonephritis, particularly in women.
3. Diabetic effects on the kidneys are the single most common cause of end-stage renal failure.

I. Neuropathy: inadequate blood supply to the nerve tissue and high blood glucose levels cause metabolic changes within the neurons.
1. Peripheral neuropathy: may be general pain and tingling; may progress to painless neuropathy.

2. Autonomic nerve damage: diarrhea or constipation, urinary incontinence or retention, decreased sweating, and orthostatic hypotension; impotence in men. Interferes with the client’s ability to recognize episode of hypoglycemia.
3. Approximately 60% of clients with diabetes experience neuropathy; it is the most common chronic complication.

J. Infections: An alteration in immune system response results in impairment of white cells for phagocytosis. Persistent glycosuria potentiates urinary tract infections.

Clinical Implications of Diabetes in Pregnancy

A. Effects of pregnancy on diabetes.
1. During the first trimester of pregnancy, there is an increase in the fetal need for glucose and amino acids; this lowers the maternal blood glucose level and decreases the maternal need for additional insulin.
2. During the second and third trimesters, the need for insulin will increase as a result of insulin resistance from major hormone changes.
3. Oral hypoglycemic agents are not used to control diabetes in the pregnant client.

B. Effects of diabetes on pregnancy.
1. Increase in size and number of islets of Langerhans in the client with type 2 diabetes.
2. Increased tendency toward the development of metabolic acidosis caused by an increase in metabolic rate.
3. Placental antagonist to insulin will decrease the effectiveness of insulin.
4. Fetal antagonists to insulin decrease the utilization of glucose.
5. Hormonal changes lead to decreased tolerance to glucose and increased insulin resistance (begins around 14 to 16 weeks).

C. Influence of pregnancy on diabetic control.
1. Prone to hypoglycemia during the first trimester of pregnancy; may need to reduce insulin dosage.
2. Increase in insulin requirements during the second and third trimesters of pregnancy may double or quadruple prepregnancy amounts.
3. Tendency to intensify the existing complications of diabetes.

D. Monitoring fetal and maternal well-being during pregnancy, labor, and delivery.
1. Antepartum period.
   a. Assess stability of mother’s blood glucose control; insulin used rather than oral hypoglycemics.
   b. Weekly or biweekly blood glucose levels to maintain optimum level of control.
   c. Third-trimester evaluation of fetal/placental function by determining 24-hour urinary estriol levels.
   d. Nonstress or oxytocin challenge test (stress test) done at 36 to 38 weeks’ gestation to determine ability of fetus to withstand stress.
   e. Amniocentesis to obtain fluid samples to determine lecithin/sphingomyelin ratio.

2. Intrapartum period.
   a. As long as there is evidence of adequate placental function and the infant’s response to stress is appropriate, the pregnancy is allowed to progress to term, with an anticipated vaginal delivery.
   b. During labor, blood glucose levels are maintained with IV glucose and regular insulin.
   c. Fetal monitoring during labor.

3. Postpartum period.
   a. Endocrine and metabolic changes will occur rapidly after delivery.
   b. Insulin requirements for mother will be markedly decreased and will fluctuate over next few weeks.
   c. Mother must go through a period of diabetic re-regulation.

E. Presence of diabetes predisposes the client to an increased incidence of:
1. Pregnancy-induced hypertension.
2. Hemorrhage.
3. Polyhydramnios (increase in volume of amniotic fluid >2000 mL).
4. Vaginal and urinary tract infections.
5. Premature delivery.
6. Intrauterine death in third trimester.
7. Compromised newborn.
   a. Respiratory distress syndrome.
   b. Hypoglycemia.
   c. Hyperbilirubinemia.
   d. Congenital anomalies: directly associated with the degree of maternal hyperglycemia in the first trimester.

Nursing Interventions (All Types)

1. Normal fasting blood glucose 110-115 mg/dL. 2. Two hours after meals or after glucose load, blood glucose is no higher than 140 mg/dL. 3. Client is in good general health and is of normal weight. 4. Glycosylated hemoglobin is less than 7%.

Goal: To return serum glucose to normal level.
A. Initially administer regular insulin on a proportional basis according to need (Box 13-2).
ALERT  Determine ability of family/support systems to provide care for client. Identify client’s and family’s strengths.

Goal: To plan and implement a teaching regimen.
A. Assess current level of knowledge regarding diabetes.
B. Evaluate cultural and socioeconomic parameters.
C. Evaluate client’s support system (family, significant others).
D. Instruct regarding sick-day guidelines (Box 13-3).

### Box 13-2 IMPLICATIONS IN THE ADMINISTRATION OF INSULIN

1. Do not administer cold insulin; it increases pain and causes irritation at injection site.
2. An open 10-mL vial of unrefrigerated insulin should be discarded after 30 days, regardless of how much was used.
3. Do not allow insulin to freeze, and keep it away from heat and sunlight.
4. Insulin pens (NPH and 70/30) should be discarded after 1 week of storage at room temperature. Regular cartridges, which do not contain preservatives, may be left unrefrigerated for up to 1 month.
5. Extreme temperatures (less than 36°F or greater than 86°F) should be avoided.
6. Roll the vial between the palms of the hands to decrease the risk for inconsistent concentration of insulin.
7. The abdomen is the primary site for subcutaneous injections of insulin. Rotate injection sites; injection sites should be 1 inch apart.
8. Abdomen area provides most rapid insulin absorption.
9. Use only insulin syringes to administer insulin.
10. Check expiration date on insulin bottle.
11. When drawing up regular insulin with a long-acting insulin, draw up the regular (clear) insulin before the longer-acting (cloudy) insulin.
12. Regular insulin is used for administration by sliding scale and periods when blood glucose is unstable and difficult to control.
13. Using alcohol to cleanse the skin before injection is not recommended for home care. If used, hold alcohol pad in place for a few seconds but do not massage.
15. Check dose with another nurse prior to administering.

B. Administer insulin 30 minutes before a meal or snack; do not administer insulin if there is no carbohydrate intake.
C. Maintain adequate fluid intake.
D. Evaluate serum electrolyte levels.
   1. Do not administer potassium unless client is voiding or if urine output begins to drop.
   2. Generally begin potassium replacement within 1 to 2 hours after starting insulin therapy.
   3. Serum potassium levels will be misleading if the client is dehydrated or in a state of ketoacidosis.
E. Evaluate hydration status.
F. Evaluate for clinical manifestations of hypoglycemia and hyperglycemia.

### Box 13-3 DIABETIC “SICK DAY” GUIDELINES

**If you do not feel well (not eating regularly or have fever, lethargy, nausea and vomiting, etc.):**

1. Check your blood glucose every 3 to 4 hours and urine ketones when voiding.
2. Increase your intake of fluids that are high in carbohydrates; every hour, drink fluids that replace electrolytes: fruit drinks, sports drinks, regular soft drinks (not diet beverages).
3. If you cannot eat and you have replaced four to five meals with liquids, notify your health care provider.
4. Get plenty of rest; if possible, have someone stay with you.
5. Do not omit or skip your insulin injections or oral medications unless specifically directed to do so by your health care provider.
6. Follow your health care provider’s instructions regarding blood glucose levels and insulin or oral hypoglycemic agents.
7. Stay warm, stay in bed, and do not overexert yourself.
8. Call your health care provider when:
   a. You have been ill for 1 to 2 days without getting any better.
   b. You have been vomiting or had diarrhea for more than 6 hours.
   c. Your urine self-testing shows moderate to large amounts of ketones.
   d. You are taking insulin and your blood glucose level continues to be greater than 240 mg/dL after you have taken two to three supplemental doses of regular insulin (prearranged with your provider).
   e. You are taking insulin and your blood glucose level is less than 60 mg/dL.
   f. You have type 2 diabetes, you are taking oral diabetic medications, and your premeal blood glucose levels are 240 mg/dL or greater for more than 24 hours.
   g. You have signs of severe hyperglycemia (very dry mouth or fruity odor to breath), dehydration, or confusion.
   h. You are sleepier or more tired than normal.
   i. You have stomach or chest pain or any difficulty breathing.
   j. You have any questions or concerns about what you need to do while ill.

**ALERT**
FIGURE 13-5 Sites used for insulin injection. The injection site can affect the onset, peak, and duration of action of the insulin. Insulin injected into the abdomen (area I) is absorbed fastest, followed by insulin injected into the arm (area II) and the leg (area III). (From Black JM, Hawks JH: Medical-surgical nursing: clinical management for positive outcomes, ed 8, Philadelphia, 2009, Saunders.)

FIGURE 13-6 Insulin pump. (From Black JM, Hawks JH: Medical-surgical nursing: clinical management for positive outcomes, ed 8, Philadelphia, 2009, Saunders.)

b. Delivers continuous infusion of short-acting insulin over a 24-hour period, allowing for tight glucose control.
c. Can deliver bolus of insulin based on excessive carbohydrates ingested.
d. Monitor insertion site for redness and swelling.
8. Disposable needles and syringes may be used for up to 3 days. Good handwashing is critical; needle should be recapped and stored in refrigerator to decrease bacterial growth. (Note: Reusing needles is controversial; today’s needles are much thinner and can bend to form a hook with just one injection, in which case, future use causes more tissue damage. Have client consult with his or her health care provider about this issue.)

9. Insulin pen is a compact portable device that is loaded with insulin; need to change needle with each injection.

F. Oral hypoglycemic agents.
1. Take medication as scheduled; do not skip or add doses.
2. Signs and symptoms of hypoglycemia.
3. Anticipate change in medication with pregnancy.

G. Monitoring blood glucose.
1. Self-monitoring of blood glucose (SMBG)—not necessary to use alcohol to cleanse site.
2. Use side of finger pad rather than near the center. If alternative site is used (i.e., forearm), may require different equipment.
3. Need only a large drop of blood.

H. Exercise.
1. Establish an exercise program.
2. Avoid sporadic exercise.
3. Review instructions regarding adjustment of insulin and food intake to meet requirements of increased activity.
4. Extremities involved in activity should not be used for insulin injection (e.g., arms when playing tennis).

I. Diet (Box 13-4).
1. Regularly scheduled mealtimes.
2. Understanding of food groups and balanced nutrition.
3. Incorporate family tendencies and cultural patterns into prescribed dietary regimen.
4. Provide client and family with written instructions regarding dietary needs.

J. Infection control.
2. Insulin requirements may increase with severe infections.

Box 13-4 OLDER ADULT CARE FOCUS

Guidelines for Food Selection

- Avoid canned fruits that are in heavy syrup; select fruit packed in water.
- Include fresh fruits and vegetables and whole-grain cereals and breads to provide adequate dietary fiber to prevent constipation.
- Avoid casseroles, fried foods, sauces and gravies, and sweets.
- Fats (oils, margarines) that are liquid at room temperature are better than those that are solid.
- Read food labels; remember that the highest-content ingredient is listed first.
- Select foods in which the majority of calories do not come from a fat source.
3. Increased problems with vaginitis, urinary tract infections, and skin irritation.

K. Avoid injury.
   1. Decreased healing capabilities, especially in lower extremities.
   2. Maintain adequate blood supply to extremities; avoid tight-fitting clothing around the legs.
   3. Proper foot care (see Chapter 16).

**Goal:** To prepare the client with diabetes for surgery.

A. Obtain base serum laboratory values for postoperative comparison.
B. Surgery should be scheduled in early morning to decrease problems with diet and insulin replacement.
C. Oral hypoglycemic agents should not be given the morning of surgery.
D. For clients with NPO (nothing by mouth) status who require insulin, an IV of 5% dextrose in water (D 5W) is frequently started.
E. Obtain a blood glucose reading about an hour before sending the client to surgery to make sure he or she is not developing hypoglycemia.

**NURSING PRIORITY** Evaluate intake; do not give insulin to a client on NPO status unless an IV is in place.

**Goal:** To maintain control of diabetic condition in the client who has had surgery.

A. IV fluids and regular insulin until client is able to take fluids orally.
B. Obtain blood glucose level four to six times a day to determine fluctuations.
C. After glucose levels stabilize, the client can usually resume taking his or her preoperative diabetic medication.
D. Observe for fluctuation of blood glucose immediately after surgery.
E. Avoid urinary bladder catheterization.
F. Evaluate peripheral circulation and prevent skin breakdown.
G. Assess for development of postoperative infections.

**Goal:** To identify diabetic ketoacidosis and help client return to homeostasis.

A. Establish IV access.
B. Anticipate rapid infusion of normal saline solution or plasma expanders initially, then at a maintenance rate. Administer with caution to clients with cardiac conditions.
C. Administer insulin: use IV drip (regular insulin only) during the acute phase; then administer subcutaneously as blood glucose level begins to decrease.
D. Frequent monitoring of vital signs.
E. Frequent serum glucose checks.
F. Hourly urine measurements: do not administer potassium if urine output is low or dropping.
G. Monitor blood glucose levels frequently (normally every hour).
H. Cardiac monitor to determine presence of dysrhythmias secondary to potassium levels.

I. Monitor serum electrolyte levels, particularly potassium levels.
   1. Hyperkalemia may occur initially in response to the acidosis.
   2. Hypokalemia occurs about 4 to 6 hours after treatment, as the acidosis is resolving.
J. Evaluate acid-base status.

**Goal:** To identify HHS and to help client return to homeostasis.

A. Establish IV access.
B. IV infusion to rehydrate client; normal saline solution is frequently used; closely monitor hydration status.
C. Low-dose insulin given intravenously at first to decrease blood glucose level slowly.
D. Evaluate urine output.
E. Monitor serum glucose level.
F. Evaluate acid-base status.
G. Assess client for presence of other chronic health problems.

**Home Care**

A. Maintain optimum weight.
B. Continue to receive long-term medical care.
C. Notify all health care providers of diagnosis of diabetes; wear medical alert identification.
D. Recognize problems of the cardiovascular system.
   1. Peripheral vascular disease.
   2. Decreased healing.
   3. Increased risk for stroke.
   4. Increased risk for myocardial infarction.
   5. Presence of retinopathy.
   6. Increased risk for renal disease.
E. Recognize problems of peripheral neuropathy.
F. Help client understand problems that diabetes imposes on pregnancy and the subsequent development of a high-risk pregnancy.
G. Help client understand the problem of increased susceptibility to infections.

**Goal:** To help the client with diabetes maintain homeostasis throughout pregnancy.

A. Prevent infection.
B. Frequent evaluation of glucose levels and monitoring of changes in insulin requirements.
C. Utilization of regular insulin rather than oral hypoglycemic agents.
D. Maintain optimum level of weight gain; labor may be induced, or cesarean delivery may be required if complications are evident.

**Goal:** To help the client with diabetes maintain homeostasis throughout labor and delivery.

A. IV D 5W, Ringer’s lactate, or D 5 Ringer’s lactate to maintain homeostasis during labor, along with continuous infusion of insulin.
B. Fetal monitoring to identify early stages of fetal distress.
C. X-ray pelvimetry to identify cephalopelvic disproportion.
D. Increased incidence of dystocia because of large infants.
E. Frequent evaluation of serum glucose levels (every 2 to 3 hours).

**Goal:** To help the client with diabetes return to homeostasis during the postpartum period.

A. Anticipate fluctuation in insulin requirements caused by:
   1. Loss of fetal insulin.
   2. Removal of placental influence on insulin.
   3. Changes in metabolic activity.

B. Observe for and monitor for rapid fluctuations in serum glucose levels.

C. Prevent postpartum infection.

**Hypoglycemia (Insulin Reaction)**

Hypoglycemia is a condition characterized by a decreased serum glucose level, which results in decreased cerebral function.

**Assessment**

A. Risk factors.
   1. Poorly controlled diabetes.
      a. Too little food.
      b. Increase in exercise without adequate food.
      c. Increase in insulin intake.
   2. Excessive alcohol intake with poor nutrition.
   3. Gastroenteritis or gastroparesis, which may impede absorption of food.
   4. Reflex action of insulin caused by increased carbohydrate intake (Somogyi effect).

B. Clinical manifestations.
   1. Lability of mood.
   2. Emotional changes, confusion.
   3. Headache, lightheadedness, seizures, coma.
   4. Impaired vision.
   5. Tachycardia, hypotension.
   7. Diaphoresis.

C. Diagnostics.
   1. Serum glucose below 70 mg/dL.
   2. Negative urine acetone test result.
   3. Normal pH.

**Treatment**

A. Carbohydrates by mouth if client is alert and can swallow.
   1. Milk preferred in children with a mild reaction; it provides immediate lactose, as well as protein and fat for prolonged action.
   2. Simple sugars for immediate response: orange juice, honey, candy, glucose tablets.
   3. If simple carbohydrates are taken to increase blood glucose, client should plan on eating protein or complex carbohydrates to prevent rebound hypoglycemia.

B. Glucagon can be given intravenously if client is unconscious.

**Nursing Interventions**

**Goal:** To increase serum glucose level.

A. Glucose/carbohydrate preparations as indicated.

**Pancreatitis**

Pancreatitis is an inflammatory condition of the pancreas.

A. Acute: characterized by an acute inflammatory process; problems range from mild edema to severe hypotension and severe hemorrhagic necrosis.

B. Chronic: characterized by progressive destruction and fibrosis of the pancreas; condition may follow acute pancreatitis but may also occur alone.

**Assessment**

A. Risk factors/etiology.
   1. Biliary tract obstructive disease, causing reflux of bile secretions.
   2. Alcohol intake precipitating an increase in the secretion of pancreatic enzymes.
   3. Hyperlipidemia is a common cause.
   4. Trauma.
   5. Certain drugs (thiazide diuretics, NSAIDs, estrogens, steroids, salicylates).

B. Clinical manifestations.
   1. Severe constant midepigastric pain.
      a. Radiates to the back or flank area.
      b. Exacerbated by eating.
   2. Acute.
      a. Persistent vomiting.
      b. Low-grade fever.
      c. Hypotension and tachycardia.
      d. Jaundice, if common bile duct is obstructed.
      e. Abdominal distention.
      f. Cullen’s sign: periumbilical discoloration.
      g. Grey Turner’s sign: flank ecchymosis.
   3. Chronic.
      a. Decrease in weight.
      b. Mild jaundice.
      c. Steatorrhea.
      d. Abdominal distention and tenderness.
      e. Hyperglycemia.
C. Diagnostics.
   1. Increase in serum amylase (hallmark) and lipase levels.
   2. Increase in urine amylase level.
   3. Hyperglycemia.
   4. Leukocytosis.
   5. Elevated C reactive protein.
   6. Hypocalcemia.
   7. Ultrasonography.
   8. Computed tomography scan.

Treatment
A. Medications.
   1. Analgesics.
   2. Antibiotics.
B. Decrease pancreatic stimulus.
   1. NPO status; IV fluids.
   2. Nasogastric suction.
   4. Diet: (if not NPO) low-fat, high-carbohydrate.
C. Surgical intervention to eliminate precipitating cause (biliary tract obstruction).

Nursing Interventions
Nursing interventions are the same for the client with acute pancreatitis and for the client with chronic pancreatitis experiencing an acute episode.

Goal: To relieve pain.
A. Administer analgesics; pain control is essential (restlessness may cause pancreatic stimulation and further secretion of enzymes).
B. Place client in side lying position with knees drawn up to chest or in semi-Fowler’s position with knees flexed toward the chest.
C. Evaluate precipitating cause.
Goal: To decrease pancreatic stimulus.
A. Bed rest.
B. Maintain NPO status initially.
C. Maintain nasogastric suctioning.
D. Small frequent feedings when food is allowed.
E. Pain control.
Goal: To prevent complications.
A. Identify electrolyte imbalances, especially hypocalcemia.
B. Maintain adequate hydration.
C. Maintain respiratory status; problems occur because of pain and ascites.
D. Assess for hypoglycemia and development of diabetes.

Home Care
A. Avoid all alcohol intake.
B. Know signs of hyperglycemia and development of diabetes; understand when to return for evaluation of blood glucose level.
C. Bland diet, low in fat, high in carbohydrates (protein recommendations vary).
D. Replacement of pancreatic enzymes.

Cancer of the Pancreas
The majority of tumors occur in the head of the pancreas. As tumors grow, the bile ducts are obstructed, causing jaundice. Tumors in the body of the pancreas frequently do not cause symptoms until growth is advanced. Cancer of the pancreas has a poor prognosis.

Assessment
A. Risk factors/etiology.
   1. Incidence is higher in men 45 to 65 years of age.
   2. History of chronic pancreatitis is common.
   3. Cigarette smoke (3 to 4 times more common among smokers)
B. Clinical manifestations.
   1. Dull, aching abdominal pain.
   2. Ascites.
   3. Nausea, vomiting.
   4. Anorexia and progressive weight loss.
   5. Jaundice.
   7. Dark, frothy urine.
C. Diagnostics.
   1. Increased carcinoembryonic antigen (CEA) level.
   2. Ultrasonography.
   3. CT scan.

Treatment
A. Surgery: Whipple’s procedure (radical pancreatic duodenectomy).
B. Radiation therapy.
C. Chemotherapy.

Nursing Interventions
Goal: To maintain homeostasis (see Nursing Interventions for pancreatitis).
Goal: To provide preoperative nursing measures if surgery is indicated.
A. Maintain nasogastric suctioning; assess for adequate hydration.
B. Control hyperglycemia.
C. Assess cardiac and respiratory stability.
D. Assess for development of thrombophlebitis.
Goal: To promote comfort, prevent complications, and maintain homeostasis in client who has undergone Whipple’s procedure.
A. General postoperative care (see Chapter 3). Extensive surgical resection of the pancreas and surrounding tissue.
B. Evaluate for hypercoagulable state as well as bleeding tendencies.
C. Monitor for fluctuation in serum glucose levels.
D. Maintain NPO status and nasogastric suction until peristalsis returns.
E. Encourage adequate nutrition when appropriate.
   1. Decrease fats and increase carbohydrates.
   2. Small, frequent feedings.
CHAPTER 13  Endocrine System

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F. Closely observe for development of problems from thromboemboli to coagulation problems; surgery and immobility.

**Home Care**

A. Evaluate for bouts of anxiety and depression caused by severity of illness and prognosis (see Chapter 10).
B. Assist client in setting realistic goals.
C. Encourage ventilation of feelings.
D. Discuss methods for pain control.

**NURSING PRIORITY** Clients experiencing problems of the adrenal medulla have severe fluctuations in blood pressure related to the levels of catecholamines.

**PHYSIOLOGY OF THE ADRENALS**

The adrenal glands are located at the apex of each kidney.

A. Adrenal medulla: secretes catecholamines, epinephrine, and norepinephrine; under the influence of the sympathetic nervous system.

B. Adrenal cortex: main body of the adrenal gland; responsible for the secretion of glucocorticoids, mineralocorticoids, and adrenal sex hormones (androgens and estrogen); adrenal cortical function is essential for life (Table 13-5).

C. Function of the adrenal cortex is controlled by the negative feedback mechanisms regulating hormone release; pituitary gland secretes adrenocorticotropic hormone (ACTH), which in turn regulates hormone release of the adrenal cortex.

**System Assessment**

A. Adrenal medulla.
   1. Evaluate changes in blood pressure.
   2. Assess for changes in metabolic rate.

B. Adrenal cortex.
   1. Evaluate changes in weight.
   2. Evaluate changes in skin color and texture, as well as the presence and distribution of body hair.
   3. Assess cardiovascular system for instability, as evidenced by a labile blood pressure and cardiac output.
   4. Evaluate GI discomfort.
   5. Assess fluid and electrolyte changes from effects of mineralocorticoids and glucocorticoids.
   6. Assess for changes in glucose metabolism.
   7. Assess for changes in reproductive system and in sexual activity.
   8. Evaluate changes in muscle mass.

**Table 13-5  SUMMARY OF ADRENAL HORMONES**

<table>
<thead>
<tr>
<th>Hormone</th>
<th>Site of Action</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenal cortex</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Mineralocorticoids* (aldosterone) | Kidney (renal tubule) | 1. Increased sodium ion absorption.  
2. Decreased potassium absorption.  
3. Conditions causing an increase in aldosterone secretion:  
   a. Decrease in serum sodium.  
   b. Shock.  
Glucocorticoids† (cortisol, hydrocortisone) | All tissues  
Metabolism of all food; enable individual to withstand stress | 1. Increased rate of gluconeogenesis.  
2. Catabolic effect on protein metabolism.  
3. Antiinflammatory effect.  
4. Increased secretion during times of increased body stress.  
Androgens: sex hormones‡ | Stimulate reproduction organs to secrete gonadal hormones | 1. Responsible for secondary sexual characteristics.  
2. Phases of menstrual cycle.  
3. Uterine changes in pregnancy.  
Females: estrogen and progesterone (ovaries)  
Males: testosterone (testes) | | 1. Development and maintenance of secondary sex characteristics.  
2. Spermatogenesis.  
Adrenal medulla§ | Cardiac muscle | 1. Stimulation of sympathetic nervous system.  
2. Produces increase in systolic blood pressure.  
Epinephrine  
Norepinephrine | Organs innervated by sympathetic nervous system | 1. Increases peripheral resistance (i.e., increase in blood pressure). |

*Mineralocorticoids are primarily controlled by the renin-angiotensin-aldosterone system.
†Glucocorticoids are primarily controlled by secretion of adrenocorticotropic hormone from the anterior pituitary.
‡Androgens are primarily controlled by secretions from the pituitary gland.
§Adrenal medulla hormones are primarily controlled by the sympathetic division of the autonomic nervous system.
DISORDERS OF THE ADRENALS

Pheochromocytoma

Pheochromocytoma is a rare disorder of the adrenal medulla characterized by a tumor that secretes an excess of epinephrine and norepinephrine.
A. An increase in these catecholamines produces vasoconstriction, with a precipitant increase in blood pressure, glyco- genolysis, and cardiac workload.
B. Condition may become evident during pregnancy, as increasing uterine pressure precipitates problems.

Assessment
A. Clinical manifestations.
   1. Persistent or paroxysmal hypertension.
   2. Palpitations, tachycardia.
   3. Hyperglycemia.
   4. Diaphoresis.
   5. Nervousness, apprehension.
B. Diagnostics (see Appendix 13-1).
   1. Increase in urinary excretion of total free catecholamine.
   2. Increase in urinary excretion of vanillylmandelic acid and metanephrine.
   3. Serum assay of catecholamine.
   4. Magnetic resonance imaging, computed tomography scan.
C. Treatment.
   1. Medications.
      a. Antihypertensive medications (alpha-adrenergic blockers to lower blood pressure quickly).
      b. Antidysrhythmic medications.
      c. Potassium replacement.

Nursing Interventions
Goal: To decrease client’s hypertension and provide preoperative nursing measures as appropriate (see Chapter 3).
A. Decrease intake of stimulants.
B. Sedate as indicated.
C. Maintain calm, quiet environment.
D. Assess vital signs frequently.
Goal: To help client return to homeostasis after adrenalectomy.
A. Maintain normal blood pressure the first 24 to 48 hours after surgery; client is at increased risk for hemorrhage or severe hypertensive episode.
   1. Assess for blood pressure changes caused by catecholamine imbalance (both hypertension and hypotension).
   2. Administer analgesics judiciously.
   3. Administer corticosteroids as indicated.
   4. Maintain quiet, cool environment.
   5. Maintain IV fluid administration.
B. Assess for hypoglycemia.

C. Tendency toward hemorrhagic problems.
D. Monitor renal perfusion and urinary output.
E. Assess for problems of adrenal insufficiency.

Goal: To maintain health after adrenalectomy.
A. Continued medical follow-up care.
B. If both adrenals are removed, client will require lifelong replacement of adrenal hormones.

Addison’s Disease (Adrenocortical Insufficiency/Adrenal Hypofunction)

Addison’s disease is caused by a decrease in secretion of the adrenal cortex hormones.
A. Decreased physiologic response to stress, vascular insufficiency, and hypoglycemia.
B. Decrease in aldosterone secretions (mineralocorticoids), which normally promote concentration of sodium and water and excretion of potassium.
C. May cause an alteration in adrenal androgen secretion necessary for secondary sex characteristics.

Assessment
A. Risk factors/etiology.
   1. An autoimmunity–induced problem.
   2. Occurs after bilateral adrenalectomy.
   3. Abrupt withdrawal from long-term corticosteroid therapy.
   4. Adrenal crisis may be precipitated by client’s failure to take medications.
   5. Acquired immunodeficiency syndrome (AIDS) is increasingly being identified as a cause of adrenal insufficiency.
   6. Increased emotional stress without appropriate hormone replacement.
   7. Tuberculosis.
   8. Septicemia.
B. Clinical manifestations (development of symptoms requires loss of 90% of both adrenal cortices).
   1. Onset is insidious; client may go for weeks to months before diagnosis is made.
   2. Fatigue, weakness.
   3. Weight loss.
   4. GI disturbances.
   5. Bronze pigmentation of the skin.
   6. Postural hypotension.
   7. Hyponatremia, hyperkalemia.
   8. Hypoglycemia.
      a. Profound fatigue.
      b. Dehydration.
      c. Vascular collapse (cyanosis and signs of shock: pallor, anxiety, weak/rapid pulse, tachypnea, and low blood pressure).

**ALERT** Determine whether vital signs are abnormal (e.g., hypotension, hypertension); notify others of change in the client’s condition.
C. Diagnostics (see Appendix 13-1).
   1. ACTH stimulation test.
   2. Decreased serum sodium level and increased serum potassium and BUN levels.
   3. Plasma ACTH level.

Treatment
Replace adrenal hormones.

Nursing Interventions

Goal: To help client return to homeostasis.
A. Initiate and maintain IV infusion of normal saline solution.
B. Administer large doses of corticosteroids through IV bolus initially, then titrate in a diluted solution.
C. Frequent evaluation of vital signs.
D. Assess sodium and water retention.
E. Evaluate serum potassium levels.
F. Keep client immobilized and quiet.

Alert: If any client is experiencing difficulty with maintaining adequate blood pressure, do not move the client unless absolutely necessary. Avoid all unnecessary nursing procedures until the client’s condition is stabilized.

Goal: To safely take steroid replacements (see Appendix 6-7).
A. Administer steroid preparations with food or an antacid.
B. Evaluate for edema and fluid retention.
C. Assess serum sodium and potassium levels.
D. Check daily weight.
E. Increase intake of protein and carbohydrates.
F. Evaluate for hypoglycemia.
G. Observe for cushingoid symptoms.

Home Care
A. Lifelong steroid therapy is necessary.
B. Dosage of steroids may need to be increased in times of additional stress.
C. Infection, diaphoresis, and injury will necessitate an increase in the need for steroids and may precipitate a crisis state.
D. Report gastric distress because it may be caused by steroids.
E. Carry a medical identification card.

Cushing’s Syndrome (Adrenal Cortex Hypersecretion/Hypercortisolism)

Cushing’s syndrome occurs as a result of excess levels of adrenal cortex hormones (primarily glucocorticoids) and, to a lesser extent, androgen and aldosterone (see Table 13-5).

Alert: Evaluate client’s use of medications. Implement procedures to counteract adverse effects of medication. The most common cause of Cushing’s syndrome is long-term steroid therapy for chronic conditions. Many chronic conditions necessitate the use of long-term steroid therapy.

Assessment
A. Risk factors/etiology.
   1. More common in women (20 to 40 years old).
   2. Pituitary hypersecretion.
   3. A benign pituitary tumor.
   4. Iatrogenic: most often a result of long-term steroid therapy.
B. Clinical manifestations (Figure 13-7).
   1. Marked change in personality (emotional lability), irritability.
   2. Changes in appearance.
      a. Moon face.
      b. Deposit of fat on the back.
      c. Thin skin, purple striae.
      d. Truncal obesity with thin extremities.
      e. Bruises and petechiae.
   3. Persistent hyperglycemia.
   4. GI distress from increased acid production.
   5. Osteoporosis.
   6. Increased susceptibility to infection.
   7. Sodium and fluid retention; potassium depletion.
   8. Hypertension.
      a. Amenorrhea (females).
      b. Hirsutism (females).
      c. Gynecomastia (males).
   10. Impotence or decreased libido.
C. Diagnostics (see Appendix 13-1).
   1. Increased serum sodium and decreased serum potassium levels.
   2. Hyperglycemia.
   3. Increased plasma cortisol levels.
   4. Loss of diurnal variation in cortisone levels.
   5. Low-dose and high-dose dexamethasone suppression test.

FIGURE 13-7 Cushing’s syndrome. (From Zerwekh J, Claborn J: Memory notebook of nursing, vol 1, ed 4, Ingram, Texas 2008, Nursing Education Consultants.)
D. Complications.
   1. Congestive heart failure, hypertension.
   2. Pathologic fractures, psychosis.

**Treatment**

Treatment depends on the cause of the problem.

**Nursing Interventions**

**Goal:** To assist client to manage hormone imbalance.

A. Restrict sodium and water intake.
B. Monitor fluid and electrolyte levels.
C. Evaluate for hyperglycemia.
D. Assess for GI disturbances.
E. Prevent infection.

**Goal:** To prevent complications.

A. Excessive sodium and water retention: monitor for edema, hypertension, congestive heart failure.
**Appendix 13-1 ENDOCRINE DIAGNOSTICS—cont’d**

<table>
<thead>
<tr>
<th>DIAGNOSTIC TEST</th>
<th>NORMAL</th>
<th>CLINICAL/NURSING IMPLICATIONS</th>
</tr>
</thead>
</table>
| Glucose fasting blood sugar (FBS) Also called *fasting blood glucose (FBG)* and *fasting plasma glucose (FPG)* | Same as serum glucose (>126 mg/dL is diagnostic for diabetes) | 1. Used as a screening test for problems of metabolism.  
2. Maintain client in fasting state for 12 hours until blood is drawn.  
3. If client is a known diabetic and experiences dizziness, weakness, or fainting, draw blood for determination of glucose level. |
| Glycosylated hemoglobin (HbA1c) | Nondiabetic range is usually 4%-6%  
2%-6.4% considered good diabetic control (goal is 7% or less)  
>8% considered poor diabetic control | 1. More accurate test of diabetic control, because it measures glucose attached to hemoglobin (indicates overall control for past 90-120 days, which is the lifespan of the RBC). |
| **ALERT** Frequently, the level of the FBS is given in a question, and it is necessary to evaluate the level and determine the appropriate nursing intervention. |
| 2-hour postprandial blood sugar | 65-139 mg/dL | 1. Involves measuring the serum glucose 2 hours after a meal; results are significantly increased with diabetes.  
2. Other intestinal conditions and inflammatory conditions cause increase. |
| Serum amylase | 30-200 U/L | 1. Used to evaluate pancreatic cell damage.  
2. Other intestinal conditions and inflammatory conditions cause increase. |
| Serum lipase | Normal values vary with method; elevated is abnormal | 1. Appears in serum after damage to pancreas. |
| Urine sugar (Clinistix, Clinitest, Labstix) | Negative for glucose | 1. Use freshly voided urine.  
2. A rough indicator of serum glucose levels.  
3. Results may be altered by various medications. |
| Ketone bodies (acetone) | Negative | 1. Ketone bodies occur in the urine before there is significant increase in serum ketones.  
2. Use freshly voided urine. |
| Urinary amylase | 2-hr specimen 2-34 U/hr  
24-hr specimen 24-408 U/24 hr | 1. In pancreatic injury, more amylase enters blood and is excreted in urine.  
2. May be done on a 2-hr or a 24-hr urine specimen. |
| **Pituitary** | |  |
| Growth hormone (GH) | <5 ng/mL in men  
<18 ng/mL in women | 1. NPO after midnight.  
2. Maintain bed rest until serum sample is drawn. |
| Osmolarity urine | 300 to 900 mOsm/kg of water | 1. Used in evaluating ADH.  
2. Do serum and urine tests at same time and compare results.  
3. Normally, urine osmolarity should be higher than serum. |
| Osmolarity serum | 285 to 295 mOsm/kg of water |  |
| **Adrenal Medulla** | |  |
| Urinary vanillylmandelic acid (VMA) | <8 mg in 24 hr  
Increased with pheochromocytoma | 1. Depending on how test is measured, there may be dietary and medication restrictions.  
2. 24-hr urine collection. |

*Continued*
### Appendix 13-1  ENDOCRINE DIAGNOSTICS—cont’d

<table>
<thead>
<tr>
<th>DIAGNOSTIC TEST</th>
<th>NORMAL</th>
<th>CLINICAL/NURSING IMPLICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine catecholamines: Epinephrine, norepinephrine</td>
<td>Increased in conditions that precipitate increase in catecholamine secretion</td>
<td>Same as for VMA.</td>
</tr>
<tr>
<td>Dopamine</td>
<td></td>
<td>1. ACTH is given as IM or IV bolus, and samples are drawn at 30 and 60 min to evaluate ability of adrenal glands to secrete steroids.</td>
</tr>
<tr>
<td>Metanephrine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normetanephrine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACTH stimulation test</td>
<td>Increase in plasma cortisol levels by more than 7–10 mcg/dL above baseline</td>
<td></td>
</tr>
</tbody>
</table>

#### Adrenal Cortex

<table>
<thead>
<tr>
<th>ACTH suppression (dexamethasone suppression test)</th>
<th>Normal suppression; 50% decrease in cortisone production (cortisol level &lt;3 mcg/dL)</th>
<th>1. An overnight test: a small amount of dexamethasone is administered in the evening, and serum and urine are evaluated in the morning; extensive test may cover 6 days. 2. Cushing's syndrome is ruled out if suppression is normal.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma cortisol levels for diurnal variations</td>
<td>Secretion high in early morning, decreased in evening. 8:00 a.m.: 5–23 mcg/dL 4:00 p.m.: 3–13 mcg/dL</td>
<td>1. Elevation in plasma cortisol levels occurs in the morning and significant decrease in evening and night—a diurnal variation.</td>
</tr>
<tr>
<td>24-hour urine for 17-hydroxycorticosteroids and 17-ketosteroids</td>
<td>Male: 3–10 mg/24 hr  Female: 2–8 mg/24 hr  Child under 15 yr: &lt;4.5 mg/24 hr</td>
<td>1. Increase in urine levels indicates hyperadrenal function.</td>
</tr>
</tbody>
</table>

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**ACTH**, Adrenocorticotropic hormone; **ADH**, antidiuretic hormone; **NPO**, nothing by mouth; **RBC**, red blood cell.

### Appendix 13-2  MEDICATIONS USED IN ENDOCRINE DISORDERS

<table>
<thead>
<tr>
<th>MEDICATIONS</th>
<th>SIDE EFFECTS</th>
<th>NURSING IMPLICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ADH Replacement</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Desmopressin (DDAVP): nasal spray, PO, IV, subQ</td>
<td>Excessive water retention, headache, nausea, flushing</td>
<td>1. Monitor daily weight; correlate with intake and output. 2. Vasopressin more likely to cause adverse cardiovascular and thromboembolic problems.</td>
</tr>
<tr>
<td>Vasopressin (Pitressin): IM, subQ</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lypressin (Diapid): nasal spray</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Antithyroid Agents</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Propylthiouracil (PTU): PO</td>
<td>Agranulocytosis; abdominal discomfort; nausea, vomiting, diarrhea; crosses placenta</td>
<td>1. May increase anticoagulation effect of heparin and oral anticoagulants. 2. May be combined with iodine preparations. 3. Monitor CBC. 4. Store Tapazole in light-sensitive container. 5. May be used before surgery or treatment with radioactive iodine.</td>
</tr>
<tr>
<td>Methimazole (Tapazole): PO</td>
<td>Same; crosses placenta more rapidly</td>
<td></td>
</tr>
<tr>
<td>Lugol’s solution: PO</td>
<td>Inhibits synthesis and release of thyroid hormone</td>
<td>1. Administer in fluid to decrease unpleasant taste. 2. May be used to decrease vascularity of thyroid gland before surgery.</td>
</tr>
</tbody>
</table>

**Saturated solution of potassium iodide (SSKI)**
### Medications

#### Radioactive Iodine
Accumulates in the thyroid gland; causes partial or total destruction of thyroid gland through radiation.

<table>
<thead>
<tr>
<th>Medication</th>
<th>SIDE EFFECTS</th>
<th>NURSING IMPLICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iodine ($^{123}$I or $^{131}$I): PO</td>
<td>Discomfort in thyroid area; bone marrow depression</td>
<td>1. Increase fluids immediately after treatment, because radioactive isotope is excreted in the urine.</td>
</tr>
<tr>
<td></td>
<td>Desired effect: permanent hypothyroidism</td>
<td>2. Therapeutic dose of radioactive iodine is low; no radiation safety precautions are required.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Contraindicated in pregnancy.</td>
</tr>
</tbody>
</table>

#### Thyroid Replacements
Replace thyroid hormone.

<table>
<thead>
<tr>
<th>Medication</th>
<th>SIDE EFFECTS</th>
<th>NURSING IMPLICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Levothyroxine sodium (Levothroid, Levoxyl, Levo-T, Novothyrox, Synthroid): PO, IM, IV Liothyronine (Cytomel, Triostat)</td>
<td>Overdose may result in symptoms of hyperthyroidism: tachycardia, heat intolerance, nervousness</td>
<td>1. Be careful in reading exact name on label of medications; micrograms and milligrams are used as units of measure.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Generally taken once a day before breakfast.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Within 3-4 days, begin to see improvement; maximum effect in 4-6 weeks.</td>
</tr>
</tbody>
</table>

#### Pancreatic Enzymes
Replacement enzymes to aid in digestion of starch, protein, and fat.

<table>
<thead>
<tr>
<th>Medication</th>
<th>SIDE EFFECTS</th>
<th>NURSING IMPLICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pancreatin (Creon): PO</td>
<td>GI upset and irritation of mucous membranes</td>
<td>1. Client is usually on a high-protein, high-carbohydrate, low-fat diet.</td>
</tr>
<tr>
<td>Pancrelipase (Pancrease, Pangestyme, Ultrase, Viokase): PO</td>
<td></td>
<td>2. Enteric-coated tablets should not be crushed or chewed.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Pancreatin may be given before, during, or within 1 hr after meals.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. Pancrelipase is given just before or with each meal or snack.</td>
</tr>
</tbody>
</table>

#### Antihypoglycemic Agent
Increases plasma glucose levels and relaxes smooth muscles.

<table>
<thead>
<tr>
<th>Medication</th>
<th>SIDE EFFECTS</th>
<th>NURSING IMPLICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucagon: IM, IV, subQ</td>
<td>None significant</td>
<td>1. Watch for symptoms of hypoglycemia and treat with food first, if conscious.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Client usually awakens in 5-20 min after receiving glucagon.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. If client does not respond, anticipate IV glucose to be given.</td>
</tr>
</tbody>
</table>

#### Oral Hypoglycemic Agents
Stimulate beta cells to secrete more insulin; enhance body utilization of available insulin (see Figure 13-3 for insulin).

<table>
<thead>
<tr>
<th>Medication</th>
<th>SIDE EFFECTS</th>
<th>NURSING IMPLICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlorpropamide (Diabinese): PO</td>
<td>Hypoglycemia, jaundice, GI disturbance, skin reactions (fewer side effects with second-generation agents)</td>
<td>1. Tolbutamide has shortest duration of action; requires multiple daily doses.</td>
</tr>
<tr>
<td>Glipizide (Glucotrol): PO</td>
<td></td>
<td>2. Glyburide has a long duration of action.</td>
</tr>
<tr>
<td>Glyburide (Micronase, DiaBeta): PO</td>
<td></td>
<td>3. Interact with calcium channel blockers, oral contraceptives, glucocorticoids, phenothiazines, and thiazide diuretics.</td>
</tr>
<tr>
<td>Glimepiride (Amaryl): PO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gliclazide (Diamicron): PO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tolbutamide (Orinase): PO</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tolazamide (Tolinase): PO</td>
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<td></td>
</tr>
</tbody>
</table>
### Appendix 13-2 MEDICATIONS USED IN ENDOCRINE DISORDERS—cont’d

<table>
<thead>
<tr>
<th>MEDICATIONS</th>
<th>SIDE EFFECTS</th>
<th>NURSING IMPLICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Biguanides</strong></td>
<td>Decrease sugar production in the liver and help the muscles use insulin to break down sugar.</td>
<td></td>
</tr>
</tbody>
</table>
| Metformin (Glucophage): PO           | Dizziness, nausea, back pain, possible metallic taste | 1. Administered with meals.  
2. Is beneficial effect on lowering lipids.  
3. Weight gain may occur. |
| **Alpha-Glucosidase Inhibitors**     | Slow down body absorption of sugar after eating; also known as *starch blockers*. |                                                                                      |
| Acarbose (Precose): PO               | Diarrhea, flatulence, abdominal pain               | 1. Take at beginning of meals; not effective on an empty stomach.  
2. Acarbose is contraindicated in clients with inflammatory bowel disease.  
3. Frequently given with sulfonylureas to increase effectiveness of both medications. |
| Miglitol (Glyset): PO                |                                                                                             |                                                                                      |
| **Thiazolidinediones**               | Enhance insulin utilization at receptor sites (they do not increase insulin production); also referred to as *insulin sensitizers*. |                                                                                      |
| Pioglitazone (Actos): PO             | Weight gain, edema                               | 1. May affect liver function; monitor LFTs.  
2. Postmenopausal women may resume ovulation; pregnancy may occur. |
| Rosiglitazone (Avandia): PO          |                                                                                             |                                                                                      |
| **Meglitinides (Nonsulfonylurea Insulin Secretagogues)** | Stimulate release of insulin from beta cells. |                                                                                      |
| Nateglinide (Starlix): PO            | Weight gain, hypoglycemia                         | 1. Rapid onset and short duration.  
2. Take 30 min before meals (or right at mealtime).  
3. Do not take if meal is missed. |
| Repaglinide (Prandin): PO            |                                                                                             |                                                                                      |
| **Dipeptidyl Peptidase-4 (DDP-4) Inhibitors** | Enhance the incretin system, stimulate release of insulin for beta cells, and decrease hepatic glucose production. |                                                                                      |
| Sitagliptin (Januvia): PO            | Upper respiratory tract infection, sore throat, headache, diarrhea                           | 1. Should not be used in type 1 diabetes or for the treatment of diabetic ketoacidosis. |
| Vildagliptin (Galvus): PO            |                                                                                             |                                                                                      |
| **Injectable Drugs for Diabetes**    |                                                                                             |                                                                                      |
| **Amylin Mimetics**                  | Complement the effects of insulin by delaying gastric emptying and suppressing glucagon secretion. |                                                                                      |
| Pramlintide (Symlin): subQ           | Hypoglycemia, nausea, injection site reactions   | 1. Teach client to take other oral medications at least 1 hour before taking or 2 hours after, because of delayed gastric emptying.  
2. Injected into thigh or abdomen.  
3. Cannot be mixed with insulin. |
| **Incretin Mimetics**                | Stimulate release of insulin, decrease glucagon secretion, decrease gastric emptying, and suppress appetite. |                                                                                      |
| Exenatide (Byetta): subQ             | Hypoglycemia, nausea, vomiting, diarrhea, headache, possible weight loss                    | 1. Used in conjunction with metformin.  
3. Not indicated for use with insulin. |

*CBC, Complete blood count; GI, gastrointestinal; IM, intramuscularly; IV, intravenously; LFTs, liver function tests; PO, by mouth (orally); subQ, subcutaneously.*
Study Questions  Endocrine System

1. A client is receiving NPH insulin 20 units subcutaneously at 0700 hours daily. At 1500 hours, the nurse finds the client apparently asleep. How would the nurse know whether the client were having a hypoglycemic reaction?
   1. Feel the client and bed for dampness.
   2. Observe the client for Kussmaul respirations.
   3. Smell the client’s breath for acetone odor.
   4. Check the client’s pupils for dilation.

2. The nurse is caring for a client postoperative thyroidectomy. What would be an important nursing intervention?
   1. Have the client speak every 5 to 10 minutes if hoarseness is present.
   2. Provide a low-calcium diet to prevent hypercalcemia.
   3. Check the dressing at the back of the neck for bleeding.
   4. Apply a soft cervical collar to restrict neck movement.

3. The nurse is assigned to care for a new client with acute pancreatitis. What would the nurse anticipate finding during the assessment of this client?
   1. Steatorrhea, abdominal pain, fever
   2. Fever, hypoglycemia, dehydration
   3. Melena, persistent vomiting, hyperactive bowel sounds
   4. Hypoactive bowel sounds, decreased amylase and lipase levels

4. A client is found to be comatose and hypoglycemic with a blood glucose level of 50 mg/dL. What nursing action is implemented first?
   1. Infuse 1000 mL of D5W over a 12-hour period.
   2. Administer 50% glucose intravenously.
   3. Check the client’s urine for the presence of sugar and acetone.
   4. Encourage the client to drink orange juice with added sugar.

5. A nurse knows the clinical manifestations of a client with Addison’s disease include which of the following? Select all that apply.
   1. Weight gain
   2. Hypothermia
   3. Hypertension
   4. Melanosis of skin
   5. Hypotension
   6. Hypernatremia

6. A client is prescribed levothyroxine (Synthroid) daily. What is the most important instruction to give the client for administration of this drug?
   1. Taper the dose and discontinue if mental and emotional statuses stabilize.
   2. Take it at bedtime to avoid the side effects of nausea and flatus.
   3. Call the doctor immediately at the onset of palpitations or nervousness.
   4. Decrease the intake of juices and fruits with high potassium and calcium contents.

7. What will the nurse teach the client with diabetes regarding exercise in his or her treatment program?
   1. During exercise the body will use carbohydrates for energy production, which in turn will decrease the need for insulin.
   2. With an increase in activity, the body will use more carbohydrates; therefore more insulin will be required.
   3. The increase in activity results in an increase in the use of insulin; therefore the client should decrease his or her carbohydrate intake.
   4. Exercise will improve pancreatic circulation and stimulate the islets of Langerhans to increase the production of intrinsic insulin.

8. The nurse is caring for a client who has exophthalmos associated with her thyroid disease. What is the cause of exophthalmos?
   1. Fluid and edema in the retro-orbital tissues, which increases pressure behind the eyes
   2. Impaired vision, which causes the client to squint in order to focus
   3. Increased intraocular pressure from an increase in circulating thyroid
   4. Decrease in extraocular eye movements, which results in the “thyroid stare”

9. What is a characteristic symptom of hypoglycemia that should alert the nurse to an early insulin reaction?
   1. Diaphoresis
   2. Drowsiness
   3. Severe thirst
   4. Coma

10. A client is scheduled for a routine glycosylated hemoglobin (HbA1c) test. What is important for the nurse to tell the client before this test?
    1. Drink only water after midnight and come to the clinic early in the morning.
    2. Eat a normal breakfast and be at the clinic 2 hours later.
    3. Expect to be at the clinic for several hours because of the multiple blood draws.
    4. Come to the clinic at the earliest convenience to have blood drawn.
11. A client has been receiving inhalation vasopressin therapy. What will the nurse evaluate to determine the therapeutic response to this medication?
   1. Urine specific gravity
   2. Blood glucose
   3. Vital signs
   4. Oxygen saturation levels

12. A client with a diagnosis of type 2 diabetes has been ordered a course of prednisone for her severe arthritic pain. An expected change that requires close monitoring by the nurse is:
   1. Increased blood glucose level
   2. Increased platelet aggregation
   3. Increased creatinine clearance
   4. Increased ketone level in urine

13. The nurse is performing an assessment on a client who has been receiving long-term steroid therapy. What would the nurse expect to find during the assessment?
   1. Jaundice
   2. Flank pain
   3. Bulging eyes
   4. Central obesity

14. It is important for the nurse to teach the client which of the following about metformin (Glucophage)?
   1. It may cause nocturia.
   2. It should be taken at night.
   3. It should be taken with meals.
   4. It may increase the effects of aspirin.

15. A nurse assessing a client with syndrome of inappropriate antidiuretic hormone (SIADH) would expect to find which laboratory values?
   1. Serum sodium = 150 mEq/L and low urine osmolality
   2. Serum potassium = 5 mEq/L and low serum osmolality
   3. Serum sodium = 120 mEq/L and low serum osmolality
   4. Serum potassium = 3 mEq/L and high serum osmolality

16. A priority nursing diagnosis for a client admitted to the hospital with a diagnosis of diabetes insipidus is:
   1. Disturbed sleep pattern related to nocturia
   2. Activity intolerance related to muscle weakness
   3. Excess fluid volume related to intake greater than output
   4. Risk for impaired skin integrity related to generalized edema

17. A client admitted with a pheochromocytoma returns from the operating room after adrenalectomy. The nurse should carefully assess this client for:
   1. Hypokalemia
   2. Hyperglycemia
   3. Marked sodium and water retention
   4. Marked fluctuations in blood pressure

18. When caring for a client in a thyroid crisis, the nurse would question an order for:
   1. IV fluids
   2. Propranolol (Inderal)
   3. Propylthiouracil
   4. A hyperthermia blanket

19. Which medication will the nurse have available for emergency treatment of tetany in the client who has had a thyroidectomy?
   1. Calcium chloride
   2. Potassium chloride
   3. Magnesium sulfate
   4. Sodium bicarbonate

20. A client with diabetes receives a combination of regular and NPH insulin at 0700 hours. The nurse teaches the client to be alert for signs of hypoglycemia at:
   1. 12 p.m. to 1 p.m. (1200 to 1300 hours)
   2. 11 a.m. and 5 p.m. (1100 and 1700 hours)
   3. 10 a.m. and 10 p.m. (1000 and 2200 hours)
   4. 8 a.m. and 11 a.m. (0800 and 1100 hours)

Answers and rationales to these questions are in the section at the end of the book titled Chapter Study Questions: Answers and Rationales.