Chapter 16

Endocrine System Disorders
Location of Endocrine Glands

- Hypothalamus
- Pituitary
- Parathyroids
- Thyroid
- Thymus
- Adrenals
- Pancreas (islets)
- Ovaries (female)
- Testes (male)

Endocrine System

- Hormones as chemical messengers
- Target receptors
- Negative feedback systems
- Chemical structure
  - Peptide
  - Steroid
Classification by Source

- Hypothalamus
- Pituitary
- Thymus
- Thyroid
- Parathyroid
- Adrenal gland
- Pancreas
- Pineal
- Ovaries
- Testes
Classification by Chemical Structure

● Steroid
  ➢ They are lipids and enter the cell nucleus to initiate transcription directly.

● Nonsteroid
  ➢ Needs a secondary messenger system to activate transcription in the nucleus
### TABLE 16-1 Sources of Major Hormones and Primary Effects

<table>
<thead>
<tr>
<th>Hormone</th>
<th>Source</th>
<th>Primary Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypothalamic-releasing hormones</td>
<td>Hypothalamus</td>
<td>Stimuli to anterior pituitary to release specific hormone</td>
</tr>
<tr>
<td>Hypothalamic-inhibiting hormones</td>
<td>Hypothalamus</td>
<td>Decrease release of specific hormone by anterior pituitary</td>
</tr>
<tr>
<td>Growth hormone (GH, somatotropin)</td>
<td>Pituitary—anteior lobe</td>
<td>Stimulates protein synthesis</td>
</tr>
<tr>
<td>Adrenocorticotropic hormone (ACTH)</td>
<td>Adenohypophysis</td>
<td>Stimulates adrenal cortex to secrete primarily cortisol</td>
</tr>
<tr>
<td>Thyroid-stimulating hormone (TSH)</td>
<td>Adenohypophysis</td>
<td>Stimulates thyroid gland</td>
</tr>
<tr>
<td>Follicle-stimulating hormone (FSH)</td>
<td>Adenohypophysis</td>
<td>Women: stimulates growth of ovarian follicles and estrogen secretion; men: stimulates sperm production</td>
</tr>
<tr>
<td>Luteinizing hormone (LH)</td>
<td>Adenohypophysis</td>
<td>Women: stimulates maturation of ovum and ovulation; men: stimulates secretion of testosterone</td>
</tr>
<tr>
<td>Prolactin (PRL)</td>
<td>Adenohypophysis</td>
<td>Stimulates breast milk production during lactation</td>
</tr>
<tr>
<td>Antidiuretic hormone (ADH, or vasopressin)</td>
<td>Pituitary—posterior lobe</td>
<td>Increases reabsorption of water in kidney</td>
</tr>
<tr>
<td>Oxytocin (OT)</td>
<td>Neurohypophysis</td>
<td>Stimulates contraction of uterus after delivery; Stimulates ejection of breast milk during lactation</td>
</tr>
<tr>
<td>Insulin</td>
<td>Pancreas—beta cells of islets of Langerhans</td>
<td>Transport of glucose and other substances into cells; Lowers blood glucose level</td>
</tr>
<tr>
<td>Glucagon</td>
<td>Pancreas—alpha cells</td>
<td>Glycogenolysis in liver; Increases blood glucose level</td>
</tr>
<tr>
<td>Parathyroid hormone (PTH)</td>
<td>Parathyroid gland</td>
<td>Increases blood calcium level by stimulating bone demineralization and increasing absorption of Ca&lt;sup&gt;2+&lt;/sup&gt; in the digestive tract and kidneys</td>
</tr>
<tr>
<td>Calcitonin</td>
<td>Thyroid gland</td>
<td>Decreases release of calcium from the bone to lower blood calcium level</td>
</tr>
<tr>
<td>Thyroxine (T&lt;sub&gt;4&lt;/sub&gt;)</td>
<td>Thyroid gland</td>
<td>Increases metabolic rate in all cells</td>
</tr>
<tr>
<td>Triiodothyronine (T&lt;sub&gt;3&lt;/sub&gt;)</td>
<td>Thyroid gland</td>
<td></td>
</tr>
<tr>
<td>Aldosterone</td>
<td>Adrenal cortex</td>
<td>Increases sodium and water reabsorption in the kidney</td>
</tr>
<tr>
<td>Cortisol</td>
<td>Adrenal cortex</td>
<td>Anti-inflammatory and decreases immune response; Catabolic effect on tissues; stress response</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>Adrenal medulla</td>
<td>General vasoconstriction</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>Adrenal medulla</td>
<td>Stress response; Visceral and cutaneous vasoconstriction; Vasodilation in skeletal muscle; Increases rate and force of heart contraction</td>
</tr>
</tbody>
</table>
Control of the Endocrine System

- The endocrine and nervous systems regulate metabolic activities.
- **Negative feedback system**
  - Positive feedback may be in a negative feedback loop.
    - Blood clotting
    - Child birth
- **Some hormones act as antagonists, such as:**
  - Calcitonin and parathyroid hormone
  - Insulin and glucagon
Negative Feedback

1. High blood glucose
2. Beta cells of pancreas increase secretion of insulin
3. Insulin promotes transport of glucose into cells
4. Low blood glucose
5. Alpha cells secrete glucagon
6. Gluconeogenesis in liver

[Diagram showing glucose, insulin, and glucagon pathways]

Hormone Release

Most often controlled by negative feedback mechanisms

- Endocrine and nervous systems work together to regulate metabolic activities.
- Complex system for some hormones
- Secretion may be controlled by more than one mechanism.
- Rate and timing of secretion may vary.
  - Cyclic patterns
Endocrine Disorders

- All disorders reflect impaired control or feedback.
- Excess hormone levels
  - Tumor produces high levels
  - Excretion by liver or kidney is impaired
  - Congenital condition produces excess hormone
Endocrine Disorders (Cont.)

● Deficit of hormone or reduced effects
  ➢ Tumor produces too little hormone
  ➢ Inadequate tissue receptors present
  ➢ Antagonistic hormone production is increased.
  ➢ Malnutrition
  ➢ Atrophy, surgical removal of gland
  ➢ Congenital deficit
Diagnostic Tests

- Blood tests
  - Check serum hormone levels
  - Radioimmunoassay
  - Immunochemical methods

- Urine tests

- Stimulation or suppression tests

- Scanning, ultrasound, magnetic resonance imaging (MRI)

- Biopsy
Treatment

- Deficit may be treated with replacement therapy.
- Excessive secretion may be treated with:
  - Medications
  - Surgery
  - Radiation
Insulin and Diabetes Mellitus

- Diabetes mellitus—basic problem is inadequate insulin effects in receptor tissues
  - Deficit of insulin secretion
  - Production of insulin antagonists
- Diabetes results in abnormal carbohydrate, protein, and fat metabolism.
- Some tissues can transport glucose in the absence of insulin:
  - CNS, kidney, myocardium, gut, skeletal muscle
    - Skeletal muscle can partially meet tissue needs without insulin.
Types of Diabetes

- Type 1
  - Autoimmune destruction of beta cells in pancreas
  - Insulin replacement required
  - Acute onset in children and adolescents
  - Not linked to obesity
  - Genetic factors may play a role.
Types of Diabetes (Cont.)

- **Type 2**
  - Non–insulin-dependent
  - Oral hypoglycemic medications may be used.
  - Caused by decreased production of insulin and/or increased resistance by body cells to insulin
  - Onset is slow and insidious, usually in those older than 50 years
  - Associated with obesity
  - Component of metabolic syndrome
  - Increasing incidence in teens and young adults
General Manifestations

- Insulin deficit results in decreased transport and use of glucose in many cells.
  - Polyphagia
  - Fatigue
- Blood glucose levels rise—hyperglycemia
- Excess glucose in urine—glucosuria
  - Dehydration results from hyperosmolar filtrate.
  - Polyuria
  - Polydipsia
Diabetes: Diagnostic Tests

- Fasting blood glucose level
- Glucose tolerance test
- Glycosylated hemoglobin test
  - Clinical and subclinical diabetes
  - Monitor glucose levels over several months.
Diabetes: Treatment Principles

- Maintenance of blood glucose levels in normal range
  - Helps reduce complications
- Diet and exercise
  - Exercise reduces blood glucose level as skeletal muscle uses glucose.
- Oral medication
  - Increase insulin secretion.
  - Reduce blood glucose levels.
- Insulin replacement
Type 1 Diabetes

- Metabolic changes
  - Catabolism of fats and proteins
    - Excessive amounts of fatty acids and metabolites
    - Ketones in the blood
  - Ketonuria
    - Decreased serum bicarbonate
    - Decrease in pH of body fluids
    - Ketoacids excreted in urine
  - Decompensated metabolic acidosis
Complications

- Complications are directly related to duration and extent of abnormal blood glucose levels.
- Many factors lead to fluctuations in serum glucose levels.
  - Variations in diet and alcohol use
  - Change in physical activity
  - Infection
  - Vomiting
- Complications may be acute or chronic.
Diabetes: Acute Complications

- Hypoglycemia (insulin shock)
  - More common with insulin replacement treatment
  - Can occur because of excess oral hypoglycemic drugs
  - Excess insulin in circulation
    - Glucose deficit in blood
    - Can be life-threatening or cause brain damage if untreated
    - Often follows strenuous exercise
    - Dosage error
    - Vomiting
    - Skipping meal after taking insulin
Hypoglycemic Shock

1. Excess insulin in blood
2. Increased transport of glucose into cells
3. Hypoglycemia
   Decreased CNS function
   • Clinical signs:
     - weakness
     - confusion
     - pallor
     - diaphoresis
     - tremors
     - tachycardia
4. Stimulates SNS
5. Increased gluconeogenesis
6. Excess insulin transports glucose into cells
7. No glucose intake
   • Glucose intake
   • Return to normal state
8. Blood glucose levels decrease further
9. Neurons cannot function
10. Coma and death

I = Insulin
G = Glucose
Hypoglycemic Shock: Signs and Symptoms

- Disorientation and change in behavior
- May appear impaired
- Anxiety or decreased responsiveness
- Decreased blood glucose level
- Decreased BP, increased heart rate
- Decreasing level of consciousness

► **NOTE:** Immediate administration of glucose is required to prevent brain damage.
Diabetes: Acute Complications (Cont.)

- Diabetic ketoacidosis
  - Occurs in insulin-dependent clients
  - More commonly seen in type 1 diabetes
  - Result of insufficient insulin in blood
  - High blood glucose levels
  - Mobilization and use of lipids to meet cellular needs result in production of ketoacids
  - May be initiated by infection or stress
  - May result from error in dosage, infection, change in diet, alcohol intake, or exercise
Development of Diabetic Ketoacidosis

- Decreased insulin secretion or increased insulin resistant-cells
  - Decreased glucose transport into cells
    - Glycogenolysis
    - Lipolysis (catabolism)
      - Gluconeogenesis
      - Ketone bodies (acidic waste-formed in large amounts)

- Polyphagia (hunger)
- Hyperglycemia (high blood glucose)
  - Glucosuria (excess glucose spills into urine)
    - Polydipsia (thirst)
    - Polyuria (osmotic diuresis-large volume)
      - Electrolyte imbalance (loss in urine)
        - Dehydration
        - Acidosis

- Ketoacidosis
- Ketonuria

Signs and Symptoms of Diabetic Ketoacidosis

- Dehydration
  - Thirst, dry, rough oral mucosa
  - Warm, dry skin
- Rapid, deep respiration—acetone breath
  - Lethargy, decreased responsiveness
- Metabolic acidosis
  - May lead to loss of consciousness
- Electrolyte imbalances
  - Abdominal cramps, nausea, vomiting, lethargy, weakness
## Effects of Insulin Deficit

### TABLE 16-3

**Progressive Effects of an Insulin Deficit (Diabetic Ketoacidosis)**

<table>
<thead>
<tr>
<th>Signs</th>
<th>Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EARLY SIGNS</strong></td>
<td></td>
</tr>
<tr>
<td>Hyperglycemia</td>
<td>Lack of insulin</td>
</tr>
<tr>
<td>Hunger (polyphagia)</td>
<td>Compensation for cell starvation</td>
</tr>
<tr>
<td>Glucosuria</td>
<td>Glucose in filtrate exceeds tubule transport</td>
</tr>
<tr>
<td>Polyuria</td>
<td>Osmotic diuresis due to glucosuria</td>
</tr>
<tr>
<td>Thirst (polydipsia)</td>
<td>Response to water loss</td>
</tr>
<tr>
<td>Weakness and weight loss</td>
<td>Loss of fluid and lack of glucose to cells</td>
</tr>
<tr>
<td><strong>PROGRESSIVE SIGNS</strong></td>
<td></td>
</tr>
<tr>
<td>Increasing hyperglycemia</td>
<td>Gluconeogenesis owing to response by epinephrine, cortisol, and glucagon</td>
</tr>
<tr>
<td>Dehydration</td>
<td></td>
</tr>
<tr>
<td>Skin warm and dry, decreased</td>
<td>Decreased interstitial fluid</td>
</tr>
<tr>
<td>turgor</td>
<td></td>
</tr>
<tr>
<td>Oral mucosa rough and dry</td>
<td>Decreased interstitial fluid</td>
</tr>
<tr>
<td>Eyeballs sunken and soft</td>
<td>Decreased interstitial fluid</td>
</tr>
<tr>
<td>Decreased blood pressure</td>
<td>Decreased blood volume</td>
</tr>
<tr>
<td>Pulse—rapid, thready</td>
<td>Compensation—sympathetic nervous system</td>
</tr>
<tr>
<td>Lethargy, weakness, confusion</td>
<td>Decreased oxygen and glucose to the brain resulting from hyperglycemia. Also, acidosis and electrolyte imbalance</td>
</tr>
<tr>
<td><strong>Ketoacidosis</strong></td>
<td>Catabolism of Fats And Protein</td>
</tr>
<tr>
<td>Serum bicarbonate and</td>
<td>Compensation for acidosis</td>
</tr>
<tr>
<td>serum pH low</td>
<td></td>
</tr>
<tr>
<td>Rapid, deep respirations</td>
<td>Compensation for acidosis</td>
</tr>
<tr>
<td>(air hunger or Kussmaul’s</td>
<td></td>
</tr>
<tr>
<td>respirations)</td>
<td></td>
</tr>
<tr>
<td>Acetone breath—sweet,</td>
<td>Acetone expired</td>
</tr>
<tr>
<td>fruity odor</td>
<td></td>
</tr>
<tr>
<td>Ketonuria</td>
<td>Ketoacids excreted in urine</td>
</tr>
<tr>
<td>Nausea, vomiting, weakness</td>
<td>Loss of Na⁺, K⁺, Cl⁻ in urine and ketonemia</td>
</tr>
<tr>
<td><strong>LATE SIGNS</strong></td>
<td></td>
</tr>
<tr>
<td>Coma</td>
<td>Central nervous system depression owing to acidosis and dehydration</td>
</tr>
</tbody>
</table>

Acute Complications: HHNK Syndrome

- **HHNK:** Hyperglycemic hyperosmolar nonketotic
- Occurs in type 2 diabetes
- Insidious in onset and diagnosis may be missed
- Often occurs in older clients and assumed to be cognitive impairment
- Results in severe dehydration and electrolyte imbalances
HHNK Manifestations

- Hyperglycemia
- Severe dehydration
  - Increased hematocrit
  - Loss of turgor
  - Increased heart rate and respirations
- Electrolyte imbalances result in:
  - Neurologic deficits
  - Muscle weakness
  - Difficulties with speech
  - Abnormal reflexes
Chronic Complications of Diabetes

- **Vascular problems**
  - Increased incidence of atherosclerosis
  - Changes may occur in small and large arteries.
- **Microangiopathy**—changes in microcirculation
  - Obstruction or rupture of small capillaries and arteries
    - Tissue necrosis and loss of function
    - Neuropathy and loss of sensation
    - Retinopathy—leading cause of blindness
    - Chronic renal failure—degeneration in glomeruli of kidney
## Vascular Problems with Diabetes

**Macroangiopathy**
- Myocardial infarction (heart attack)
- Cerebrovascular accident (stroke)
- Peripheral vascular disease (ischemia, gangrene, and amputation affecting the legs)
- Atherosclerosis in large arteries related to hyperlipidemia, hypertension, and degenerative changes in the intimal layer of the arterial wall

**Microangiopathy**
- Kidneys
  - Diabetic nephropathy
  - Chronic renal failure
- Eyes
  - Retinopathy
- Nervous system
  - Neuropathy in the central nervous system and peripheral nerves
  - Decreased function of sensory, motor, and autonomic nervous system fibers

**Note**—In addition to ischemia, there is also a metabolic abnormality that causes degeneration of myelin and deficit of myo-inositol, essential in the conduction of nerve impulses.
Diabetic Nephrosclerosis
Chronic Complications of Diabetes (Cont.)

- Macroangiopathy—affects large arteries
  - Result of abnormal lipid levels
    - High incidence of heart attacks, strokes, peripheral vascular disease
    - May result in ulcers on feet and legs—slow-healing
    - Frequent infections and gangrenous ulcers
    - Amputation may be necessary.

- Peripheral neuropathy
  - Common complication caused by ischemia in microcirculation to peripheral nerves
    - Impaired sensation, numbness, tingling, weakness, muscle wasting
Neuropathic Diabetic Foot Ulcer
Chronic Complications of Diabetes (Cont.)

● Infections
  ➢ Common and often more severe in diabetics
  ➢ Infections in feet and legs caused by vascular and neurological impairment
  ➢ Fungal infections common
    • Caused by *Candida*
    • In vagina and/or oral cavity
  ➢ Urinary tract infections
  ➢ Dental caries
  ➢ Gingivitis and periodontitis
Periodontal Disease in Diabetics


Chronic Complications of Diabetes (Cont.)

- **Cataracts**
  - Opacity of lens in eye
  - Related to abnormal metabolism of glucose

- **Pregnancy**
  - Complications in both mother and fetus may occur.
  - Increased incidence of spontaneous abortions
  - Infants born to diabetic mothers:
    - Increased size and weight for date
    - May experience hypoglycemia in first hours postnatally
Potential Complications of Diabetes Mellitus

- **Brain**: Cerebrovascular accident (Atherosclerosis)
- **Eyes**: Cataract, Retinal microaneurysms
- **Heart**: Myocardial infarction, Arrhythmias
- **Kidneys**: Nephropathy and infection
- **Neuropathy**: Impotence and infertility, Urinary incontinence, Numbness, weakness
- **Peripheral vascular disease**: Ulcers, Delayed healing, Gangrene

**Diabetes mellitus**
- Decreased insulin available
- Increased blood glucose
- Altered lipid and protein metabolism

**Acute complications**
- Hypoglycemic shock
- Diabetic ketoacidosis

Normal Control and Feedback of Calcium
Control of Type 2 Diabetes

- Diet should contain:
  - Increased fiber
  - Reduced lipids and simple carbohydrates
- Regular exercise to reduce glucose levels
- Reduce insulin resistance by reducing BMI to normal range
Control of Type 2 Diabetes (Cont.)

- Monitoring blood glucose levels as ordered
- Medication to stimulate the beta cells of the pancreas to produce more insulin
- If insulin-dependent—proper administration of insulin to maintain glucose levels in normal range
- Routine follow-up and blood testing
Calcium and Parathyroid Hormone Relationships
Parathyroid Hormone and Calcium

- Hypoparathyroidism
  - Leads to hypocalcemia
    - Weak cardiac muscle contractions
    - Increased excitability of nerves—spontaneous contractions of skeletal muscle
  - Causes
    - Tumor
    - Congenital lack of parathyroid
    - Surgery or radiation in neck region
    - Autoimmune disease
Parathyroid Hormone and Calcium (Cont.)

● Hyperparathyroidism
  ➢ Results in hypercalcemia
    • Forceful cardiac contractions
    • Osteoporosis
    • Predisposition to kidney stones
  ➢ Causes
    • Tumor
    • Secondary to renal failure
    • Paraneoplastic syndrome
Common Effects of Parathyroid Hormone Imbalance

HYPOPARATHYROIDISM

HYPOCALCEMIA

Nervous system
Increased neuroexcitability
Tingling in fingers and around mouth
Hyperactive reflexes

Skeletal muscle
Muscle spasm
Tetany

Heart
Weak cardiac muscle contraction
Arrhythmias
Hypotension

Gastrointestinal
Increased peristalsis
Diarrhea, nausea
Cramps

HYPOPARATHYROIDISM

HYPERCALCEMIA

Decreased neuroexcitability
Apathy, fatigue
Personality change

Muscle weakness
Decreased tone

Forceful cardiac contraction
Arrhythmias, bradycardia
Hypertension

Kidneys
Polyuria, thirst
Renal insufficiency
Renal calculi

Decreased peristalsis
Constipation, nausea

Bone
Osteoporosis, fractures
Pituitary Hormones

- Adenomas are the most common cause of pituitary disorders.
- Effect of mass
  - May cause pressure in the skull
    - Headaches, seizures, drowsiness, visual deficits
- Effect on hormone secretion
  - Dependent on cells and location involved
  - May cause excessive or decreased release of hormones
Growth Hormone (GH)

- **Dwarfism**
  - Deficit in growth hormone production and release

- **Gigantism**
  - Excess GH prior to puberty and fusion of epiphysis

- **Acromegaly**
  - Excess GH secretion in adults
  - Often associated with adenoma
  - Bones become broader and heavier.
  - Soft tissue grows.
    - Enlarged hands and feet, change in facial features
Effects of Growth Hormone
Antidiuretic Hormone (ADH)

- Diabetes insipidus—deficit of ADH
  - Adenoma
  - May originate in the neurohypophysis
    - Head injury or surgery
    - Possible genetic problem
    - Replacement treatment required

- Inappropriate ADH syndrome
  - Excess ADH
    - May be temporary, triggered by stress; may be secreted by an ectopic source, such as a tumor
  - Treatment
    - Diuretics
    - Sodium supplements
Hypothalamus-Pituitary-Thyroid Gland Feedback

1. STIMULUS (cold or stress)

2. HYPOTHALAMUS increases secretion of TRH into blood

3. PITUITARY glandular cells increase secretion of TSH into circulation

4. THYROID GLAND increases secretion of T<sub>3</sub> and T<sub>4</sub> into blood

5. TARGET CELLS increase metabolism

6. NEGATIVE FEEDBACK High levels of T<sub>3</sub> and T<sub>4</sub> inhibit secretion of TSH and TRH
Goiter—enlargement of thyroid gland

- Endemic goiter
  - Hypothyroid condition in regions with low iodine levels in soil and food

- Goitrogens
  - Foods that contain elements to block synthesis of triiodothyronine (T₃) and thyroxine (T₄)

- Toxic goiter
  - Results from hyperactivity of thyroid gland
Endemic Goiter
Thyroid Disorders: Hyperthyroidism

- Hyperthyroidism (Graves’ disease)
  - Related to autoimmune factor
  - Hypermetabolism and increased stimulation of SNS
    - Increased body temperature
    - Sweating
    - Soft silky hair and skin
    - Reduced BMI
    - Insomnia
    - Hyperactivity
Thyroid Disorders: Hyperthyroidism (Cont.)

- Toxic goiter
- Exophthalmos
  - Presence of protruding, staring eyes, decreased blink and eye movement
  - Result of increased tissue mass in the orbit
  - May result in visual impairment
Exophthalmos
Hypothyroidism

- Hypothyroidism
  - Iodine deficit
  - Hashimoto’s thyroiditis
    - Autoimmune disorder
  - Tumor
  - Surgical removal or treatment of gland
  - Cretinism
    - Results in short stature and severe cognitive deficits
    - Untreated congenital hypothyroidism
    - May be related to iodine deficiency during pregnancy
Hypothyroidism Manifestations

- Goiter if cause is endemic iodine deficiency
- Intolerance to cold
- Increased BMI
- Lethargy and fatigue
- Decreased appetite
- Myxedema in severe, untreated hypothyroidism
  - Nonpitting edema in face, thickened tongue
  - Myxedema coma—acute hypotension, hypoglycemia, and hypothermia result in loss of consciousness; life-threatening if untreated
## Comparison of Hypothyroidism and Hyperthyroidism

<table>
<thead>
<tr>
<th></th>
<th>Hypothyroidism</th>
<th>Hyperthyroidism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum levels of $T_3$ and $T_4$</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td>Metabolic rate</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td>Goiter</td>
<td>Present with endemic goiter</td>
<td>Present with Graves’ disease</td>
</tr>
<tr>
<td>Skin</td>
<td>Pale, cool, with edema</td>
<td>Flushed and warm</td>
</tr>
<tr>
<td>Temperature tolerance</td>
<td>Cold intolerance</td>
<td>Heat intolerance</td>
</tr>
<tr>
<td>Eyes</td>
<td>No changes</td>
<td>Exophthalmos with Graves’ disease</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Bradycardia, enlarged heart</td>
<td>Tachycardia, increased blood pressure</td>
</tr>
<tr>
<td>Nervous system</td>
<td>Lethargic, slow intellectual functions</td>
<td>Restless, nervous, tremors</td>
</tr>
<tr>
<td>Body weight</td>
<td>Some weight increase with decreased appetite</td>
<td>Thin, but increased appetite</td>
</tr>
</tbody>
</table>
Adrenal Glands

- Adrenal medulla
  - Pheochromocytoma
    - Benign tumor of the adrenal medulla—secretes epinephrine, norepinephrine, and possibly other substances
    - Occasionally, multiple tumors
    - Headache, heart palpations, sweating, intermittent or constant anxiety
Adrenal Glands: Adrenal Cortex

- Cushing’s syndrome
  - Caused by an excessive level of glucocorticoids;
  - possible result of:
    - Adrenal adenoma
    - Pituitary adenoma
    - Ectopic carcinoma
    - Iatrogenic conditions
    - Substance abuse
Cushing’s Syndrome

- Changes associated with Cushing’s syndrome
  - Change in person’s appearance
    - Round face, with ruddy color
    - Truncal obesity, with fat pad between scapulae
    - Thin limbs
    - Thin hair
    - Fragile skin, striae
Cushing’s Syndrome (Cont.)

- Retention of sodium and water
- Suppression of the immune response
- Stimulation of erythrocyte production
- Emotional lability and euphoria
- Increased catabolism of bone and protein
- Delayed healing
- Increased insulin resistance and possible glucose intolerance
Cushing’s Syndrome (Cont.)

- Mood swings, insomnia, and loss of libido
- Supraclavicular fat pad
- Buffalo hump
- Thinning hair
- Moon face and ruddy complexion
- Hirsutism
- Truncal obesity with pendulous breasts and abdomen
- Broad purple striae
- Thinning pubic and axillary hair in women
- Ecchymoses
- Impaired wound healing and immune response
- Thin, fragile skin
Addison’s Disease

- Deficiency of adrenocorticotoid secretions
- Autoimmune reaction is a common cause.
- Adrenal gland may be destroyed by hemorrhage or infection
Addison’s Disease (Cont.)

● Manifestations
  ➢ Decreased blood glucose levels
  ➢ Inadequate stress response
  ➢ Fatigue
  ➢ Weight loss, frequent infections
  ➢ Low serum sodium concentration
    • Decreased blood volume
    • Hypotension
    • High potassium levels
### Comparison of Addison’s Disease and Cushing’s Syndrome

<table>
<thead>
<tr>
<th>Addison’s Disease (Adrenal Insufficiency)</th>
<th>Cushing’s Syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deficit of corticosteroids&lt;br&gt;(glucocorticoids, mineralocorticoids)</td>
<td>Excess glucocorticoids&lt;br&gt;(cortisol)</td>
</tr>
<tr>
<td>High risk of infection</td>
<td>High risk of infection</td>
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<tr>
<td>Poor stress response</td>
<td>Poor stress response</td>
</tr>
<tr>
<td>Weight loss, fatigue</td>
<td>Moon face, buffalo hump, obese trunk, muscle wasting in limbs, osteoporosis</td>
</tr>
<tr>
<td>Anorexia, nausea, diarrhea</td>
<td>Striae, bruising of skin, high risk of infection</td>
</tr>
<tr>
<td>Hypotension, syncope</td>
<td>Hypertension, glucose intolerance</td>
</tr>
<tr>
<td>Hyperpigmentation</td>
<td>Fatigue, weakness, delayed healing</td>
</tr>
</tbody>
</table>