BIOS 2015 … CHAPTER 16- Endocrine Disorders

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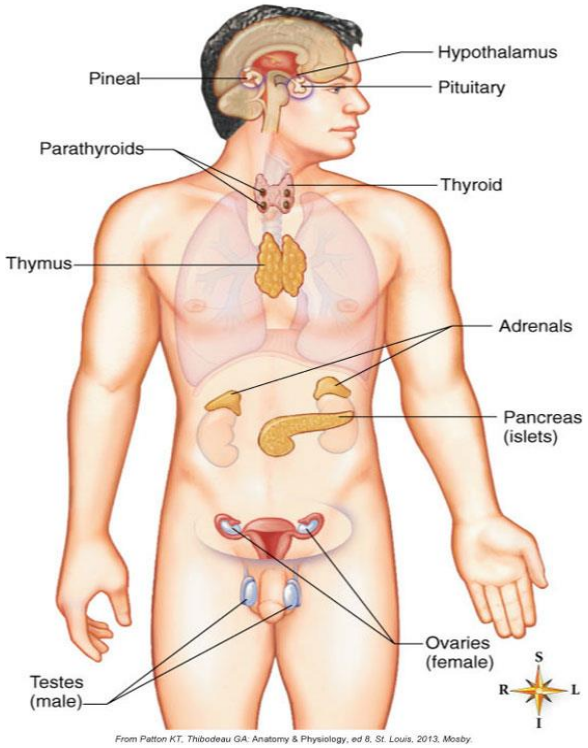
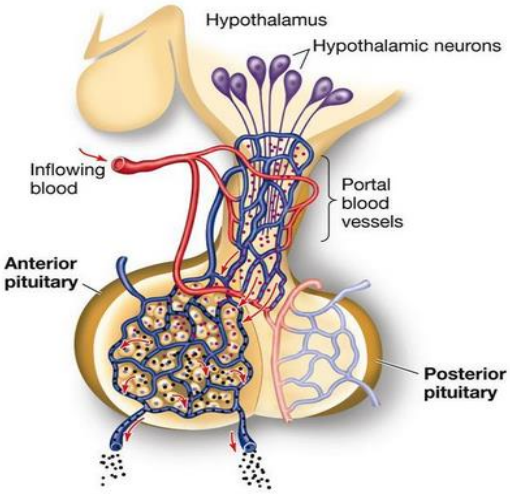
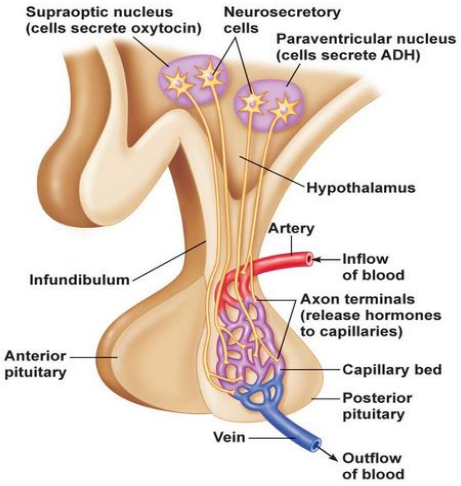
	 From Patton KT, Thibodeau GA: Anatomy & Physiology, ed 8, St. Louis, 2013, Mosby. Endocrine vs Exocrine **Exocrine Glands** produce a secretion that is dispensed locally via a duct into a cavity (saliva into mouth, pancreatic juice into small intestine). **Endocrine Glands** produce hormones that are released into the blood stream an act at distant sites from the gland. the target tissue cells have receptors for the hormone. negative feedback systems are often in place. hormones are peptides or steroids.
	Two types of hormones:
	1. Steroid (a polycyclic lipid) passes through cell membranes and enters nucleus to initiate transcription.
	2. Peptides must bind a cell surface receptor and transduce a signal.

Pituitary Hormones





Hypothalamic neurons release hormone in the stalk and it is carried by the hypothalamic-hypophyseal portal vessels to the anterior pituitary. These are releasing and inhibiting hormones that regulate release of the anterior pituitary hormones.
Example: GHRH, growth hormone releasing hormone, is released by the hypothalamus to cause the anterior pituitary to release growth hormone.

Hypothalamic neurons release hormone directly into the posterior pituitary to be picked up by capillaries there.

ANTERIOR HORMONES

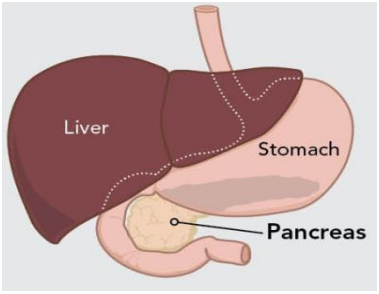
Growth hormone (GH, somatotropin)
Stimulates protein synthesis
Adrenocorticotropic hormone (ACTH)
Stimulates adrenal cortex to secrete primarily cortisol
Thyroid-stimulating hormone (TSH)
Stimulates thyroid gland
Follicle-stimulating hormone (FSH)
Women: stimulates growth of ovarian follicles and estrogen secretion; men: stimulates sperm production
Luteinizing hormone (LH)
Women: stimulates maturation of ovum and ovulation; men: stimulates secretion of testosterone
Prolactin (PRL)

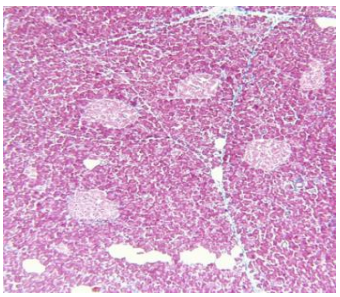
POSTERIOR HORMONES

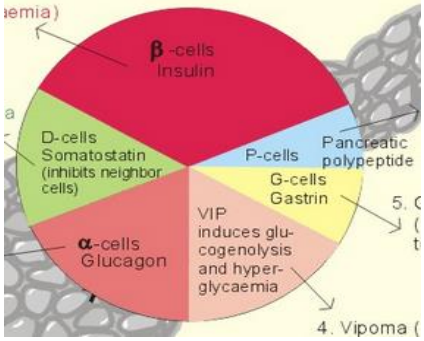
Antidiuretic hormone
(ADH, or vasopressin):
Increases reabsorption of water in kidney

Oxytocin (OT):
Stimulates contraction of uterus after delivery

PANCREAS

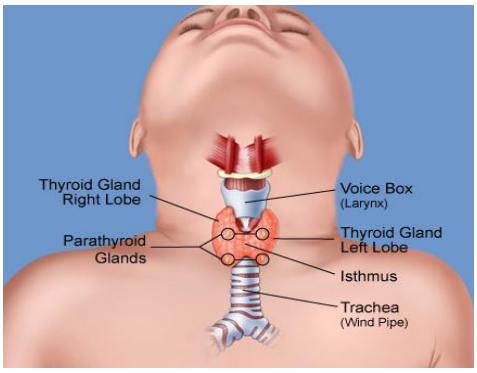






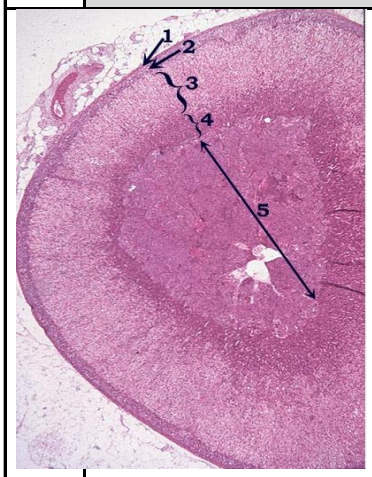
Insulin	Pancreas—beta cells of islets of Langerhans	Transport of glucose and other substances into cells Lowers blood glucose level
Glucagon	Pancreas—alpha cells	Glycogenolysis in liver Increases blood glucose level

Thyroid and Parathyroid Glands

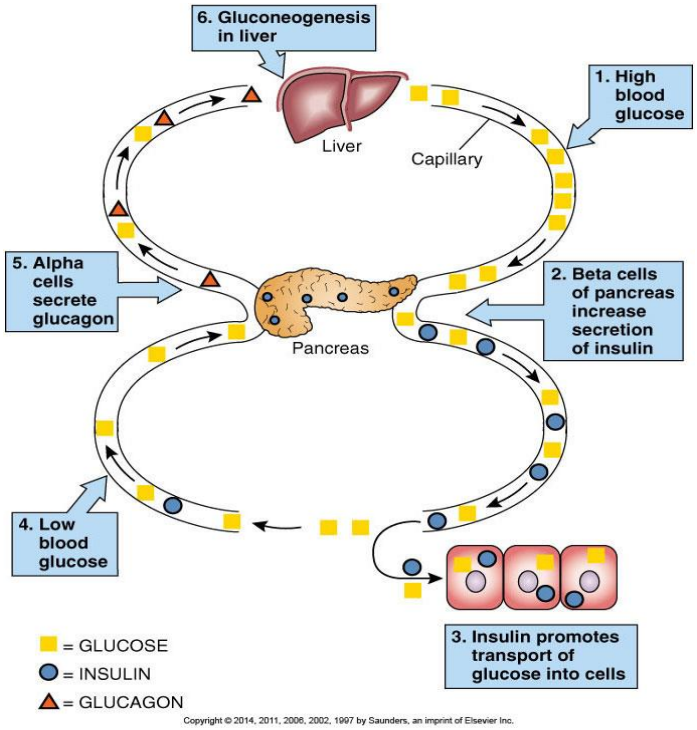


Parathyroid hormone (PTH)	Parathyroid gland	Increases blood calcium level by stimulating bone demineralization and increasing absorption of Ca^{++} in the digestive tract and kidneys
Calcitonin	Thyroid gland	Decreases release of calcium from the bone to lower blood calcium level

ADRENAL GLAND



Aldosterone (a mineralocorticoid)	Adrenal cortex	Increases sodium and water reabsorption in the kidney
Cortisol (a glucocorticoid)	Adrenal cortex	Anti-inflammatory and decreases immune response Catabolic effect on tissues; stress response
Norepinephrine	Adrenal medulla	General vasoconstriction
Epinephrine	Adrenal medulla	Adrenal medulla Stress response Visceral and cutaneous vasoconstriction Vasodilation in skeletal muscle Increases rate and force of heart contraction Bronchodilation



Negative Feedback:
High Glucose > Insulin release //
Low glucose inhibits insulin release.

Complementary Systems:
Insulin lowers blood glucose //
Glucagon raises blood glucose.

Note:
A system fails if:
1. not enough hormone is produced,
or
2. if the receptors that the hormone must bind to on the target cells is faulty

Hormones are tested by:

1. Direct measure of hormone in the blood.
2. Stimulation test, example: give ACTH and measure plasma cortisol (should rise).
3. Suppression test, example: give cortisol and measure ACTH levels (should drop)

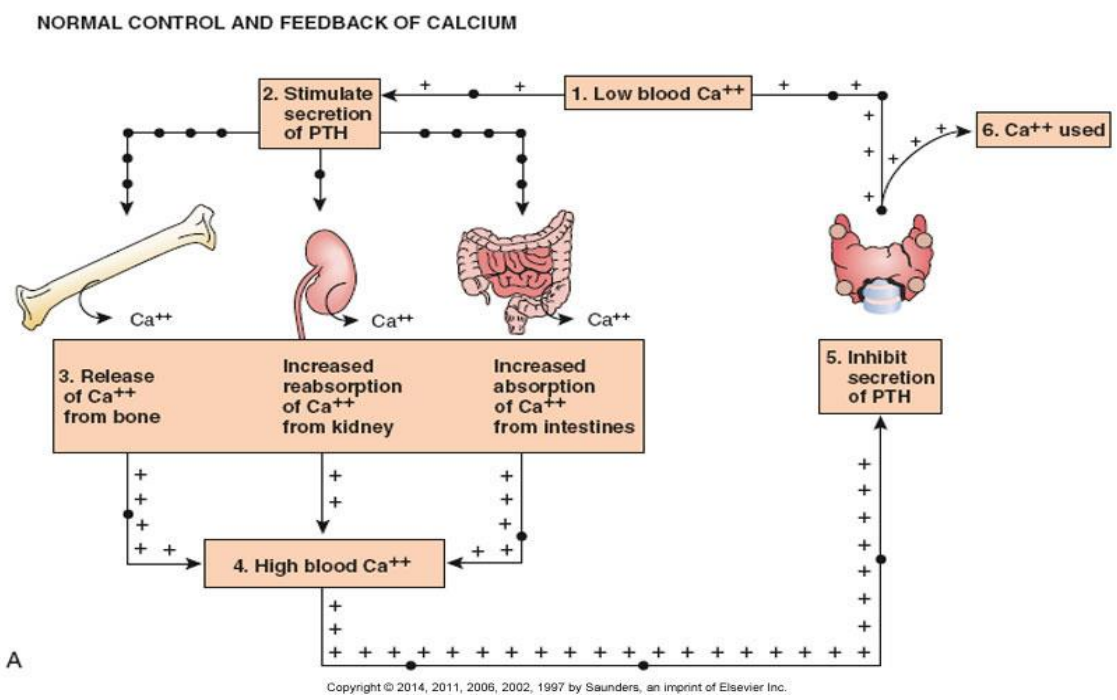
Treat with replacement therapy when hormone is low or absent.

	Diabetes Mellitus
	Diabetes mellitus—basic problem is inadequate insulin effects in receptor tissues - Deficit of insulin secretion from pancreas. - Cells do not respond to insulin (insulin resistance) 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
	Type 1 **Autoimmune destruction of beta cells in pancreas** Insulin replacement required IDDM (insulin dependent diabetes mellitus) Acute onset in children and adolescents Not linked to obesity Genetic factors may play a role.
	Type 2 **Non Insulin Dependent Diabetes Mellitus (NIDDM).** Onset is slow and insidious, usually in those older than 50 years Increasing incidence in teens and young adults Caused by decreased production of insulin and/or increased resistance by body cells to insulin Associated with obesity Oral hypoglycemic medications may be used.
	General Manifestations:
	Blood glucose levels rise—hyperglycemia Excess glucose in urine—glucosuria - Dehydration results from hyperosmolar filtrate. - Polyuria (increased urination) - Polydipsia (increased drinking) Insulin deficit results in decreased transport and use of glucose in many cells. - Polyphagia (increased appetite). - Fatigue
	Diagnostic Tests:
	Fasting blood glucose level **Glucose tolerance test** - Give oral glucose - Watch for rise and fall in blood **Glycosylated hemoglobin test** - Hemoglobin A1c - 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 **Cells can not access glucose so they burn fats and proteins.** Catabolism of fats and proteins produces: - **Excessive amounts of fatty acids** and metabolites - **Ketones** (acetoacetic acid and β-hydroxybutyrate) increase in blood and subsequently in urine. **- Ketoacidosis and Ketonuria.** - Also known as DKA (diabetic ketoacidosis).
	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.**
	Acute Complications:
	Hypoglycemia (insulin shock) - More common with insulin replacement treatment (taking too much or not eating after taking) 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: 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.**

	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
	Signs and Symptoms of Diabetic Ketoacidosis
	Dehydration - Thirst, dry, rough oral mucosa - Warm, dry skin **Rapid, deep respiration—acetone breath** - Trying to blow off CO_2 to resolve acidosis. **Metabolic acidosi**s - May lead to loss of consciousness **Electrolyte imbalances** - Abdominal cramps, nausea, vomiting, lethargy, weakness
	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
	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
	Infections Common and often more severe in diabetics Infections in feet and legs caused by vascular and neurological impairment, poor wound healing. Fungal infections common - Caused by Candida - In vagina and/or oral cavity Urinary tract infections Dental caries Gingivitis and periodontitis
	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 (macrosomia) and weight for date - May experience hypoglycemia in first hours postnatally

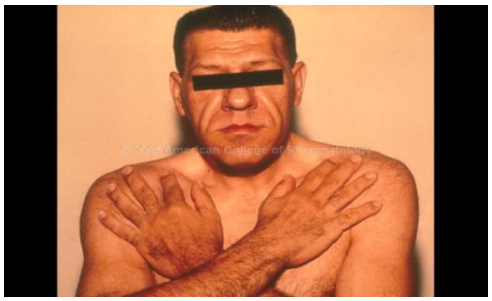
	CALCIUM - control and feedback
	
	Parathyroid Hormone and Calcium
	Hypoparathyroidism **Leads to hypocalcemia** - Weak cardiac muscle contractions - Increased excitability of nerves—spontaneous contractions of skeletal muscle **Causes** - Congenital lack of parathyroid - Surgery or radiation in neck region - Autoimmune disease - Tumor in bone producing hypercalcemia leads to negative feedback on parathyroid hormone - production.
	Hyperparathyroidism **Results in hypercalcemia** - Forceful cardiac contractions - Osteoporosis - Predisposition to kidney stones **Causes** - Parathyroid Hyperplasia - Paraneoplastic syndrome (PTH secreting tumor). - Secondary to renal failure Decreased activation of vitamin D > decreased calcium absorption > elevated PTH Increased Phosphate retention > hypocalcemia > elevated PTH

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 (pressure on optic chiasm > tunel vision)

Effect on hormone secretion
- Dependent on cells and location involved
- May cause excessive or decreased release of hormones

Acromegaly:
Big hands, Large Mandible



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

Antidiuretic Hormone (ADH)

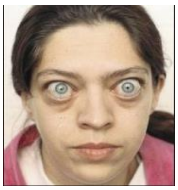
Diabetes insipidus—Deficit of ADH
- Adenoma
- May originate in the neurohypophysis
  Head injury or surgery
  Possible genetic problem
  Replacement treatment required

Inappropriate ADH syndrome - Excess ADH
- May be temporary, triggered by stress; may be secreted by an ectopic source, such as a tumor
- Produces fluid retention and hyponatremia.
- Treatment
  Diuretics and Sodium supplements

THYROID DISORDERS - Hyperthyroidism

Hyperthyroidism (Graves' disease)
Autoimmune disease with antibodies that mimic TSH and stimulate the TSH receptors in the thyroid resulting in elevated T3 and T4.

Hypermetabolism and increased stimulation of SNS
- Increased body temperature
- Sweating
- Soft silky hair and skin
- Reduced BMI
- Insomnia
- Hyperactivity

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

Hypothyroidism, causes include
Iodine deficit
Hashimoto's thyroiditis
- Autoimmune disorder, destruction of thyroid tissue.
Tumor
- Surgical removal or treatment of gland
Cretinism
- Untreated congenital hypothyroidism
- Results in short stature and severe cognitive deficits
- May be related to iodine deficiency during pregnancy

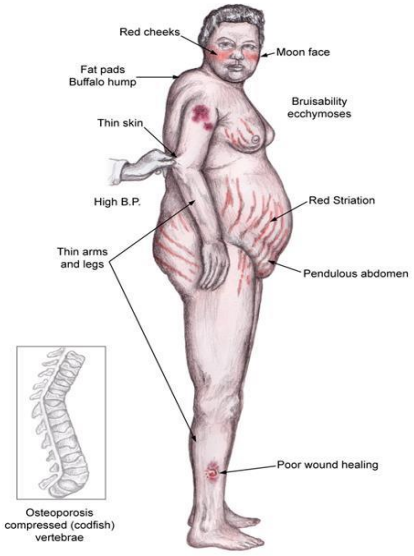
Hypothyroidism Manifestations

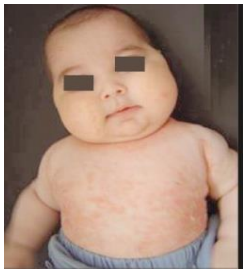
Goiter if cause is endemic iodine deficiency
Intolerance to cold
Increased BMI
Lethargy and fatigue
Decreased appetite
Myxedema in severe, untreated hypothyroidism
- Nonpitting edema in face, thickened tongue
- Myxedema coma—acute hypotension, hypoglycemia, and hypothermia result in loss of consciousness;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

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, severe hypertension.

Cushing's syndrome
Caused by an excessive level of glucocorticoids; possible result of:
- Adrenal adenoma (making cortisol)
- Pituitary adenoma (making ACTH > cortisol)
- Ectopic carcinoma (making hormone).
- Iatrogenic conditions (glucocorticoid-steroid administration, cortisol and hydrocortisone)





Changes associated with Cushing's syndrome

Change in person's appearance
- Round "moon" face, with ruddy color
- Truncal obesity, with fat pad between scapulae, "buffalo hump"
- Thin limbs
- Thin hair
- Fragile skin, striae

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

Addison's Disease

Deficiency of adrenocorticoid secretions
Both glucocorticoid – cortisol and mineralocorticoid – aldosterone.
Autoimmune reaction is a common cause.
Adrenal gland may be destroyed by hemorrhage or infection

Manifestations of Addison's Disease
Decreased blood glucose levels
Inadequate stress response
Fatigue
Weight loss, frequent infections
Low serum sodium concentration and loss of water retention.
- Decreased blood volume
- Hypotension
- High potassium levels