Learning Objectives

• Describe how hormones secreted from the endocrine glands help body maintain homeostasis.
• Describe anatomy and physiology of pancreas and how its hormones maintain normal glucose metabolism.

Endocrine System Anatomy and Physiology

• Composed of ductless glands and tissues that produce and secrete hormones
  – Major endocrine glands
  – Pituitary
  – Thyroid
  – Parathyroid glands
  – Adrenal cortex and medulla
  – Pancreatic islets
  – Ovaries and testes
• Other specialized groups of cells that secrete hormones are found in kidneys and mucosa of GI tract
Endocrine Gland Functions

- Secrete hormones directly into bloodstream and regulate various metabolic functions
- Products of endocrine glands travel via blood (or tissue fluids)
  - Able to exert effects at widespread sites, often distant from source of origin

Endocrine Gland Functions

- Endocrine hormones are released
  - In response to change in cellular environment
  - To maintain normal level (i.e. homeostasis) of hormones, other substances
  - To simulate or inhibit organ functions

Endocrine Gland Functions

- This integrated chemical and coordination system enables
  - Reproduction
  - Growth and development
  - Regulation of energy
- Target organs and body tissues have hormone receptors and are able to respond to a particular hormone
How are hormones and their target organs like a lock and key?

Hormone Receptors

- Hormone categories
  - Proteins
  - Polypeptides
  - Derivatives of amino acids
  - Lipids
- Each hormone may affect specific organ or tissue, or can have general effect on entire body

Hormone Receptors

- Also classified as steroid or nonsteroid
- Steroid hormones
  - Synthesized by endocrine cells from cholesterol
    - Cortisol
    - Aldosterone
    - Estrogen
    - Progesterone
    - Testosterone
Hormone Receptors

- Nonsteroid hormones
  - Synthesized chiefly from amino acids
    - Insulin
    - Parathyroid hormone

Hormone Receptors

- Hormones affect only cells with appropriate receptors
  - Act on cells to initiate specific cell functions or activities
  - Hormone receptor sites may be on outside of cell membrane or in interior of cell
  - Cells with fewer receptor sites bind with less hormone than cells with many receptor sites
  - In addition, abnormalities in presence or absence of specific hormone receptors can result in endocrine disorders
Hormone Secretion Regulation

- Hormones operate with feedback systems
  - Either positive or negative
  - Help to maintain optimal internal environment

Hormone Secretion Regulation

- Negative feedback is mechanism most commonly used to maintain homeostasis
  - Example: after person eats a candy bar
    - Glucose from ingested lactose or sucrose is absorbed in intestine
    - Consequently, level of glucose in blood rises
    - Increase in blood glucose concentration stimulates pancreas to release insulin
    - Insulin facilitates entry of glucose into cells; as result, blood glucose level falls
    - When blood glucose level has dropped sufficiently, endocrine cells in pancreas stop producing and releasing insulin
Disorders of Endocrine System

• Arise from effects of
  – Imbalance in production of one or more hormones
  – Change in body’s ability to use hormones produced

• Clinical effects of endocrine gland disorders are determined by
  – Degree of dysfunction
  – Age and sex of affected person

Pancreas Disorder: Diabetes Mellitus

• Systemic disease of the endocrine system
  – Usually results from dysfunction of pancreas
  – Complex disorder of fat, carbohydrate, and protein metabolism that affects more than 24 million adults in U.S.
  – Another 57 million people have prediabetes
  – Potentially lethal
  – Can put patient at risk for several kinds of true medical emergencies

Pancreas Anatomy and Physiology

• Pancreas
  – Important in absorption and use of carbohydrates, fat, protein
  – Chief regulator of glucose levels in blood
  – Located retroperitoneally adjacent to duodenum on right and extending to spleen on left
  – Healthy pancreas has exocrine and endocrine functions
Pancreas Anatomy and Physiology

• Exocrine glands
  - Secrete substances through duct onto inner surface of organ or outer surface of body
  - Portion consists of acini (glands that produce pancreatic juice) and duct system
    • Duct system carries the pancreatic fluids to the small intestine
• Endocrine glands secrete chemicals directly (not through duct) into bloodstream
  - Portion consists of pancreatic islets (islets of Langerhans) that produce hormones

Islets of Langerhans and Pancreatic Hormones

• About 500,000 to 1 million pancreatic islets are dispersed among ducts and acini of pancreas
  - Each islet composed of
    • Beta cells: produce and secrete insulin
    • Alpha cells: produce and secrete glucagon
    • Delta cells: produce somatostatin
Islets of Langerhans and Pancreatic Hormones

- Somatostatin
  - Inhibits secretion of growth hormone and TSH
  - Inhibits secretion of insulin and glucagon
  - Acts as buffer to avoid rapid swings in blood glucose levels
  - Nerves from both divisions of ANS innervate pancreatic islets
    - Each islet is surrounded by well-developed capillary network

If part of the pancreas must be removed because it has undergone trauma, will the patient still be able to produce insulin and glucagon?

Insulin

- Small protein
  - Released by beta cells when blood glucose levels rise
- Main functions
  - Increase glucose transport into cells
  - Increase glucose metabolism by cells
  - Increase liver glycogen levels
  - Decrease blood glucose concentration toward normal
- Functions antagonize effects of glucagon
Insulin

- Glucagon
  - Protein released by alpha cells when blood glucose levels fall
  - Effects
    - Increase blood glucose levels
    - Glycogenolysis
      - Stimulates liver to release glucose stores from glycogen and other glucose storage sites
    - Stimulates gluconeogenesis (glucose formation) through breakdown of fats and fatty acids, thereby maintaining normal blood glucose level

Growth Hormone

- Polypeptide hormone
- Produced and secreted by anterior pituitary gland
  - Secretion is triggered by physiological stimuli
    - Exercise
    - Stress
    - Sleep
    - Hypoglycemia
Growth Hormone

- Acts as insulin antagonist
  - Decreases insulin actions on cell membranes
  - Reduces capacity of muscles and adipose and liver cells to absorb glucose

Glucose Metabolism Regulation

- Under normal conditions, body maintains serum glucose level in blood at 60 to 120 mg/dL
  - Normal fasting blood glucose is < 100 mg/dL
- Understanding food intake and digestion required to understand glucose metabolism

Dietary Intake

- Three main organic components of food
  - Carbohydrates
  - Fats
  - Proteins
Dietary Intake

- Carbohydrates
  - Found in all sugary, starchy foods
  - Ready source of near-instant energy
  - First food substances to enter bloodstream after meal is ingested
  - Yield simple sugar glucose
  - If not “burned” for immediate energy, glucose is stored
    - In liver and muscles as glycogen for short-term energy needs
    - In adipose tissue for intermediate and long-term needs

Digestion Process

- Before food compounds can be used by body cells, must be digested and absorbed into bloodstream
- Digestion begins in mouth
  - Accomplished by physical forces (chewing) and chemical (enzymatic) forces
  - Begins process that reduces food to soluble molecules and particles small enough to be absorbed

Digestion Process

- After food is swallowed, it enters stomach
  - There, various nutrients are absorbed into circulatory system
    - Glucose
    - Salts
    - Water
    - Other substances (alcohol and certain other drugs)
  - Remaining material (chyme) is shunted from stomach into intestine for further digestion
Digestion Process

- Duodenum signals release of hormones that mobilize pancreas to contribute its molecule-splitting enzymes and gallbladder to release bile salts
  - Enzymes and salts neutralize acids and help emulsify fats

Digestion Process

- Carbohydrates are absorbed as simple sugars
- Fats are absorbed as fatty acids and glycerol
- Proteins are absorbed as amino acids
- All are carried from intestine to liver by way of portal vein

Digestion Process

- Water and remaining salts are absorbed from food residues in colon
- Liver synthesizes glycogen from absorbed glucose, lipoproteins from absorbed fatty acids, many proteins required for health from absorbed amino acids
Why do diabetics eat carbohydrates instead of proteins or fats when they sense that their glucose levels are low?

Carbohydrate Metabolism

- Secretion of insulin is controlled by chemical, neural, and hormonal means
- Insulin release from pancreas beta cells after dietary increase of carbohydrates caused by
  - Increased concentration of blood glucose
  - Parasympathetic stimulation
  - GI hormones involved with regulation of digestion

Carbohydrate Metabolism

- Insulin travels through blood to target tissues
  - There it combines with specific chemical receptors on surface of cell membrane to permit glucose to enter cell
- Allows cells to use glucose for energy
  - Prevents breakdown of alternative energy sources (proteins and fat cells)
  - Promotes uptake of glucose into liver, converted to glycogen for storage
  - Prevents large increase in blood glucose levels, even just after normal meals
Carbohydrate Metabolism

• When blood glucose level begins to fall, liver releases glucose back into circulating blood
  – Liver removes excess glucose from blood after meal
  – Returns it to blood when it is needed between meals
  – Under normal circumstances, about 60 percent of glucose in meal is stored in liver as glycogen and released later

Carbohydrate Metabolism

• If muscles are not exercised after meal, much of the glucose transported into muscle cells by insulin is stored as muscle glycogen
  – Muscle glycogen differs from liver glycogen
    • Cannot be reconverted into glucose and released into circulation
    • Stored glycogen must be used by muscle for energy

Carbohydrate Metabolism

• Brain is different from other body tissues with regard to glucose uptake
  – Insulin has little/no effect on uptake or use of glucose by brain
  – Cells of brain do not have adequate storage capacity
  – Brain normally uses only glucose for energy, cannot depend on stored supplies of glycogen
    • Serum glucose must be maintained at level that provides adequate energy to these tissues
Carbohydrate Metabolism

- When serum glucose level falls too low, signs and symptoms of hypoglycemia can develop quickly
  - Progressive irritability
  - Altered mental status
  - Fainting
  - Convulsions
  - Coma

Fat Metabolism

- Only limited amount of glycogen can be stored in liver and skeletal muscles
  - 1/3 of any glucose passing through liver is converted to fatty acids
  - Under influence of insulin, fatty acids are converted to triglycerides (storable fats)
    - Stored in adipose tissue

Fat Metabolism

- In absence of insulin, stored fat is broken down
  - Plasma concentration of free fatty acids rapidly increases
  - Low level of insulin in blood can result in high levels of triglycerides and cholesterol (in form of lipoproteins) in plasma
  - This is thought to contribute to development of atherosclerosis in patients with serious diabetes
Fat Metabolism

- If needed (as in absence of insulin), fatty acids in liver can be metabolized and used for energy
- Byproduct of breakdown of fatty acids in liver is acetate
  - Converted to acetoacetic acid and beta hydroxybutyric acid
    - Released into circulating blood as ketone bodies
    - Ketone bodies may cause acidosis and coma (diabetic ketoacidosis) in diabetic patients

Protein Metabolism

- Insulin causes proteins, carbohydrates, fats, to be stored
- Amino acids
  - Actively transported into various cells of body
  - Most used as building blocks to form new proteins (protein synthesis)
    - Some enter metabolic cycle by being converted to glucose after initial breakdown in liver

Protein Metabolism

- In absence of insulin
  - Protein storage stops, protein breakdown (particularly in muscle) begins
    - Releases large amounts of amino acids into circulation
    - Excess amino acids are used directly for energy or as substrates for gluconeogenesis
    - Degradation of amino acids leads to increased urea excretion in urine
    - This “protein wasting” leads to extreme weakness and dysfunction of many organs
Functions of Glucagon

- Functions opposite of that of insulin
  - Increase blood glucose concentration
  - Glucagon has two major effects on glucose metabolism
    - Breakdown of liver glycogen (glycogenolysis)
    - Generation of glucose (gluconeogenesis)

- As serum glucose level returns to normal (several hours after dietary intake), insulin secretion decreases with continued fasting
  - Blood sugar level begins to drop
  - Glucagon, cortisol, GH, and epinephrine (from sympathetic stimulation) are secreted
    - Initiates release of glucose from glycogen and other glucose-storage sites
    - Glycogen is converted back to glucose and released into blood
    - Uptake of glucose by most tissues helps to maintain blood glucose at levels necessary for normal function
What signs and symptoms will the patient have in response to the release of epinephrine when blood glucose falls?

**Functions of Glucagon**

- Four mechanisms for achieving adequate blood glucose regulation
  - Liver functions as blood glucose buffer system
    - Removes glucose from blood when in excess (stores it as glycogen)
    - Returns glucose to blood when glucose concentration and insulin secretion decline

**Functions of Glucagon**

- Four mechanisms for achieving adequate blood glucose regulation
  - Insulin and glucagon function as negative feedback control system
    - Work to maintain normal serum glucose concentrations
    - When serum glucose level rises, insulin is secreted to lower it toward normal
    - When serum glucose level falls, glucagon is secreted to raise serum glucose level toward normal
Functions of Glucagon

- Four mechanisms for achieving adequate blood glucose regulation
  - Low serum glucose levels stimulate sympathetic nervous system to secrete epinephrine
    - Have glucagon-like effect that promotes liver glycogenolysis
  - GH and cortisol play role in less immediate regulation of serum glucose levels
    - Secreted in response to more prolonged hypoglycemic episodes
    - Increase rate of glucose production
    - Tend to decrease rate of glucose use

Lesson 26.2
Diabetes and Diabetic Emergencies

Learning Objective

- Discuss pathophysiology as a basis for key signs and symptoms, patient assessment, and patient management for diabetes and diabetic emergencies of hypoglycemia, diabetic ketoacidosis, and hyperosmolar hyperglycemic nonketotic syndrome.
Diabetes Mellitus Pathophysiology

- Diabetes is seventh leading cause of death in U.S.
- Characterized by deficiency of insulin or by inability of body to respond to insulin
- Often associated with
  - Increased intake of fluid (polydipsia)
  - Excretion of large quantities of urine that contains glucose (polyuria, glucosuria)
  - Weight loss

Diabetes Mellitus Pathophysiology

- Classified as type 1 or type 2
  - Type 1 diabetes
    - Previously called insulin-dependent diabetes mellitus (IDDM) or juvenile diabetes
  - Type 2
    - Previously called non-insulin-dependent diabetes mellitus (NIDDM) or adult-onset diabetes

Diabetes Mellitus Pathophysiology

- New classification system endorsed by American Diabetes Association and World Health Organization identifies four types of diabetes
  - Type 1
  - Type 2
  - Gestational diabetes
  - “Other specific types,” to address continuum of hyperglycemia (elevated blood glucose) and insulin requirements
Type 1 Diabetes

• Characterized by inadequate production of insulin by pancreas
  – May occur any time after birth
  – Usually occurs in teenagers and young adults, typically “peaks” at 12 years of age
  – Heredity is factor
  – Appears to be autoimmune phenomenon
  – Results from genetic abnormality or susceptibility that causes body to destroy its own insulin-producing cells

Type 1 Diabetes

• Person with parent or sibling with type 1 diabetes has 10 percent chance of developing disease by age 50
• Requires lifelong treatment with insulin, exercise, diet regulation

Type 1 Diabetes

• Symptoms usually appear suddenly
  – Polyuria
  – Polydipsia
  – Dizziness
  – Blurred vision
  – Rapid, unexplained weight loss
### Type 2 Diabetes

- **Characterized by decrease in production of insulin by pancreatic beta cells and diminished tissue sensitivity to insulin (insulin resistance)**
- **Insulin resistant**
  - Have either too few insulin receptors or faulty insulin receptors
  - Circulating insulin cannot be properly utilized
  - Majority of people with type 2 are insulin resistant
  - Occurs most often in adults who are 40+ years of age, minorities, overweight

### Type 2 Diabetes

- **Obesity predisposes person**
  - Larger amounts of insulin are needed for metabolic control in obese individuals than in those with normal weight
  - Accounts for 90 to 95% of all diagnosed cases
  - Increase in childhood obesity: growing number of children and young adults being diagnosed

### Type 2 Diabetes

- **Most patients require oral hypoglycemic medications, exercise, and dietary regulation to control illness**
  - Small number require insulin
  - Warning signs (if present) are gradual
    - All those associated with type 1 diabetes
    - Fatigue
    - Changes in appetite
    - Tingling
    - Numbness
    - Pain in extremities
Would patients with type 1 or type 2 diabetes have an increased risk of complications related to this disease?

Gestational Diabetes Mellitus

- Develops in some women during late pregnancy
  - Usually resolves with childbirth
  - Some women will go on to develop type 2 diabetes within 5 to 10 years

Other Types of Diabetes

- Less common, caused by
  - Genetic defects of beta cells of pancreas
  - Genetic defects in insulin action, resulting in body’s inability to control blood glucose levels
  - Diseases of pancreas or conditions that damage pancreas, such as pancreatitis and cystic fibrosis
  - Excess amounts of certain hormones resulting from some medical conditions (e.g., cortisol in Cushing’s syndrome) that work against action of insulin
Other Types of Diabetes

- Caused by
  - Medications that reduce insulin action, such as glucocorticoids, or chemicals that destroy beta cells
  - Infections, such as congenital rubella and cytomegalovirus
  - Rare immune-mediated disorders
  - Genetic syndromes associated with diabetes, such as Down syndrome

Diabetes Effects

- Most can be attributed to one of three effects of decreased insulin levels
  - Decreased use of glucose by body cells, with resultant increase in serum glucose level
  - Markedly increased mobilization of fats from fat storage areas, causing abnormal fat metabolism, which may result in short term in ketoacidosis and in long term in severe atherosclerosis
  - Depletion of protein in body tissues and muscle wasting

Glucose Loss in Urine

- When amount of glucose entering kidneys rises above kidneys’ ability to reabsorb it, significant portion of glucose “spills” into urine
  - Loss of glucose in urine causes diuresis
    - Osmotic effect of glucose prevents kidneys from reabsorbing water (osmotic diuresis)
    - Effect is dehydration
    - If untreated, dehydration can lead to hypovolemic shock
Acidosis in Diabetes

- Shift from carbohydrate to fat metabolism results in formation of ketone bodies (ketoacids)
  - Acids and their continuous production leads to metabolic acidosis
  - Often respiratory system at least partly compensates for acidosis (indicated by Kussmaul respirations)
  - Kidneys’ ability to clear acid is overwhelmed by continuous production of ketone bodies

Acidosis in Diabetes

- Profound acidosis eventually occurs
  - This acidosis, along with the usually severe dehydration that occurs as a result of osmotic diuresis, can lead to death
  - Hyperkalemia, secondary to acidosis, also leads to cardiac dysrhythmias (some that may be lethal)
  - Treatment of this condition can be lifesaving

Acidosis in Diabetes

- Diabetes mellitus is systemic disease with many long-term complications
  - Blindness
    - 5,000 diabetics lose their sight each year
  - Kidney disease
    - 10 percent of all diabetics develop some form of kidney disease, including end-stage kidney failure, which requires dialysis or kidney transplant
Acidosis in Diabetes

- Diabetes mellitus is systemic disease with many long-term complications
  - Peripheral neuropathy
    - Results in nerve damage to hands and feet
    - Increased incidence of foot infections
  - Autonomic neuropathy
    - Damages nerves controlling voluntary and involuntary functions
    - May affect sexual function, bladder and bowel control, and BP

Acidosis in Diabetes

- Diabetes mellitus long-term complications
  - Heart disease and stroke
  - High blood glucose and blood fat levels
    - Contribute to atherosclerosis
  - Diabetics are two to four times as likely to develop heart disease as nondiabetics and are two to six times as likely to have stroke
  - Peripheral vascular disease
    - Secondary to atherosclerosis
    - Results in need for amputations

Acidosis in Diabetes

- Patients also suffer from decreased immune function as long-term complication of disease
  - Places patient at higher risk for increased morbidity and mortality from infectious disease such as influenza
  - Prone to infection-related complications from surgeries and other invasive procedures (e.g., IV therapy, bladder catheterization)
Diabetes Management

• Treatment
  – Allow patients to control their serum glucose levels
  – Help restore normal metabolism
  – Pancreatic transplants remain experimental treatment
  – Drug therapy (insulin or oral hypoglycemic agents)
  – Diet regulation
  – Exercise

Diabetes Management

• Genetically engineered human insulin is available in rapid-, intermediate-, and long-acting preparations
  – Administered by injection or nasal inhalation
  – Protein that would be digested if it were consumed orally
  – Insulin-dependent diabetic usually takes one or two doses of long-acting insulin preparation each day
  – Also takes additional amounts of a rapid-acting insulin (lasting only few hours) at meal times

Diabetes Management

• Another way for patient to self-administer insulin is with an insulin infusion pump
  – Delivers continuous “basal” level of insulin
  – Patient supplements level with bolus before ingestion of food
  – Patients calculate amount of insulin to be taken based on their carbohydrate intake
  – Glucose level must be regularly monitored to ensure adequate medication control
Diabetes Management

• Another way for patient to self-administer insulin is with an insulin infusion pump
  – Medication balance is delicate
    • Same dosage of insulin that appears correct at one time may be too much or too little at another time
    • Dosage depends on various factors (including exercise and infection)
Oral Hypoglycemic Agents

- Stimulate release of insulin from pancreas
  - Effective only in patients who have functioning beta cells that produce some insulin (type 2 diabetes)
  - Other agents help body to better utilize insulin or prevent body from manufacturing glucose
- Can have important side effects
  - Require careful patient monitoring (e.g., periodic tests for liver and kidney function)

Diabetic Emergencies

- Three life-threatening conditions may result from diabetes mellitus
  - Hypoglycemia (insulin shock)
  - Hyperglycemia (diabetic ketoacidosis)
  - Hyperosmolar hyperglycemic nonketotic syndrome (HHNS)

Hypoglycemia

- Syndrome related to blood glucose levels less than 70 mg/dL
- Symptoms usually occur at levels less than 60 mg/dL or at slightly higher blood glucose levels if fall has been rapid
Hypoglycemia

• May occur in patients who are not diabetic
  – Usually result of
    • Excessive response to glucose absorption
    • Physical exertion
    • Alcohol or drug effects
    • Pregnancy and lactation
    • Decreased dietary intake

Hypoglycemia

• In diabetics, hypoglycemic reactions usually are caused by
  – Too much insulin (or some types of oral hypoglycemic medication)
  – Decreased dietary intake (delayed or missed meal)
  – Unusual or vigorous physical activity
  – Administration of certain antibiotics (with oral hypoglycemic agents)

Hypoglycemia

• Less common causes and predisposing factors
  – Chronic alcoholism (alcohol depletes liver glycogen stores)
  – Adrenal gland dysfunction
  – Liver disease (i.e., hepatic insufficiency or failure)
  – Malnutrition
  – Pancreatic tumor
  – Cancer
### Hypoglycemia

#### Less common causes and predisposing factors
- Hypothermia
- Sepsis
- Administration of beta blockers (e.g., propranolol)
- Administration of salicylates (e.g., aspirin) in ill infants or children
- Intentional overdose with insulin, oral hypoglycemic agents, or salicylates

#### Signs and symptoms
- Usually appear quickly, often within minutes
- Related to release of epinephrine as body tries to compensate for drop in blood sugar
- In early stages, patient may complain of extreme hunger

#### Signs and symptoms due to decreased glucose availability to the brain
- Nervousness, trembling
- Irritability
- Psychotic (combative) behavior
- Weakness and incoordination
- Confusion
- Appearance of intoxication
- Weak, rapid pulse
- Cold, clammy skin
- Drowsiness
- Seizures
- Coma (in severe cases)
- Cardiac arrest
Hypoglycemia

- Should be suspected in any diabetic patient with
  - Behavioral changes
  - Confusion
  - Abnormal neurological signs
  - Unconsciousness
- True emergency
  - Requires immediate administration of glucose to prevent permanent brain damage or death

Why might this call be dispatched as a behavioral emergency (hypoglycemia)?

Diabetic Ketoacidosis

- Results from absence of or resistance to insulin
  - Low insulin level prevents glucose from entering cells
    - As result, glucose accumulates in blood
    - Cells become starved for glucose and begin to use other sources of energy (principally fat)
    - Metabolism of fat generates fatty acids and glycerol
    - Glycerol provides some energy to cells, but fatty acids are further metabolized to form ketoacids, resulting in acidosis
Diabetic Ketoacidosis

- **Acidosis**
  - Increases transport of potassium from intracellular space into intravascular space
    - Subsequent diuresis results in high potassium concentration in urine and total body potassium deficit
    - Sodium concentration in extracellular fluid usually decreases (through osmotic dilution)
    - Sodium replaced by increased hydrogen ions, adding greatly to acidosis

- **As blood sugar rises, patient undergoes massive osmotic diuresis**
  - Combined with vomiting, causes dehydration and shock
  - Although there is "overall" loss of potassium, patient is still hyperkalemic
    - Can lead to cardiac dysrhythmias and cardiac abnormalities (peaked T waves, prolonged P-R interval, widening of the QRS complex)
    - Electrolyte imbalances may also cause altered neuromuscular activity, including seizures

- **Signs and symptoms**
  - Usually related to hypovolemia and acidosis
  - Usually slow in onset (over 12 to 48 hours) including
    - Diuresis
    - Warm, dry skin
    - Dry mucous membranes
    - Tachycardia, thready pulse
    - Postural hypotension
    - Weight loss
    - Polyuria
    - Polydipsia
Diabetic Ketoacidosis

• Signs and symptoms (cont’d)
  – Polyphagia
  – Acidosis
  – Abdominal pain (usually generalized)
  – Anorexia, nausea, vomiting
  – Acetone breath odor (fruity odor)
  – Kussmaul respirations in an attempt to reduce carbon dioxide levels
  – Decreased level of consciousness

Diabetic Ketoacidosis

• Patients seldom are deeply comatose
  – Unresponsive patients should be assessed for another cause
    • Head injury
    • Stroke
    • Drug overdose

How can you distinguish Kussmaul respirations from hyperventilation?
Hyperosmolar Hyperglycemic Nonketotic Syndrome

• Acute diabetic decompensation
• Life-threatening emergency characterized by
  – Marked hyperglycemia
  – Hyperosmolarity and dehydration
  – Decreased mental functioning that may lead to coma
  – It often occurs in older patients with type 2 diabetes or in patients with undiagnosed diabetes

Hyperosmolar Hyperglycemic Nonketotic Syndrome

• Easily mistaken for DKA
  – Differs from DKA in that enough insulin may be present to prevent metabolism of fats (ketogenesis) and development of ketoacidosis
  – Amount of insulin may not be enough to prevent glucose use by peripheral tissues or to reduce gluconeogenesis by liver

Hyperosmolar Hyperglycemic Nonketotic Syndrome

• Develops from sustained hyperglycemia that produces hyperosmolar state
  – Causes osmotic diuresis that results in marked dehydration and electrolyte losses
  – Protein and fats are not used to create new supplies of glucose to same degree as in DKA
  – Ketotic cycle is either never started or does not occur until glucose is extremely elevated
  – Patients usually have blood glucose levels over 600 mg/dL
    • They have less ketone formation
    • Results in less acidemia than in patients with DKA
Hyperosmolar Hyperglycemic Nonketotic Syndrome

- Tends to develop slowly, often over several days
- High mortality rate
- Early signs and symptoms mostly related to volume depletion
  - Polyuria
  - Polydipsia

Hyperosmolar Hyperglycemic Nonketotic Syndrome

- Associated signs and symptoms
  - Orthostatic hypotension
  - Dry mucous membranes
  - Tachycardia
- CNS dysfunction may result in
  - Lethargy
  - Confusion
  - Coma
Hyperosmolar Hyperglycemic Nonketotic Syndrome

• Precipitating factors
  – Advanced age
  – Preexisting cardiac or renal disease
  – Inadequate insulin secretion or action (type 2 diabetes)
  – Increased insulin requirements
    • Stress
    • Infection
    • Trauma
    • Burns
    • Myocardial infarction

Hyperosmolar Hyperglycemic Nonketotic Syndrome

• Precipitating factors
  – Medication use
    • Thiazide and thiazide diuretics
    • Glucocorticoids
    • Phenytoin
    • Sympathomimetics
    • Propranolol
    • Immunosuppressants
  – Supplemental parenteral and enteral feedings

Diabetic Patient Assessment

• Diabetic emergency signs and symptoms
  – May mimic other, more commonly encountered conditions
  – Must have high degree of suspicion for illness related to diabetes
  – Search for medical alert information, insulin pump, insulin syringes, diabetic medications (insulin is often kept in the refrigerator)
Diabetic Patient Assessment

• Patient history important components
  – Food intake
  – Insulin or oral hypoglycemic use
  – Alcohol or other drug consumption
  – Predisposing factors (exercise, infection, illness, stress)
  – Any associated symptoms

Diabetes Management: Conscious Patient

• If conscious and able to talk, pertinent history should be obtained
  – Do this while assessing patient’s airway, breathing, and circulation
  – If appropriate, give glucose

Diabetes Management: Conscious Patient

• Protocols may include
  – Drawing blood sample for laboratory testing before glucose administered
  – Glucose testing in field is done with glucometer
    • Patients with glucose reading less than 70 mg/dL (varies by protocol) and who have signs and symptoms consistent with hypoglycemia generally should be given glucose
    • Some patients who have experienced a diabetic reaction may be treated at scene and released
    • Others may need to be transported for evaluation by a physician
Diabetes Management: Conscious Patient

• Consult with medical direction or follow established protocol
  – Before leaving scene, ensure patient who received glucose but refused transport is advised of possibility of recurrent hypoglycemia
  – Ensure patient has “meal” available that is high in complex carbohydrates and protein
    • Consumed within 30 minutes of receiving glucose

Diabetes Management: Conscious Patient

• Methods of glucose administration vary
  – If patient is alert and able to swallow, sugar should first be administered orally
  – Can be given as
    • Candy bar
    • Glass of orange juice mixed with sugar
    • Nondiet soft drink
    • Sublingual or buccal administration of a glucose gel preparation
  – Alternate method is to slowly administer 50 percent dextrose through large, stable peripheral vein (may be repeated according to protocol)
What steps should you take if the patient refuses transport (after treatment with dextrose) before you leave the scene?

Diabetes Management: Unconscious Patient

- Prehospital management should be directed at
  - Airway management
  - Administration of high-concentration oxygen
  - Ventilatory and circulatory support

- Depending on protocol, IV line of lactated Ringer’s solution or saline solution should be established for rehydration
  - Flow rate should be determined by patient’s blood pressure and heart rate
  - Before glucose is given, draw blood sample for laboratory analysis
  - If alcoholism or other drug abuse suspected, administer thiamine, naloxone, or both before glucose
Diabetes Management: Unconscious Patient

• If an IV line cannot be established, glucagon may be given by subcutaneous route, intramuscular route, or intranasally via mucosal atomization (per protocol)
  – Can help to raise serum glucose levels
  – Stimulates breakdown of liver glycogen
  – Glucagon not effective in any patient decreased liver glycogen
    • Chronic alcoholics
    • Liver disease
    • Malnourished
    • Certain diets

Diabetes Management: Unconscious Patient

• Definitive treatment for patients with DKA or HHNS requires
  – Administration of insulin
  – Fluid replacement
  – Electrolyte monitoring
  – In-hospital observation

Diabetes Management: Unconscious Patient

• While at scene and during transport, patient should be closely monitored for serious dysrhythmias
  – Rhythm disturbances can lead to cardiac arrest
    • Result from electrolyte abnormalities (hyperkalemia)
    • May require drug therapy (albuterol, calcium, sodium bicarbonate)
    • May require large volumes of IV fluid (1 to 1.5 L within first hour) for rehydration while at scene and during transport
Differential Diagnosis

• Determining cause of diabetic emergency sometimes is difficult in prehospital setting
  – When not sure of cause, all diabetic patients should receive glucose if hypoglycemia is confirmed by testing
  – Difference in signs and symptoms in diabetic emergencies should help to identify cause

Lesson 26. 3

Thyroid Gland, Cushing Syndrome and Addison Disease

Learning Objectives

• Discuss pathophysiology as a basis for key signs and symptoms, patient assessment, and patient management for disorders of the thyroid gland
• Discuss pathophysiology as a basis for key signs and symptoms, patient assessment, and management of emergencies related to Cushing syndrome and Addison disease
Thyroid Gland Disorders

• Common disorders include hyperthyroidism and hypothyroidism
  – Hyperthyroidism
    • Excess of thyroid hormones in blood
    • May result in thyrotoxicosis
  – Hypothyroidism
    • Insufficiency of thyroid hormones in blood
    • May result in myxedema

Thyrotoxicosis

• Mild form of hyperthyroidism
  – Fairly common, develops over time
• Thyroid storm
  – Life-threatening form of hyperthyroidism
  – Rare condition that may occur spontaneously
  – May be brought on by infection, stress, or a thyroidectomy
• Most cases of hyperthyroidism occur as consequence of toxic diffuse goiter (Graves’ disease)

What other medical emergencies could cause similar signs and symptoms (thyroid storm)?
Thyrotoxicosis

- Graves’ disease
  - Type of excessive thyroid activity
  - Characterized by
    - Generalized enlargement of gland (goiter)
    - Leads to swollen neck
    - Often, protruding eyes (exophthalmos)
  - Most often occurs in young women
  - May arise from autoimmune process in which antibody stimulates thyroid cells

Thyroid Gland Anatomy and Physiology

- Thyroid gland
  - Situated in front of neck just below larynx
  - Consists of two lobes
    - One on each side of trachea
    - Joined by narrower portion of tissue called isthmus
Thyroid Gland Anatomy and Physiology

- Thyroid tissue composed of two types of secretory cells
  - Follicular cells
    - Make up most of gland
    - Arranged in form of hollow, spherical follicles
    - Secrete iodine-containing hormones thyroxine (T4) and triiodothyronine (T3)
  - Parafollicular cells (C cells)
    - Occur singly or in small groups in spaces between follicles
    - Secrete hormone calcitonin, which helps regulate level of calcium in body

Thyroid Gland Anatomy and Physiology

- Thyroid hormones
  - Play key role in controlling body metabolism
  - Essential in children for normal physical growth and mental development
  - Secretion of T3 and T4 controlled by feedback system
    - Involves pituitary gland and hypothalamus
Thyroid Gland Anatomy and Physiology

- Disorders of thyroid gland
  - May result from defects in gland itself
  - May result from disruption of hypothalamic-pituitary hormonal control system
  - Advances in slow fashion
    * May have nonspecific signs and symptoms over months to years
    * May culminate in acute episode (thyroid storm)

Thyroid Gland Anatomy and Physiology

- Nonspecific signs and symptoms of thyroid hyperfunction
  - Fatigue
  - Anxiety
  - Palpitations
  - Sweating
  - Weight loss
  - Diarrhea
  - Heat intolerance

Thyroid Gland Anatomy and Physiology

- In acute episodes of thyroid storm, signs and symptoms are those related to adrenergic hyperactivity
  - Severe tachycardia
  - Heart failure
  - Cardiac dysrhythmias
  - Shock
  - Hyperthermia
  - Restlessness
  - Agitation and paranoia
  - Abdominal pain
  - Delirium
  - Coma
Hyperthyroidism Management

- Mild hyperthyroidism requires no emergency therapy
  - Best managed with physician follow-up
- Thyroid storm is true emergency, requires immediate treatment
  - Provide airway, ventilatory, and circulatory support
  - Rapid transport

Hyperthyroidism Management

- In-hospital care
  - Focuses on inhibiting hormone synthesis
  - Block hormone release and peripheral effects of thyroid hormone with antithyroid drugs
  - Provide general support of patient’s vital functions
  - Beta blockers are also given to control heart rate, tremors, anxiety

Myxedema

- Condition that results from hypothyroidism
  - Associated with inflammation or atrophy of thyroid gland
  - May be consequence of treatment for hyperthyroidism
  - Causes accumulation of mucinous material in skin
    - Results in thickening and coarsening of skin and other body tissues (most notably lips, nose, throat)
  - Most common in adults (especially women) over age 40
Myxedema

- Myxedema coma is a rare illness
  - Characterized by hypothermia and reduced level of consciousness
  - Medical emergency that may be precipitated by
    - Exposure to cold
    - Infection (usually pulmonary)
    - Congestive heart failure
    - Trauma
    - Drugs (sedatives, hypnotics, anesthetics)
    - Stroke
    - Internal hemorrhage
    - Hypoxia
    - Hypercapnia
    - Hyponatremia
    - Hypoglycemia

Myxedema Management

- Prehospital care
  - Directed at managing life-threatening conditions
    - Airway, ventilatory, and circulatory compromise
  - Provide rapid transport
    - En route, maintain body temperature, closely monitor ECG for cardiac dysrhythmias
    - Once other causes of coma have been ruled out and patient's condition has been stabilized, treatment of myxedema can begin with oral administration of thyroxine
    - Treatment must be continued for life
Adrenal Glands Disorders

- Two disorders of adrenal gland are Cushing syndrome and Addison disease
  - Cushing syndrome is caused by excessive activity of adrenal cortex
  - Addison disease is caused by inactivity of adrenal cortex

Adrenal Glands Anatomy and Physiology

- Adrenal glands are triangular-shaped endocrine glands that are located on top of both kidneys
  - Each gland consists of medulla (center of gland), which is surrounded by cortex
    - Medulla responsible for producing epinephrine and norepinephrine
    - Adrenal cortex produces other hormones necessary for fluid and electrolyte balance in body (e.g., cortisone and aldosterone)

Cushing Syndrome

- Caused by abnormally high circulating level of corticosteroid hormones
  - May be produced directly by adrenal gland tumor
    - Causes excessive secretion of corticosteroids
  - May be produced by administration of corticosteroid drugs
    - Prednisone
    - Dexamethasone
    - Methylprednisolone
Cushing Syndrome

- May be produced by enlargement of both adrenal glands as result of pituitary tumor
  - Pituitary gland controls activity of adrenal gland by producing adrenocorticotropic hormone (ACTH)
    - Stimulates cortex of adrenal gland to grow
  - Syndrome is rare
  - Mainly affects women 30 to 50 years of age

Cushing Syndrome

- Characteristic appearance
  - Face appears round ("moon face") and red
  - Trunk tends to become obese from disturbances in fat metabolism
  - Limbs become wasted from muscle atrophy
  - Acne develops, and purple stretch marks may appear on abdomen, thighs, breasts
  - Skin often thins and bruises easily
Cushing Syndrome

- Weakened bones are at increased risk of fracture
- Other features
  - Increased body and facial hair
  - Hump on back of neck ("buffalo hump")
  - Supraclavicular fat pads
  - Weight gain
  - Hypertension
  - Psychiatric disturbances (depression, paranoia)
  - Insomnia
  - Diabetes mellitus
How do you think a patient who suffers from this disease feels about his or her body image?

Cushing Syndrome Management

- Prehospital care
  - Mainly supportive
  - Disease is diagnosed through measurement of hormone levels in blood and urine, and by radiological imaging (e.g., computed tomography [CT] scan)
  - If cause of syndrome is overtreatment with corticosteroid drugs, condition usually is reversible when drug dosages are adjusted

Cushing Syndrome Management

- Prehospital care
  - If cause is tumor or overgrowth of adrenal gland, gland may require surgical removal
  - If tumor is in pituitary gland, usual treatment involves surgery, radiation, medication
  - Treatment is usually successful
  - Lifelong hormone replacement therapy is required
Addison Disease

• Rare disorder that can be life-threatening
• Caused by deficiency of corticosteroid hormones cortisol and aldosterone
  – These hormones are normally produced by adrenal cortex

Addison Disease

• Can be caused by any disease process that destroys adrenal cortices
  – Such disease processes may include
    • Adrenal hemorrhage or infarction
    • Infections (tuberculosis, fungi, viruses)
    • Autoimmune diseases
• Most common cause is shrinking of adrenal tissue
  – When this occurs, production of corticosteroid hormones is inadequate to meet body’s metabolic requirements

Addison Disease

• Signs and symptoms
  – Progressive weakness
  – Progressive weight loss
  – Progressive anorexia
  – Skin hyperpigmentation (caused by increased hormone production by the pituitary gland, which stimulates melanin)
  – Hypotension
  – Hyponatremia
  – Hyperkalemia
  – GI disturbances (nausea, vomiting, diarrhea)
Addison Disease

- Slow onset and chronic course
- Symptoms develop gradually over months to years
  - Acute episodes (Addisonian crisis) may be brought on by emotional and physiological stress

Addison Disease

- Stressors
  - Surgery
  - Alcohol intoxication
  - Hypothermia
  - MI
  - Severe illness
  - Trauma
  - Hypoglycemia
  - Infection
Addison Disease

- During these events, adrenal glands cannot increase production of corticosteroid hormones to help body cope with stress
  - Result
    - Blood glucose levels drop
    - Body loses ability to regulate content of sodium, potassium, and water in body fluids (causing dehydration and extreme muscle weakness)
    - Blood volume and BP fall
    - Body may not be able to maintain circulation efficiently

Addison Disease

- Prehospital treatment
  - Airway, ventilatory, and circulatory support
  - Closely monitor serum glucose
  - Some EMS services carry hydrocortisone (Solu Cortef) to manage adrenal insufficiency
- ECG findings as result of hypokalemia may include
  - Peaked T waves
  - Flattened P waves
  - Widening of QRS complex

Addison Disease Management

- In-hospital treatment
  - Maintain patient’s vital functions and correct sodium deficiency and dehydration
  - After life-threatening episode managed, corticosteroids
  - Patient often advised to increase dosage during times of emotional and physiological stress
Other Endocrine Disorders

• Many types of endocrine disorders and diseases
• Most endocrine disorders are related to hormone imbalance or nutritional deficiencies
• Following diagnosis, most patients will require treatment for life

Summary

• Endocrine system consists of ductless glands and tissues
  — Produce and secrete hormones
• Endocrine glands secrete their hormones directly into bloodstream
  — Exert a regulatory effect on various metabolic functions
  — All hormones operate within feedback systems (these are either positive or negative)
    • Work to maintain optimal internal environment

Summary

• Pancreatic islets are composed of beta cells, alpha cells, and other cells
  — Beta cells secrete insulin
  — Alpha cells secrete glucagon
  — Other cells are of questionable function
• Chief functions of insulin are to increase glucose transport into cells, increase glucose metabolism by cells, increase the liver glycogen level, and decrease the blood glucose concentration toward normal
**Summary**

- Glucagon has two major effects
  - (1) increase blood glucose levels by stimulating the liver to release glucose stores from glycogen and other glucose storage sites (glycogenosis) and
  - (2) stimulate gluconeogenesis through the breakdown of fats and fatty acids, thereby maintaining a normal blood glucose level

**Summary**

- Diabetes mellitus is characterized by a deficiency of insulin production or an inability of the body to respond to insulin
  - Generally classified as type 1 or type 2
    - Type 1 diabetics have inadequate insulin production
    - Treatment for type 1 consists of insulin administration, exercise, and diet regulation
    - Type 2 diabetes is caused by cellular resistance to insulin and ultimately, decreased insulin production
    - Most patients with type 2 diabetes require oral hypoglycemic medications, exercise, and dietary regulation to control illness; some require insulin administration

**Summary**

- Hypoglycemia is syndrome related to blood glucose levels below 70 mg/dL
  - Diabetic patient with behavioral changes or unconsciousness should be treated for hypoglycemia
  - This condition is a true emergency, requires immediate administration of glucose to prevent permanent brain damage or death
Summary

- Diabetic ketoacidosis results from an absence of or a resistance to insulin
  - Signs and symptoms of DKA are related to hypovolemia
  - Usually are slow in onset

Summary

- Hyperosmolar hyperglycemic nonketotic syndrome is a life-threatening emergency
  - Often occurs in older patients with type 2 diabetes
  - Frequently occurs in undiagnosed diabetics
  - Hyperglycemia produces a hyperosmolar state, causing osmotic diuresis, dehydration, and electrolyte imbalances

Summary

- Important components of patient history in assessment of diabetic patients include onset of symptoms, food intake, insulin or oral hypoglycemic use, alcohol or other drug consumption, predisposing factors, and any associated symptoms
- Any patient with a glucose reading below 70 mg/dL (varies by protocol) and signs and symptoms consistent with hypoglycemia generally should be given dextrose
Summary

• Thyrotoxicosis is any toxic condition that results from overactivity of the thyroid gland
• Thyroid storm is a life-threatening condition resulting from an overactive thyroid gland
  – Thyroid hormones play a key role in controlling body metabolism
  – Essential in children for normal physical growth and development

Summary

• Myxedema is condition that results from a thyroid hormone deficiency
  – Myxedema coma is a rare illness
  – In addition to myxedema, is characterized by hypothermia and mental obtundation
  – Is medical emergency
• Cushing syndrome is caused by an abnormally high circulating level of corticosteroid hormones.
  – Produced naturally by adrenal glands

Summary

• Addison disease is a rare but life-threatening disorder
  – Caused by a deficiency of the corticosteroid hormones cortisol and aldosterone
  • Normally produced by the adrenal cortex