Caring for Patients with Common Health Problems of the Endocrine System

Module D

Classroom Objectives

- Review the functions and hormones secreted by each of the endocrine glands
- Identify the diagnostic tests used to determine alterations in function of the endocrine glands
- Outline the teaching needs of patients requiring hormone and steroid therapy
- Discuss the relationship of the endocrine system and the nervous system as they control homeostasis
- Discuss the pharmacological and nursing implications of hormonal and steroid therapy

Objectives Continued

- Describe etiologic factors associated with diabetes
- Relate the clinical manifestations of diabetes to the associated pathophysiologic alterations
- Describe the relationship between diet, exercise, and medication for people with diabetes
- Describe management strategies for a person with diabetes to use during sick days
- Describe the major macrovascular, microvascular, and neuropathic complications of diabetes and selfcare behaviors important in their prevention

Endocrine Glands

- Controls many body functions
  - exerts control by releasing special chemical substances into the blood called hormones
  - Hormones affect other endocrine glands or body systems
- Ductless glands
- Secrete hormones directly into bloodstream
  - Hormones are quickly distributed by bloodstream throughout the body

Hormones

- Chemicals produced by endocrine glands
- Act on target organs elsewhere in body
- Control/coordinate widespread processes:
  - Homeostasis
  - Reproduction
  - Growth & Development
  - Metabolism
  - Response to stress
    - Overlaps with the Sympathetic Nervous System

Hormones

- Hormones are classified as:
  - Proteins
  - Polypeptides (amino acid derivatives)
  - Lipids (fatty acid derivatives or steroids)

Hormones

- Amount of hormone reaching target tissue directly correlates with concentration of hormone in blood.
  - Constant level hormones
    - Thyroid hormones
  - Variable level hormones
The Endocrine System
• Consists of several glands located in various parts of the body
• Specific Glands
  • Hypothalamus
  • Pituitary
  • Thyroid
  • Parathyroid
  • Adrenal
  • Kidneys
  • Pancreatic Islets
  • Ovaries
  • Testes

Pituitary Gland
• Small gland located on stalk hanging from base of brain –
  • “The Master Gland”
  • Primary function is to control other glands.
  • Produces many hormones.
  • Secretion is controlled by hypothalamus in base of brain.

Pituitary Gland
• Two areas
  • Anterior Pituitary
  • Posterior Pituitary
• Structurally, functionally different

Pituitary Gland
• Anterior Pituitary
  • Thyroid-Stimulating Hormone (TSH)
    • stimulates release of hormones from Thyroid
    –thyroxine (T4) and triiodothyronine (T3): stimulate metabolism of all cells,
    –thyroid gland produces 90% of T4 and 10% of T3.
    –calcitonin: lowers the amount of calcium in the blood by inhibiting breakdown
    of bone
    • released when stimulated by TSH or cold
    • abnormal conditions
    –hyperthyroidism: too much TSH release
    –hypothyroidism: too little TSH release

Pituitary Gland
• Anterior Pituitary
  • Growth Hormone (GH)
    • stimulates growth of all organs and increases blood glucose concentration
    –decreases glucose usage
    –increases consumption of fats as an energy source
  • Adreno-Corticotropic Hormone (ACTH)
    • stimulates the release of adrenal cortex hormones

Pituitary Gland
Anterior Pituitary
- Follicle Stimulating Hormone (FSH)
  - females - stimulates maturation of ova; release of estrogen
  - males - stimulates testes to grow; produce sperm
- Luteinizing Hormone (LH)
  - females - stimulates ovulation; growth of corpus luteum
  - males - stimulates testes to secrete testosterone

Pituitary Gland
- Anterior Pituitary
  - Prolactin
    - stimulates breast development during pregnancy; milk production after delivery
  - Melanocyte Stimulating Hormone (MSH)
    - stimulates synthesis, dispersion of melanin pigment in skin

Pituitary Gland
- Posterior Pituitary
  - Stores, releases two hormones produced in hypothalamus
    - Antidiuretic hormone (ADH)
    - Oxytocin

Pituitary Gland
- Posterior Pituitary
  - Antidiuretic hormone (ADH)
    - Stimulates water retention by kidneys
      - reabsorb sodium and water
    - Abnormal conditions
      - Undersecretion: diabetes insipidus ("water diabetes")
      - Oversecretion: Syndrome of Inappropriate Antidiuretic Hormone (SIADH)
  - Oxytocin
    - Stimulates contraction of uterus at end of pregnancy (Pitocin®); release of milk from breast

Hypothalamus
- Produces several releasing and inhibiting factors that stimulate or inhibit anterior pituitary’s secretion of hormones.
- Produces hormones that are stored in and released from posterior pituitary

Hypothalamus
- Also responsible for:
  - Regulation of water balance
  - Esophageal swallowing
  - Body temperature regulation (shivering)
  - Food/water intake (appetite)
  - Sleep-wake cycle
  - Autonomic functions

Thyroid
- Located below larynx and low in neck
  - Not over the thyroid cartilage
- Thyroxine ($T_4$) and Triiodothyronine ($T_3$)
  - Stimulate metabolism of all cells
- Calcitonin
  - Decreases blood calcium concentration by inhibiting breakdown of bone

Parathyroids
• Located on posterior surface of thyroid
• Frequently damaged during thyroid surgery
• Parathyroid hormone (PTH)
  • Stimulates Ca\(^{2+}\) release from bone
  • Promotes intestinal absorption and renal tubular reabsorption of calcium

22 Parathyroids
• Underactivity
  • Decrease serum Ca\(^{2+}\)
    • Hypocalcemic tetany
    • Seizures
    • Laryngospasm

23 Parathyroids
• Overactivity
  • Increased serum Ca\(^{2+}\)
    • Pathological fractures
    • Hypertension
    • Renal stones
    • Altered mental status
    • “Bones, stones, hypertones, abdominal moans”

24 Thymus Gland
• Located in anterior chest
• Normally absent by ~ age 4
• Promotes development of immune-system cells (T-lymphocytes)

25 Adrenal Glands
• Small glands located near (ad) the kidneys (renals)
• Consists of:
  • outer cortex
  • inner medulla

26 Adrenal Glands
• Adrenal Medulla
  • the Adrenal Medulla secretes the catecholamine hormones *norepinephrine* and *epinephrine*
  • Epinephrine and Norepinephrine
    • Prolong and intensify the sympathetic nervous system response during stress

27 Adrenal Glands
• Adrenal Cortex
  • Aldosterone (Mineralocorticoid)
    • Regulates electrolyte (potassium, sodium) and fluid homeostasis
  • Cortisol (Glucocorticoids) (most potent)
    • Antiinflammatory, anti-immunity, and anti-allergy effects.
    • Increases blood glucose concentrations
  • Androgens (Sex Hormones)
    • Stimulate sexual drive in females

28 Adrenal Glands
• Adrenal Cortex
  • Glucocorticoids
    • accounts for 95% of adrenal cortex hormone production
    • ↑ the level of glucose in the blood
    • Released in response to stress, injury, or serious infection - like the hormones
from the adrenal medulla

**Adrenal Glands**
- Adrenal Cortex
  - Mineralcorticoids
    - work to regulate the concentration of potassium and sodium in the body

**Ovaries**
- Located in the abdominal cavity adjacent to the uterus
- Under the control of LH and FSH from the anterior pituitary
- Produce eggs for reproduction
- Produce hormones
  - estrogen
  - progesterone
  - Functions include sexual development and preparation of the uterus for implantation of the egg

**Ovaries**
- Estrogen
  - Development of female secondary sexual characteristics
  - Development of endometrium
- Progesterone
  - Promotes conditions required for pregnancy
  - Stabilization of endometrium

**Testes**
- Located in the scrotum
- Controlled by anterior pituitary hormones FSH and LH
- Produce sperm for reproduction
- Produce testosterone -
  - promotes male growth and masculinization
  - promotes development and maintenance of male sexual characteristics

**Pancreas**
- Located in retroperitoneal space between duodenum and spleen
- Has both endocrine and exocrine functions
  - **Exocrine Pancreas**
    - Secretes key digestive enzymes
  - **Endocrine Pancreas**
    - Alpha Cells - glucagon production
    - Beta Cells - insulin production
    - Delta Cells - somatostatin production

**Pancreas**
- Exocrine function
  - Secretes
    - amylase
    - lipase

**Pancreas**
- **Alpha Cells**
  - Glucagon
    - Raises blood glucose levels
- **Beta Cells**
  - Insulin
Lowers blood glucose levels

**Disorders of the Endocrine System**

**Abnormal Thyroid Function**
- Hypothyroidism
  - Too little thyroid hormone
- Hyperthyroidism
  - (Thyrotoxicosis / Thyroid Storm)
  - Too much thyroid hormone

**Thyroid Function Tests**
- Serum Immunoassay
  - TSH/Sensitivity and Specificity >95%
  - Free Thyroxine 4
    - TSH=Values above 0.4 to 6.15mu/ml indicate Hypothyroidism
    - Low values indicate Hyperthyroidism

**Serum T3 and T4**
- Measurement of total T3 or T4 includes protein bound and free hormone levels that occur in response to TSH secretion

**T3 Resin Uptake Test**
- Indirect measure of unsaturated TBG
- Determines the amount of thyroid hormone bound to TBG and the number of available binding sites
- Provides an index to identify amount of thyroid hormone present in circulation

**Radioactive Iodine Uptake**
- Test measures the rate of iodine uptake by the thyroid gland
  - Tracer dose of Iodine-123

**Hypothyroidism**
- Thyroid hormone deficiency causing a decrease in the basal metabolic rate
  - Person is “slowed down”
- **Causes of Hypothyroidism:**
  - **Primary Causes**
    - Defective hormone synthesis, iodine deficiency, congenital defects or loss of thyroid tissue after treatment of hyperthyroidism
  - **Secondary Causes (Less common)**
    - Insufficient stimulation of the normal gland, causing TSH deficiency

**Hypothyroid Conditions**
- **Primary hypothyroidism**
  - Acute thyroiditis
  - Subacute thyroiditis
  - Autoimmune thyroiditis (Hashimoto disease, chronic lymphocytic thyroiditis)

**Congenital Hypothyroidism**
- Occurs in infants as a result of absent thyroid tissue, and hereditary defects in thyroid hormone synthesis
- thyroid hormone is essential for embryonic growth especially brain tissue
- Clinical manifestations of hypothyroidism may not be evident until after 4 months of age.
Continued
- Hypothyroidism is difficult to identify at birth
  - Suggestive signs include
    - High birth weight, hypothermia, delay in passing meconium, and neonatal jaundice are suggestive signs
    - Cord blood can be examined in the first days of life for T4 and TSH levels.

Clinical Manifestations
- Confusion, drowsiness, coma
- Cold intolerant
- Hypotension, Bradycardia
- Muscle weakness
- Decreased respirations
- Weight gain, Constipation
- Non-pitting peripheral edema
- Depression
- Facial edema, loss of hair
- Dry, coarse skin

Hypothyroidism
- Myxedema Coma
  - Severe hypothyroidism that can be fatal
- Management of Myxedema Coma
  - Support oxygenation, ventilation
  - IV fluids
  - Later
    - Levothyroxine (Synthroid®)

Hyperthyroidism
- Excessive levels of thyroid levels cause hypermetabolic state
  - Person is “sped up”.
- Causes of Hyperthyroidism
  - Overmedication with levothyroxine (Synthroid®) - Fad diets
  - Goiter (enlarged, hyperactive thyroid gland)
  - Graves Disease

Clinical Manifestations
- Nervousness, irritable, tremors, paranoid
- Warm, flushed skin
- Heat intolerant
- Tachycardia - High output CHF
- Hypertension
- Tachypnea
- Diarrhea
- Weight loss
- Exophthalmos
- Goiter

Hyperthyroidism
- Medical Management
  - Airway/Ventilation/Oxygen
  - ECG monitor
  - IV access - Cautious IV fluids/Acetaminophen
  - Beta-blockers/Anxiety Medications
• Tapazole
• PTU (propylthiouracil)
  ● Usually short-term use prior to more definitive treatment
  ● Radioactive Iodine Therapy

51 **Thyroid Storm/Thyrotoxicosis**
  ● Severe form of hyperthyroidism that can be fatal
  ● Acute life-threatening hyperthyroidism
  ● Cause
    ● Increased physiological stress in hyperthyroid patients

52 **Thyroid Storm/Thyrotoxicosis**
  ● Severe tachycardia
  ● Heart Failure
  ● Dysrhythmias
  ● Shock
  ● Hyperthermia
  ● Abdominal pain
  ● Restlessness, Agitation, Delirium, Coma

53 **Thyroid Storm/Thyrotoxicosis**
  ● Management
    ● Airway/Ventilation/Oxygen
    ● ECG monitor
    ● IV access - cautious IV fluids
    ● Control hyperthermia
      ● Active cooling
      ● Acetaminophen
    ● Inderal (beta blockers)
    ● Consider benzodiazepines for anxiety
    ● Propylthiouracil (PTU)

54 **Thyroid Surgery/Post-Op Care**
  ● Check dressings for bleeding
  ● Complaints of sensation of pressure or fullness on incision site can indicate bleeding
  ● Difficulty in respirations occurs in result of edema of glottis, hematoma, or injury to laryngeal nerve

55 **Hyperparathyroidism**
  ● Overproduction of parathyroid hormone
  ● Half of the patients do not have symptoms
  ● Secondary hyperparathyroidism
    ● Chronic Renal Failure

56 **Clinical Manifestations**
  ● Apathy
  ● Fatigue
  ● Muscle weakness
  ● N/V
  ● Constipation
  ● Cardiac Dysrhythmias
  ● Irritability/Neurosis

57 **Diagnostic Findings**
  ● Elevated Calcium levels
  ● Elevated Parahormone levels
  ● Radioimmunoassays sensitive
Bone Changes

Medical Management
- Hydration Therapy
- Mobility
- Diet and Medications
- Surgery

Hypoparathyroidism
- Inadequate secretion of parathyroid hormone
- Surgical removal of parathyroid gland tissue

Clinical Manifestations
- Tetany - muscle hypertonia, tremor and spasmodic or uncoordinated contractions that occur with or without efforts to make voluntary movements

Assessment/Diagnostic Findings
- Trousseau’s sign
- Chvostek’s sign
- Calcium levels lower than 5mg/dl

Medical Management
- Raise serum calcium to 9-10mg/dl
- Calcium gluconate
- Pentobarbital
- Parenteral Parahormone
- Tracheostomy/Mechanical Vent.

Abnormal Adrenal Function
- Hyperadrenalism
  - Excess activity of the adrenal gland
  - Cushing’s Syndrome & Disease
  - Pheochromocytoma
- Hypoadrenalism (adrenal insufficiency)
  - Inadequate activity of the adrenal gland
  - Addison’s disease

Hyperadrenalism
- Primary Aldosteronism
  - Excessive secretion of aldosterone by adrenal cortex
    - Increased Na+/H2O
  - Presentation
    - headache
    - nocturia, polyuria
    - fatigue
    - hypertension, hypervolemia
    - potassium depletion

Hyperadrenalism
- Adrenogenital syndrome
  - “Bearded Lady”
  - Group of disorders caused by adrenocortical hyperplasia or malignant tumors
  - Excessive secretion of adrenocortical steroids especially those with androgenic or
estrogenic effects
• Characterized by
  ● masculinization of women
  ● feminization of men
  ● premature sexual development of children

66 Hyperadrenalism
● Cushing’s Syndrome
• Results from increased adrenocortical secretion of cortisol
• Causes include:
  ● ACTH-secreting tumor of the pituitary (Cushing’s disease)
  ● excess secretion of ACTH by a neoplasm within the adrenal cortex
  ● excess secretion of ACTH by a malignant growth outside the adrenal gland
  ● excessive or prolonged administration of steroids

67 Hyperadrenalism
● Cushing’s Syndrome
• Characterized by:
  ● truncal obesity
  ● moon face
  ● buffalo hump
  ● acne, hirsutism
  ● abdominal striae
  ● hypertension
  ● psychiatric disturbances
  ● osteoporosis
  ● amenorrhea

68 Hyperadrenalism
● Cushing’s Disease
• Too much adrenal hormone production
  ● adrenal hyperplasia caused by an ACTH secreting adenoma of the pituitary
• “Cushingoid features”
  ● striae on extremities or abdomen
  ● moon face
  ● buffalo hump
  ● weight gain with truncal obesity
  ● personality changes, irritable

69 Hyperadrenalism
● Cushing’s Syndrome
• Management
  ● Surgery/Radiation if indicated
  ● Supportive care
  ● Assess for cardiovascular event requiring treatment
    – severe hypertension
    – myocardial ischemia

70 Hyperadrenalism
● Pheochromocytoma
• Catecholamine secreting tumor of adrenal medulla
• Presentation
  ● Anxiety
  ● Pallor, diaphoresis
  ● Hypertension – BP 250/150
• Tachycardia, Palpitations
• Dyspnea
• Hyperglycemia

**Hyperadrenalism**
• Pheochromocytoma
  • Management
    • Measurements of urine and plasma catecholamines
    • Calm/Reassure
    • Assess blood glucose
    • Consider beta blocking agent - Labetalol
    • Consider benzodiazepines

**Hypoadrenalism**
• Adrenal Insufficiency
  • Decrease production of glucocorticoids, mineralcorticoids and androgens
• Causes
  • Primary adrenal failure (Addison’s Disease)
  • Infection (TB, fungal, Meningococcal)
  • Autoimmune destruction
  • AIDS
  • Prolonged steroid use

**Hypoadrenalism**
• Addison’s
  • Hypotension, Shock (Addison’s Crisis)
  • Hyponatremia, Hyperkalemia
  • Progressive Muscle weakness
  • Progressive weight loss and anorexia
  • Skin hyperpigmentation
    • Areas exposed to sun, pressure points, joints and creases
  • Arrhythmias
  • Hypoglycemia
  • N/V/D

**Hypoadrenalism**
• Immediate TX is directed toward shock
  • VS/ECG monitor
  • IV fluids
  • Assess blood glucose - D50 if hypoglycemic
  • Steroids
    • Hydrocortisone or dexamethasone
    • Florinef (mineralcorticoid)
  • Vasopressors if unresponsive to IV fluids

**Diabetes Mellitus**
• Chronic metabolic disease
• One of the most common diseases in North America
  • Affects 5% of USA population (12 million people)
• Results in
  • ↓ insulin secretion by the Beta (β) cells of the islets of Langerhans in the pancreas, AND/OR
  • Defects in insulin receptors on cell membranes leading to cellular resistance to insulin
Leads to an ↑ risk for significant cardiovascular, renal and ophthalmic disease

**Regulation of Glucose**

**Dietary Intake**
- **Components of food**:
  - Carbohydrates
  - Fats
  - Proteins
  - Vitamins
  - Minerals

**Regulation of Glucose**
- The other 3 major food sources for glucose are
  - carbohydrates
  - proteins
  - fats
- Most sugars in the human diet are complex and must be broken down into simple sugars: glucose, galactose and fructose - before use

**Regulation of Glucose**
- **Carbohydrates**
  - Found in sugary, starchy foods
  - Ready source of near-instant energy
  - If not “burned” immediately by body, stored in liver and skeletal muscle as glycogen (short-term energy) or as fat (long-term energy needs)
  - After normal meal, approximately 60% of the glucose is stored in liver as glycogen

**Regulation of Glucose**
- **Fats**
  - Broken down into fatty acids and glycerol by enzymes
  - Excess fat stored in liver or in fat cells (under the skin)

**Regulation of Glucose**
- Pancreatic hormones are required to regulate blood glucose level
  - **glucagon** released by Alpha (α) cells
  - **insulin** released by Beta Cells (β)
  - **somatostatin** released by Delta Cells (δ)

**Regulation of Glucose**
- Alpha (α) cells release glucagon to control blood glucose level
  - When blood glucose levels fall, α cells ↑ the amount of glucagon in the blood
  - The surge of glucagon stimulates liver to release glucose stores by the breakdown of glycogen into glucose (glycogenolysis)
  - Also, glucagon stimulates the liver to produce glucose (gluconeogenesis)

**Regulation of Glucose**
- Beta Cells (β) release insulin (antagonistic to glucagon) to control blood glucose level
  - Insulin ↑ the rate at which various body cells take up glucose ⇒ insulin lowers the blood glucose level
  - Promotes glycogenesis - storage of glycogen in the liver
  - Insulin is rapidly broken down by the liver and must be secreted constantly

**Regulation of Glucose**
- Delta Cells (δ) produce somatostatin, which inhibits both glucagon and insulin
  - inhibits insulin and glucagon secretion by the pancreas
  - inhibits digestion by inhibiting secretion of digestive enzymes
  - inhibits gastric motility
  - inhibits absorption of glucose in the intestine
Regulation of Glucose
- Breakdown of sugars carried out by enzymes in the GI system
  - As simple sugars, they are absorbed from the GI system into the body
- To be converted into energy, glucose must first be transmitted through the cell membrane
  - Glucose molecule is too large and does not readily diffuse

Regulation of Glucose
- Glucose must pass into the cell by binding to a special carrier protein on the cell’s surface.
  - Facilitated diffusion - carrier protein binds with the glucose and carries it into the cell.
- The rate at which glucose can enter the cell is dependent upon insulin levels
  - Insulin serves as the messenger - travels via blood to target tissues
  - Combines with specific insulin receptors on the surface of the cell membrane

Regulation of Glucose
- Body strives to maintain blood glucose between 60 mg/dl and 120 mg/dl.
- Glucose
  - brain is the biggest user of glucose in the body
  - sole energy source for brain
  - brain does not require insulin to utilize glucose

Regulation of Glucose
- Glucagon
  - Released in response to:
    - Sympathetic stimulation
    - Decreasing blood glucose concentration
  - Acts primarily on liver to increase rate of glycogen breakdown
  - Increasing blood glucose levels have inhibitory effect on glucagon secretion

Regulation of Glucose
- Insulin
  - Released in response to:
    - Increasing blood glucose concentration
    - Parasympathetic innervation
  - Acts on cell membranes to increase glucose uptake from blood stream
  - Promotes facilitated diffusion of glucose into cells

Diabetes Mellitus
- 2 Types historically based on age of onset (NOT insulin vs. non-insulin)
  - Type I
    - juvenile onset
    - insulin dependent
  - Type II
    - historically adult onset
    - now some morbidly obese children are developing Type II diabetes
    - non-insulin dependent
    - may progress to insulin dependency

Types of Diabetes Mellitus
- Type I
- Type II
- Secondary
- Gestational
Pathophysiology of Type I Diabetes Mellitus
- Characterized by inadequate or absent production of insulin by pancreas
- Usually presents by age 25
- Strong genetic component
- Autoimmune features
  - body destroys own insulin-producing cells in pancreas
  - may follow severe viral illness or injury
- Requires lifelong treatment with insulin replacement

Pathophysiology of Type II Diabetes Mellitus
- Pancreas continues to produce some insulin however disease results from combination of:
  - Relative insulin deficiency
  - Decreased sensitivity of insulin receptors
- Onset usually after age 25 in overweight adults
  - Some morbidly obese children develop Type II diabetes
- Familial component
- Usually controlled with diet, weight loss, oral hypoglycemic agents
  - Insulin may be needed at some point in life

Secondary Diabetes Mellitus
- Pre-existing condition affects pancreas
  - Pancreatitis
  - Trauma

Gestational Diabetes Mellitus
- Occurs during pregnancy
  - Usually resolves after delivery
- Occurs rarely in non-pregnant women on BCPs
- Increased estrogen, progesterone antagonize insulin

Presentation of New Onset Diabetes Mellitus
- 3 Ps
  - Polyuria
  - Polydipsia
  - Polyphagia
- Blurred vision, dizziness, altered mental status
- Rapid weight loss
- Warm dry skin,
- Weakness, Tachycardia, Dehydration

Subject Data
- Onset and duration
- Presence of polytriad
- Associated symptoms
- Past medical history
- Family History

Objective Data
- Physical Examination
- Laboratory Data

Long Term Treatment of Diabetes Mellitus
• Diet regulation
  • *e.g.* 1400 calorie ADA diet
• Exercise
  • increase patient’s glucose metabolism
• Oral hypoglycemic agents
  • Sulfonylureas
• Insulin
  • Historically produced from pigs (porcine insulin)
  • Currently genetic engineering has lead to human insulin (Humulin)

### Long Term Treatment of Diabetes Mellitus

**Insulin**

- Available in various forms distinguished on onset and duration of action
  - **Onset**
    - rapid (Regular, Semilente, Novolin 70/30)
    - intermediate (Novolin N, Lente)
    - slow (Ultralente)
  - **Duration**
    - short, 5-7 hrs (Regular)
    - intermediate, 18-24 hrs (Semilente, Novolin N, Lente, NPH)
    - long-acting, 24 - 36+ hrs (Novolin 70/30, Ultralente)

- Must be given by injection as insulin is protein which would be digested if given orally
  - extremely compliant patients may use an insulin pump which provides a continuous dose
  - current research studying inhaled insulin form

### Emergencies Associated Blood Glucose Level

**Hyperglycemia**

- Diabetic Ketoacidosis (DKA)
- Hyperglycemic Hyperosmolar Nonketotic Coma (HHNC)

**Hypoglycemia**

- “Insulin Shock”

### Hyperglycemia

- Defined as blood glucose > 200 mg/dl
- Causes
  - Failure to take *medication* (insulin)
  - Increased *dietary* intake
  - *Stress* (surgery, MI, CVA, trauma)
• Fever
• Infection
• Pregnancy (gestational diabetes)

Hyperglycemia

- Two hyperglycemic diabetic states may occur
  - Diabetic Ketoacidosis (DKA)
  - Hyperglycemic Hyperosmolar Non-ketotic Coma (HHNC)

Diabetic Ketoacidosis (DKA)

- Occurs in Type I diabetics (insulin dependency)
- Usually associated with blood glucose level in the range of 200 - 600 mg/dl
- No insulin availability results in ketoacidosis

Diabetic Ketoacidosis (DKA)

- Pathophysiology
  - Results from absence of insulin
    - Prevents glucose from entering the cells
    - Leads to glucose accumulation in the blood
  - Cells become starved for glucose and begin to use other energy sources (primarily fats)
    - Fat metabolism generates fatty acids
    - Further metabolized into ketoacids (ketone bodies)

Diabetic Ketoacidosis (DKA)

- Pathophysiology (cont)
  - Blood sugar rises above renal threshold for reabsorption (blood glucose > 180 mg/dl)
    - Glucose "spills" into the urine
    - Loss of glucose in urine causes osmotic diuresis
  - Results in
    - Dehydration
    - Acidosis
    - Electrolyte imbalances (especially K+)

Diabetic Ketoacidosis (DKA)

- Presentation
  - Gradual onset with progression
  - Warm, pink, dry skin
  - Dry mucous membranes (dehydrated)
  - Tachycardia, weak peripheral pulses
  - Weight loss
  - Polyuria, polydipsia
  - Abdominal pain with nausea/vomiting
  - Altered mental status
  - Kussmaul respirations with acetone (fruity) odor

Diabetic Ketoacidosis

Management of DKA

- Airway/Ventilation/Oxygen NRB mask
- Assess blood glucose level & ECG
- IV access, large bore NS
  - Normal saline bolus and reassess
  - Often requires several liters
- Assess for underlying cause of DKA
- Transport
Hyperosmolar Hyperglycemic Nonketotic Coma (HHNC)

- Usually occurs in type II diabetics
- Typically very high blood sugar (>600mg/dl)
- Some insulin available
- Higher mortality than DKA

Pathophysiology

- Some minimal insulin production
  - enough insulin available to allow glucose to enter the cells and prevent ketogenesis
  - not enough to decrease gluconeogenesis by liver
  - no ketosis
- Extreme hyperglycemia produces hyperosmolar state causing diuresis
- Severe dehydration
- Electrolyte disturbances

Presentation

- Same as DKA but with greater severity
  - Higher blood glucose level
  - Non-insulin dependent diabetes
  - Greater degree of dehydration

Management of HHNC

- Secure airway and assess ventilation
  - Consider need to assist ventilation
  - Consider need to intubate
- High concentration oxygen
- Assess blood glucose level & ECG
- IV access, large bore NS
  - Normal saline bolus and reassess
  - Often requires several liters
- Assess for underlying cause of HHNC
- Transport

Further Management of Hyperglycemia

- Insulin (regular)
- Correct hyperglycemia
- Correction of acid/base imbalances
  - Bicarbonate (severe cases documented by ABG)
- Normalization of electrolyte balance
- DKA may result in hyperkalemia \(2^\circ\) to acidosis
  - \(H^+\) shifts intracellularly, \(K^+\) moves to extracellular space
  - Urinary \(K^+\) losses may lead to hypokalemia once therapy is started

Hypoglycemia

- True hypoglycemia defined as blood sugar < 60 mg/dl
- All hypoglycemia is NOT caused by diabetes
- Can occur in non-diabetic patients
  - Thin young females
  - Alcoholics with liver disease
alcohol consumption on empty stomach will block glucose synthesis in liver (gluconeogenesis)

Hypoglycemia causes impaired functioning of brain which relies on constant supply of glucose

Hypoglycemia

Causes of hypoglycemia in diabetics
- Too much insulin
- Too much oral hypoglycemic agent
  - Long half-life requires hospitalization
- Decreased dietary intake (took insulin and missed meal)
- Vigorous physical activity

Pathophysiology
- Inadequate blood glucose available to brain and other cells resulting from one of the above causes

Presentation
- Hunger (initially), Headache
- Weakness, Incoordination (*mimics a stroke*)
- Confusion, Unusual behavior
  - may appear intoxicated
- Seizures
- Coma
- Weak, rapid pulse
- Cold, clammy skin
- Nervousness, trembling, irritability

Hypoglycemia: Pathophysiology

Management of Hypoglycemia

Secure airway manually
- suction prn
- Ventilate prn

High concentration oxygen

Vascular access
- Large bore IV catheter
- Saline lock, D₅W or NS
- Large proximal vein preferred

Assess blood glucose level

Oral glucose
- ONLY if intact gag reflex, awake & able to sit up
- 15gm-30gm of packaged glucose, or
- May use sugar-containing drink or food
- Oral route often slower

Intravenous glucose
- Adult: Dextrose 50% (D₅₀) 25gms IV in patent, free-flowing vein, may repeat
- Children: Dextrose 25% (D₂₅) @ 2 - 4 cc/kg (0.5 - 1 gm/kg) [Infants - may choose Dextrose 10% @ 0.5 - 1 gm/kg or 5 - 10 cc/kg]

Glucagon
- Used if unable to obtain IV access
• 1 mg IM
• Requires glycogen stores
• Slower onset of action than IV route

Management of Hypoglycemia
• Have patient eat high-carbohydrate meal
• Transport?
  • Patient Refusal Policy
    • Contact medical control
    • Leave only with responsible family/friend for 6 hours
    • Must educate family/friend to hypoglycemic signs/symptoms
    • Advise to contact personal physician
• Transport
  • Hypoglycemic patients on oral agents (long half life)
  • Unknown, atypical or untreated cause of hypoglycemia

Long-term Complications of Diabetes Mellitus
• Blindness
  • Retinal hemorrhages
• Renal Disease
• Peripheral Neuropathy
  • Numbness in “stocking glove” distribution (hands and feet)
• Heart Disease and Stroke
  • Chronic state of Hyperglycemia leads to early atherosclerosis

Complications in Pregnancy

Long-term Complications of Diabetes Mellitus
• Diffuse Atherosclerosis
  • AMI
  • CVA
  • PVD
    • Hypertension
    • Renal failure
    • Diabetic retinopathy/blindness
    • Gangrene

Long-term Complications of Diabetes Mellitus
• Peripheral Neuropathy
  • Silent MI
    • Vague, poorly-defined symptom complex
      – Weakness
      – Dizziness
      – Malaise
      – Confusion
    • Suspect MI in any diabetic with MI signs/symptoms with or without CP

Diabetes in Pregnancy
• Early pregnancy (<24 weeks)
  • Rapid embryo growth
  • Decrease in maternal blood glucose
  • Episodes of hypoglycemia

Diabetes in Pregnancy
• Late pregnancy (>24 weeks)
  • Increased resistance to insulin effects
Diabetes in Pregnancy

- Increased maternal risk for:
  - Pregnancy-induced hypertension
  - Infections
    - Vaginal
    - Urinary tract

- Increased fetal risk for:
  - High birth weight
  - Hypoglycemia
  - Liver dysfunction-hyperbilirubinemia
  - Hypocalcemia

Assessment of the Diabetic Patient

- Maintain high-degree of suspicion
- Assess blood glucose level in all patients with
  - seizure, neurologic S/S, altered mental status
  - vague history or chief complaint
- Blood glucose assessment IS NOT necessary in all patients with diabetes mellitus!!

Assessment of the Diabetic Patient

- History and Physical Exam includes
  - Look for insulin syringes, medical alert tag, glucometer, or insulin (usually kept in refrigerator)
  - Last meal and last insulin dose
  - Missed med or missed meal?
  - Signs of infection
    - Foot cellulitis / ulcers
    - Recent illness or physiologic stressors

Blood Glucose Assessment

- Capillary vs. venous blood sample
  - Depends on glucometer model
  - Usually capillary preferred
- Dextrostick vs Glucometer
  - Dextrostick - colorimetric assessment of blood provides glucose estimate
  - Glucometer - quantitative glucose measurement
- Neonatal blood
  - Many glucometers are not accurate for neonates