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 pathophysiological 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 self-care 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**
    - Epinephrine (adrenaline) release
  - **Cyclic level hormones**
    - Reproductive hormones
    - Diurnal/Happening during the day

**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-Corticotrophic Hormone (ACTH)
    - stimulates the release of adrenal cortex hormones

- FSH
  - stimulates maturation of ova; release of estrogen
  - stimulates testes to grow; produce sperm

- LH
  - females - stimulates ovulation; growth of corpus luteum
  - males - stimulates testes to secrete testosterone

- 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

- ADH
  - Stimulates water retention by kidneys; reabsorb sodium and water
  - Abnormal conditions
    - Undersecretion: diabetes insipidus (“water diabetes”)
    - Oversecretion: Syndrome of Inappropriate Antiuretic 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

**Parathyrionds**
- 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

**Parathyroids**
- Underactivity
  - Decrease serum $Ca^{2+}$
    - Hypocalcemic tetany
    - Seizures
    - Laryngospasm

**Parathyroids**
- Overactivity
  - Increased serum $Ca^{2+}$
    - Pathological fractures
    - Hypertension
    - Renal stones
    - Altered mental status
  - “Bones, stones, hypertones, abdominal moans”

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

- Small glands located near (ad) the kidneys (renals)
- Consists of:
  - outer cortex
  - inner medulla

- 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

- 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

- 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

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 6.15 µg/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

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

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

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)

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

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

**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

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
Hyperadrenalism

- **Cushing’s Syndrome**
  - Characterized by:
    - truncal obesity
    - moon face
    - buffalo hump
    - acne, hirsutism
    - abdominal striae
    - hypertension
    - psychiatric disturbances
    - osteoporosis
    - amenorrhea

- **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

- **Management**
  - Surgery/Radiation if indicated
  - Supportive care
  - Assess for cardiovascular event requiring treatment
    - severe hypertension
    - myocardial ischemia

Hyperadrenalism

- **Pheochromocytoma**
  - Catecholamine secreting tumor of adrenal medulla
  - Presentation
    - Anxiety
    - Pallor, diaphoresis
    - Hypertension – BP 250/150
    - Tachycardia, Palpitations
    - Dyspnea
    - Hyperglycemia

- **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
- 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
- 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

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

- 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)

- 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)

- 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

- 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)

Long Term Treatment of Diabetes Mellitus

- Insulin
  - 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
Long Term Treatment of Diabetes Mellitus

- Oral Hypoglycemic Agents
  - Stimulate the release of insulin from the pancreas, thus patient must still have intact beta cells in the pancreas.
  - Common agents include:
    - Glucotrol® (glipizide)
    - Micronase® or Diabeta® (glyburide)
    - Glucophage® (metformin) [Not a sulfonylurea]

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)

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+)  

- **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**

- Inadequate insulin
- Increased Blood Sugar
- Cells Can’t Burn Glucose
- Polyphagia
- Ketone Bodies
- Fruity Breath
- Osmotic Diuresis
- Polyuria
- Polydipsia
- Metabolic Acidosis
- Kussmaul Breathing

**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 (>600 mg/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

**Hyperosmolar Hyperglycemic Nonketotic Coma (HHNC)**

- 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
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Hyperosmolar Hyperglycemic Nonketotic Coma (HHNC)

- Inadequate insulin
- Increased Blood Sugar
- Osmotic Diuresis
- Polyuria
- Volume Depletion
- Shock
- Polydipsia

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+ 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

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
Hypoglycemia

- **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

- Blood Glucose Falls
- Brain Lacks Glucose
- SNS Response
  - Anxiety
  - Pallor
  - Tachycardia
  - Diaphoresis
  - Nausea
  - Dilated Pupils
  - Altered LOC
  - Seizures
  - Headache
  - Dizziness
  - Bizarre Behavior
  - Weakness
  - Anxiety
  - Pallor
  - Tachycardia
  - Diaphoresis
  - Nausea
  - Dilated Pupils

Management of Hypoglycemia

- Secure airway manually
  - suction prn
  - Ventilate prn
- High concentration oxygen
- Vascular access
  - Large bore IV catheter
  - Saline lock, D$_{50}$W or NS
  - Large proximal vein preferred
- Assess blood glucose level

Management of Hypoglycemia

- 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% (D$_{50}$) 25gms IV in patent, free-flowing vein, may repeat
  - Children: Dextrose 25% (D$_{25}$) @ 2 - 4 cc/kg (0.5 - 1 gm/kg) [Infants - may choose Dextrose 10% @ 0.5 - 1 gm/kg or 5 - 10 cc/kg]

Beta Blockers may mask symptoms by inhibiting sympathetic response

Management of Hypoglycemia

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

What persons are likely to have inadequate glycogen stores?
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
  - Diabetic retinopathy/blindness
  - Gangrene

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

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
  - Increased blood glucose
  - Ketoacidosis

- 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

- 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

- 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

Blood Glucose Assessment

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!!