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ENDOCRINOLOGY

Advanced Care Paramedicine

Module: 05

Section: 05

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Lecture Outline

- Introduction
- Disorders of the pancreas
- Disorders of the thyroid gland
- Disorders of the adrenal glands

Endocrine System

- Closely linked to the nervous system
- Exerts control over the body through hormones
  - Specialized chemical messengers
- Endocrine glands
  - Eight major glands
  - Secrete hormones directly into circulation

Endocrine Glands

- Tend to have widespread effects
- Hormones act on distant tissues
  - Specific effect on specific target tissues
  - Some have many targets, some have one
- Important role in the regulation of body functions

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Disorders of the Pancreas

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Diabetes

Statistics

- 1.2-1.4 million Canadians affected
- Same number probably living undiagnosed
- Diabetes is the 10th leading cause of death
  - Type I Diabetes accounts for most diabetes related deaths
  - Heart disease is 2 – 4 X's more common
  - Leading cause of adult blindness (approximately 5000 per year)
  - Account for 28% of all new cases of serious kidney disease
  - Half or more of all non-traumatic limb amputations are due to diabetes

Diabetes Mellitus

- Marked by inadequate insulin activity in the body
  - Diabetes: to siphon
  - Mellitus: honey sweet
- A result of:
  - No insulin production
  - Inadequate insulin production
  - Diminished tissue sensitivity to insulin

Insulin

- Secreted by beta cells in response to high blood sugar
- Cellular effects
  - ↑ cellular uptake
- Hepatic effects
  - ↑ production of glycogen, protein, fat
- No effect on brain cells

Insulin

- Insulin effectiveness
  - Sufficient insulin in circulation
  - Must be able to bind to cells to facilitate adequate stimulation
- Basis for understanding the types of diabetes

Alternative Sources of Energy

- Occasionally the body cannot use glucose as a primary energy source
- Fat metabolism
  - Adipose broken down into fatty acids
  - Increased ketone bodies
  - Contribute to increased metabolic acids

Table 30-2 Summary of Glucose Metabolism

Hormonal Effects of Insulin and Glucagon

Insulin	Glucagon
Dominant hormone when blood glucose level is high	Dominant hormone when blood glucose level is low
Major Effects on Target Tissues	***Major Effects on Target Tissues***
all cells: ↑ uptake glucose	
liver: ↑ production of glycogen, protein, fat	liver: ↑ glycogenolysis → glucose
liver, fat: ↑ production of fats	liver: ↑ gluconeogenesis (protein, fat → glucose)

Regulation of Blood Glucose

- Hypoglycemia
  - Low blood sugar (<4.0 mmol/L)
  - Stimulates α cells
  - Release glucagon
- Hyperglycemia
  - High blood sugar
  - Stimulates β cells
  - Release insulin
  - Also lost in urine (osmotic diuresis)

Classifications

- Type 1 (IDDM)
  - Juvenile onset diabetes (10% of cases)
- Type 2 (NIDDM)
  - Maturity onset diabetes/obesity onset diabetes (90% of cases)
- Type 3 (Gestational Diabetes)
  - 2 – 4 % of all pregnancies
- “Other specific types”
  - Maturity Onset Diabetes in Youth (MODY)
  - Steroid diabetes

Type 1 (IDDM)

- A multisystem disease with both biochemical and anatomical consequences
- It is a chronic disease of carbohydrate, fat, and protein metabolism caused by the lack of insulin
- Insulin is functionally absent because of the destruction of the beta cells of the pancreas
- Occurs most commonly in juveniles
  - but can occur in adults, especially in those in their late 30s and early 40s

Type 1 (IDDM)

- Catabolic disorder in which circulating
  - insulin is very low or absent
  - plasma glucagon is elevated
  - pancreatic beta cells fail to respond to all insulin-secretory stimuli
- Patients need exogenous insulin
  - To reverse this catabolic condition, prevent ketosis, decrease hyperglucagonemia, and normalize lipid and protein metabolism
- An autoimmune disease
  - Lymphocytic infiltration and destruction of insulin-secreting cells of the islets of Langerhans, causing insulin deficiency
  - Approximately 85% of patients have circulating islet cell antibodies, and the majority also have detectable anti-insulin antibodies before receiving insulin therapy

Type 1 (IDDM)

- Race
  - More common among non-Hispanic whites, followed by African Americans and Hispanic Americans
  - Uncommon among Asians.
- Sex
  - More common in men than in women
- Age
  - Usually starts in children aged 4 years or older, with the peak incidence of onset at 11-13 years of age, coinciding with early adolescence and puberty
  - Also, a relatively high incidence exists in people in their late 30s and early 40s, when it tends to present in a less aggressive manner

Type 1 (IDDM)

- Results in pronounced hyperglycemia.
  - Polydipsia
  - Polyuria
  - Polyphagia
  - Weight loss
  - Weakness

Type 2 (NIDDM)

- Characterized by:
  - Decrease in insulin production
  - Diminished tissue sensitivity to insulin
- Results in less-pronounced hyperglycemia
  - Less risk of fat-based metabolism.
  - Managed with dietary and lifestyle changes with oral drugs to stimulate insulin production and increase receptor effectiveness.
  - Patients are not absolutely dependent upon insulin for life, even though many of these patients ultimately are treated with insulin

Type 2 (NIDDM)

- Most often occurs in adults (> 40 or overweight)
  - Accounts for 90% of all diagnosed diabetes patients
- Presumably related to multiple genes
  - Inherited components for both pancreatic beta cell failure and insulin resistance
- Most patients have both insulin resistance and some degree of insulin deficiency
- Obesity
  - predisposes a person because larger quantities of insulin required for metabolic control in obese individual then in those with normal weight

Type 3 (Gestational)

- GDM is defined as glucose intolerance, but with its first onset during pregnancy
- Approximately 3.5% of non-Aboriginal women, and up to 18% of Aboriginal women will develop GDM
- Risk factors for developing this condition include:
  - Previous diagnosis of GDM
  - Age over 35 years
  - Obesity
  - History of polycystic ovary syndrome
  - Hirsutism (excessive body and facial hair)
  - Acanthosis nigricans (a skin disorder characterized by the appearance of darkened patches of skin)
  - Member of a population considered to be at high risk for diabetes

Type 3 (Gestational)

- Caused by hormones produced by the placenta that are produced for the baby, that in turn block insulin's action in the mother
- All women should be screened for GDM between 24 and 28 weeks' gestation using a glucose tolerance test
- Prompt diagnosis is important, as it carries several risks to both mother and infant
- Children born to mothers with GDM may be "macrosomic"

Effects of DM

- Can be attributed to one of the following effects of decreased insulin:
  - Decreased use of glucose
  - Marked increase in mobilization of fats from fat storage areas, causing abnormally high fat metabolism
    - With the body burning fat to try to obtain energy, ketoacidosis can occur
    - Long term will result in severe atherosclerosis
  - Depletion of proteins in body tissue and muscle wasting

Diabetic Emergencies

- Hypoglycemia
- Hyperglycemia
- Diabetic ketoacidosis (DKA)
- Hyperosmolar Hyperglycemic Nonketonic (HHNK) Coma

Hypoglycemia

- A syndrome characterized by a reduction in plasma glucose concentration to a level that may induce symptoms of low blood sugar
- Hypoglycemia typically arises from abnormalities in the mechanisms involved in glucose homeostasis
- To diagnose hypoglycemia, The Whipple triad is characteristic
  - Documentation of low blood sugar
  - Presence of symptoms
  - Reversal of these symptoms when the blood sugar level is restored to normal

Hypoglycemia

- Low blood glucose level
  - Below 4.0 mmol/L for EHSNS
  - Below 3.6 mmol/L for most hospitals
- Onset – rapid
- Can quickly become a medical emergency requiring prehospital/hospital assistance
- By the time signs and symptoms develop, most of the body's stores have been used

Causes

- Too much insulin
- Excessive use of oral hypoglycemic medications
- Decreased dietary intake
- Increased activity
- Drug Use
  - Ethanol, haloperidol, pentamidine, quinine, salicylates, and sulfonamides

- Less common
  - Chronic Alcoholism (Depletion of glycogen stores)
  - Adrenal gland dysfunction
  - Liver disease
  - Malnutrition
  - Tumors
  - Hypothermia
  - Sepsis
  - Administration of Beta Blockers
  - OD (insulin, hypoglycemics or salicylates)

Pathophysiology

- Symptoms are related to the brain and the sympathetic nervous system
- Decreased levels of glucose lead to:
  - Deficient cerebral glucose availability (neuroglycopenia)
  - Stimulation of the sympatho-adrenal nervous system
  - Adrenergic symptoms often precede the neuroglycopenic symptoms
  - Studies show that the primary stimulus for catecholamine release is the absolute level of plasma glucose rather then the rate of decrease of glucose

Symptoms

- Neurogenic (adrenergic)
  - Cold, clammy skin
  - Shakiness
  - Tachycardia (Weak)
  - Anxiety
  - Sensation of hunger
- Neuroglycopenic
  - Weakness, tiredness, dizziness
  - Psychotic behavior
  - Irritability
  - Inappropriate behavior (sometimes mistaken for inebriation)
  - Confusion
  - Blurred vision
  - Seizures
  - Coma and death

Hypoglycemia

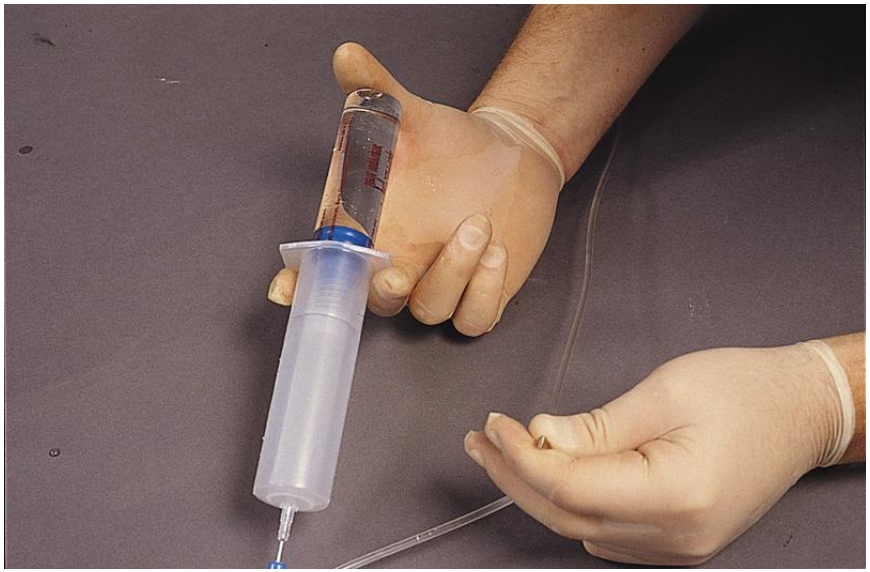
- Must remember
  - Hypoglycemia should be suspected in any diabetic patient with behavioral changes, confusion, abnormal signs or unconsciousness
- This condition is a true emergency that requires the administration of immediate glucose to prevent permanent brain damage or death

Hypoglycemia

- Some people with diabetes, especially if they have had diabetes for a long period of time, can become sensitized to the feelings of a low blood glucose level
- Since they do not feel that their glucose level is falling, they will not know to treat with juice etc.
- This situation often results in the need for assistance.

Hypoglycemia

- Treatments
  - Oral Glucose
  - Glucagon
  - IV Glucose
  - Oxygen
  - Thiamine



Severity	Blood Glucose	Symptoms
Mild	Below 4.0 mmol/L	Sweating, trembling, hunger, nausea, tingling of lips, anxiety, and heart palpitations
Moderate	Below 4.0 mmol/L	Headache, dizziness, difficulty concentrating and speaking, confusion, drowsy, tired
Severe	Below 2.8 mmol/L	Difficult or unable to wake up (coma), seizures

Hyperglycemia

- Higher then normal blood glucose level
- Over 10 mmol/L
  - Extreme prolonged hyperglycemia will cause DKA
- Usually a slow onset

Hyperglycemia

- Produced by lack of endogenous insulin
  - Absolute Type 1 DM
  - Relative Type 2 DM
    - Relative usually occurs because of resistance to the actions of insulin in muscle, fat, and the liver and an inadequate response by the pancreatic beta cell
    - This pathophysiologic abnormality results in decreased glucose transport in muscle, elevated hepatic glucose production, and increased breakdown of fat.

Hyperglycemia

- Classic S/S
  - Polyphagia (↑ hunger)
  - Polydipsia (↑ thirst)
  - Polyuria (↑ urination)

- Other symptoms
  - Blurred vision
  - Fatigue
  - Weight loss
  - Poor wound healing (cuts, scrapes, etc.)
  - Dry mouth
  - Dry or itchy skin
  - Impotence (male)
  - Recurrent infections
    - vaginal yeast infections
    - groin rash
    - external ear infections

•It is important to remember that not everyone with diabetes will have all these symptoms

•Many people with Type 2 diabetes may not have any of them

Hyperglycemia

- Missed insulin
- Not enough insulin
- Excessive nutrition
- Stress
- Illness
- Infection
- Pregnancy
- Malfunction in insulin pump
- Denatured insulin (heat, extreme cold)
- Decreased absorption of insulin at inject site (overuse of site)
- Decreased metabolic rate
- Alcohol consumption

Diabetic Ketoacidosis (Dka)

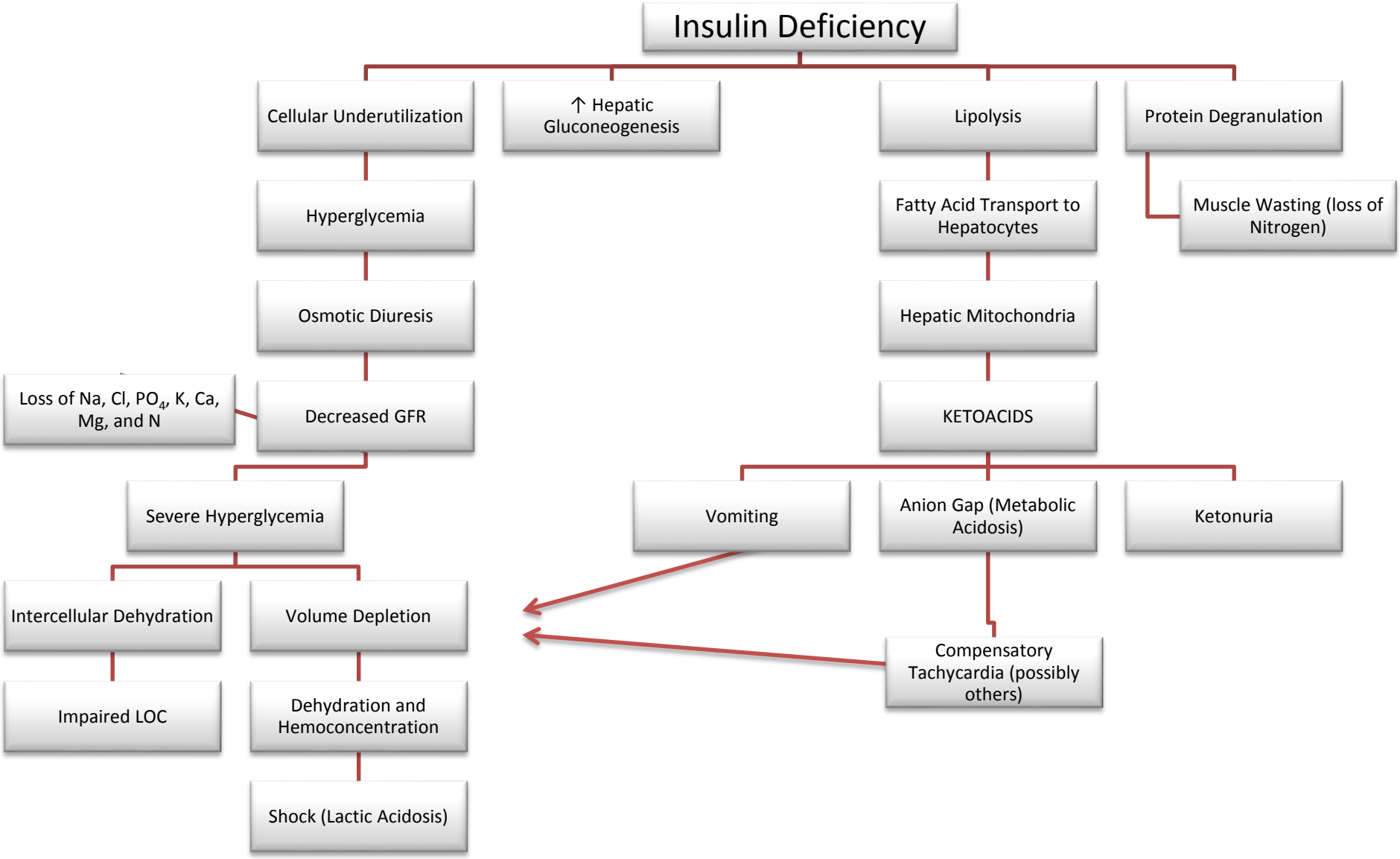
- Usually associated with Type I diabetes
- DKA results from an absence of or resistance to insulin.
  - Inadequate insulin dose, not taken
  - Infection
  - Increased stress
  - Increased dietary intake
  - Decreased metabolic rate
  - ETOH
  - Pregnancy
- The low insulin level presents glucose from entering the cells and causes glucose to accumulate in the blood
- As a result, the cells become starved for glucose and begin to use other sources of energy, particularly fat deposits
  - Build up of ketones produces significant acidosis
- Hyperglycemia also leads to dehydration

Diabetic Ketoacidosis (Dka)

- Presentation
  - Extended period of onset (12–24 hrs)
  - Sweet, fruity breath odor.
    - Increased ketone production
- Potassium-related cardiac dysrhythmias.
  - Cellular effects
- Kussmaul's respiration.
  - Direct effect of ketones on the brain stem
- Decline in mental status and coma.

Dka
Signs and Symptoms

- Warm, dry skin
- Dry mucous membranes
- Tachycardia, thready pulse
- Hypotension
- Weight loss
- Polyphagia
- Polyuria
- Polydipsia
- Acidosis
- Abdominal pain (generalized)
- Nausea/vomiting
- Acetone breath (fruit odor)
- Kussmaul's respirations in attempt to reduce CO_2 levels (from acidosis)
- Decreased LOC


Insulin Deficiency
Cellular Underutilization
↑ Hepatic Gluconeogenesis
Lipolysis
Protein Degranulation
Hyperglycemia
Fatty Acid Transport to Hepatocytes
Muscle Wasting (loss of Nitrogen)
Osmotic Diuresis
Hepatic Mitochondria
Loss of Na, Cl, PO_4, K, Ca, Mg, and N
Decreased GFR
KETOACIDS
Severe Hyperglycemia
Vomiting
Anion Gap (Metabolic Acidosis)
Ketonuria
Intercellular Dehydration
Volume Depletion
Compensatory Tachycardia (possibly others)
Impaired LOC
Dehydration and Hemoconcentration
Shock (Lactic Acidosis)

Management

- Medical emergency
  - Hospitalization necessary
    - Insulin (to lower blood glucose level)
    - Potassium (needed to fix hypokalemia that is usually present from metabolic acidosis, osmotic diuresis and vomiting)
    - Magnesium may be used for hypomagnesium due to osmotic diuresis
    - Sodium Bicarbonate for pH
    - Need to find and fix the reason for the DKA.
  - Prehospital
    - Fluid challenge

Why hospitalization?

- Treating Dka is complicated
  - Requires frequent patient monitoring to adjust fluids, insulin, and electrolyte levels
  - Blood glucose needs to be monitored hourly
    - Appropriate adjustments to induce a gradual decline in blood glucose
  - Ketone levels will be corrected within several hours
  - pH and bicarbonate usually improve significantly within 6 to 8 hours, but the return of a normal plasma bicarbonate level may take 24 hours
  - When blood glucose falls to 250 to 300 mg/dL (13.88 to 16.65 mmol/L)
    - Glucose is added to the IV fluids to reduce the risk of hypoglycemia
    - Insulin dosage may then be reduced, but the continuous IV infusion of regular insulin should be maintained until plasma and urine are consistently negative for ketones
  - Once the patient is stabilized he/she will be switched to subcutaneous regular insulin every 4 to 6 hours
  - Any lapse in insulin therapy during the first 24 hours after recovery may result in a rapid resurgence of hyperketonemia

Dka

- Significant mortality rate - 10%
- Hypotension and/or coma adversely affects prognosis
- Major causes of death
  - Circulatory collapse
  - Hypokalemia
  - Infection
  - Acute cerebral edema
    - Rare
    - Frequently fatal complication and occurs primarily in children more often than in adolescents and young adults

Hyperosmolar Hyperglycemia Nonketotic Coma (HHNK)

- Usually develops more insidiously than DKA
  - Results in blood glucose levels up to 55 mmol/L
- Life threatening emergency
  - Older patients with type 2 diabetes
  - Undiagnosed diabetes
- Differs from DKA
  - Patient is still hyperglycemic, but there is enough insulin to avoid ketongenesis
  - But not enough to adequately get glucose into cells
  - Sustained hyperglycemia results in marked dehydration
  - Often related to dialysis, infection, and medications

Hyperosmolar Hyperglycemia Nonketotic Coma (HHNK)

- Common in older patients who do not monitor glucose levels on a regular basis
- Common with:
  - Preexisting cardiac history
  - Inadequate insulin secretion
  - Medication use
    - thiazides, glucocorticoids, phenytoin, sympathomimetics, propranolol, immunosuppresants
- Similar signs and symptoms as DKA, but no ketone bodies are produced.
- Mortality rate of 20 – 50%

Hyperosmolar Hyperglycemia Nonketotic Coma (HHNK)

- Presentation
  - Gradual onset over days.
  - Profound polydipsia and polyuria, orthostatic hypotension, and altered mental status.
- Assessment and management
  - Difficult to distinguish from diabetic ketoacidosis in the prehospital setting.
  - Treatment is identical to diabetic ketoacidosis.

Table 30-3 Diabetic Emergencies

Diabetic Ketoacidosis	Hyperglycemic Hyperosmolar Nonketotic (HHNK) Acidosis	Hypoglycemia
Common Causes	**Common Causes**	**Common Causes**
Cessation of insulin injections	Physiologic stress (such as infection or stroke) producing hyperglycemia and a noncompensated diuresis, modulated by both insulin and glucagon activity	Excessive administration of insulin
Physiologic stress (such as infection or surgery) that causes release of catecholamines, potentiating glucagon effects and blocking insulin effects		Excess insulin for dietary intake
		Overexertion, resulting in lowered blood glucose level
Signs and Symptoms	**Signs and Symptoms**	**Signs and Symptoms**
Polyuria, polydipsia, polyphagia	Polyuria, polydipsia, polyphagia	Weak, rapid pulse
Warm, dry skin and mucous membranes	Warm, dry skin and mucous membranes	Cold, clammy skin
Nausea/vomiting	Orthostatic hypotension	Weakness, uncoordination
Abdominal pain	Tachycardia	Headache
Tachycardia	Decreased mental function or frank coma	Irritable, agitated behavior
Deep, rapid respirations (Kussmaul's respirations)		Decreased mental function or bizarre behavior
Fruity odor on breath		Coma (severe cases)
Fever (if associated with infection)		
Decreased mental function or frank coma		
Management	**Management**	**Management**
Fluids, insulin as directed	Fluids, insulin as directed	Dextrose

Table 30-4 Diagnostic Signs by System for Diabetic Emergencies

System	Diabetic Emergency		
	Diabetic Ketoacidosis	*HHNK Coma*	*Hypoglycemia*
Cardiovascular			
Pulse	Rapid	Rapid	Normal
Blood Pressure	Low	Normal to Low (may be affected by position, or orthostatic)	Normal
Respiratory			
Respiration rate	Exaggerated air hunger	Normal, unlabored	Normal or shallow
Breath odor	Acetone (sweet fruity)	None	None
Nervous			
Headache	Absent	None	Present
Mental state	Restlessness/ unconsciousness	Lethargy/ unconsciousness	Apathy, irritability/ unconsciousness
Tremors	Absent	Absent	Present
Convulsions	None	Possible	In late stages
Gastrointestinal			
Mouth	Dry	Dry	Drooling
Thirst	Intense	Excessive	Absent
Vomiting	Common	Common	Uncommon
Abdominal pain	Frequent	Common	Absent
Ocular			
Vision	Dim	Normal	Double vision (diplopia)

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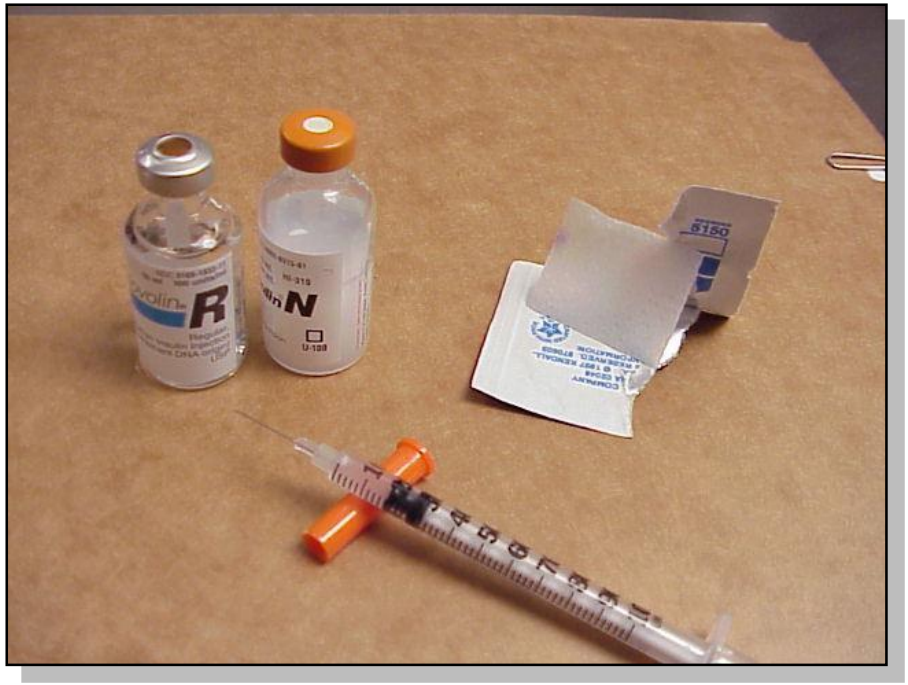
Diabetic Medications

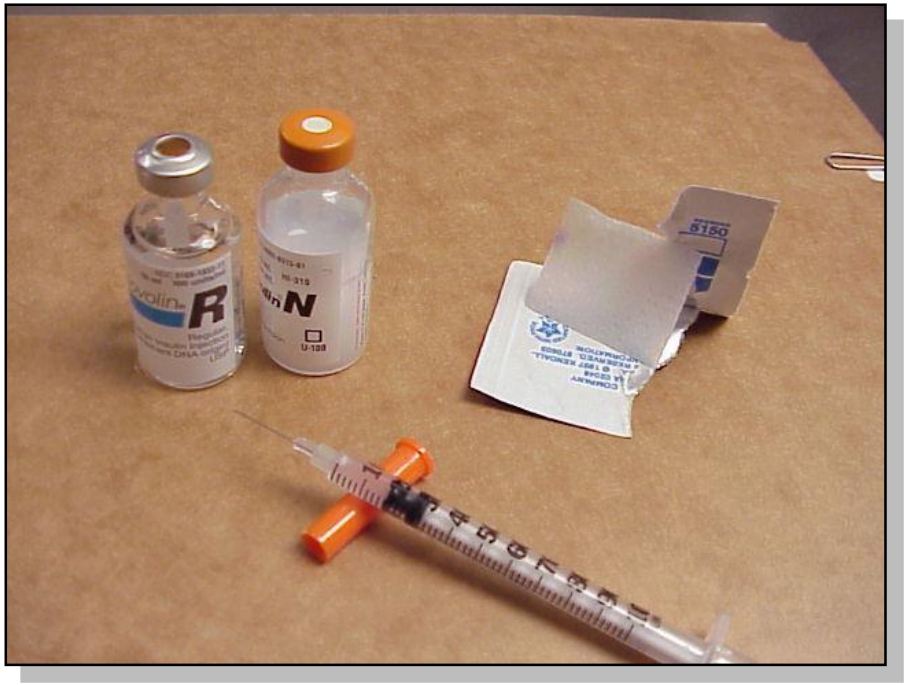
Medications - IDDM

- Different types available from different sources:
  - Beef/Pork
  - Synthetic
- Major companies:
  - Novolin
  - Humalin



Medications - IDDM

- Insulin available as U100 insulin
- This means there are 100 units of insulin in every milliliter of liquid



Insulin – SQ Injections

- Insulin is injected subcutaneous
- Injection sites include:
  - Arms, Thighs, Calfs (children), Abdomen, Buttocks.
- Insulin injections are taken as few as once a day and as many as 6 – 7 per day.
- Lifestyle dictates how many injections you take.
- Insulin works differently in different people and depends on:
  - Injection site
  - Amount of insulin

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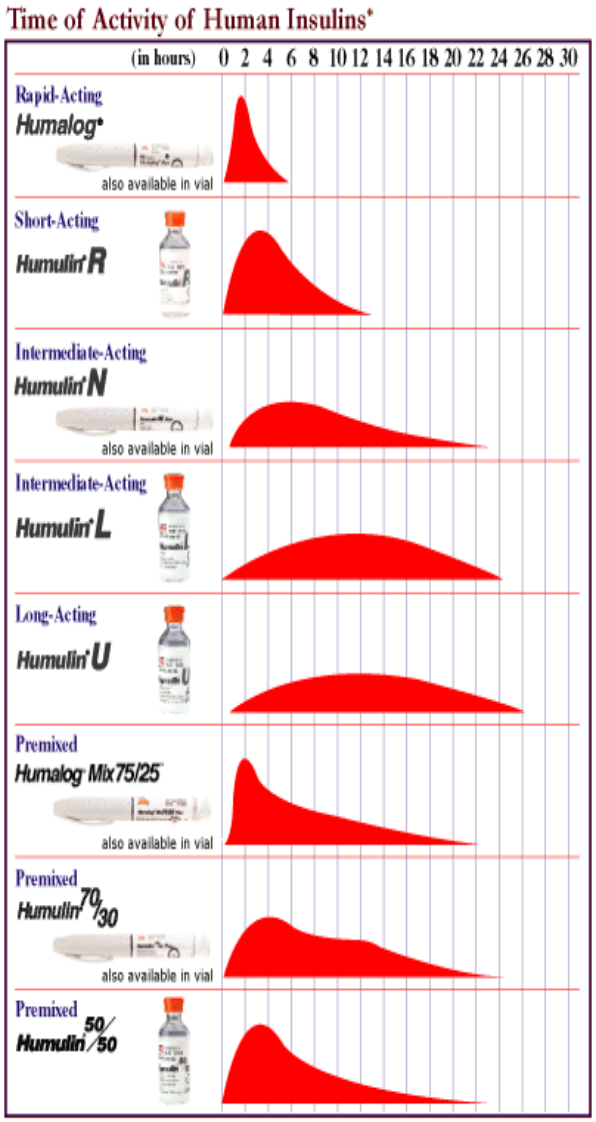
Types of Insulin

Types of insulin (approved for use in Canada)		
Insulin type/action (appearance)	**Brand names (generic name in brackets)**	**Dosing Schedule**
•Rapid-acting analogue (clear) •Onset: 10-15 minutes •Peak: 60-90 minutes •Duration: 3-5 hours	Apidra® (insulin glulisine) Humalog® (insulin lispro) NovoRapid® (insulin aspart)	Usually taken right before eating, or to lower high blood glucose
•Short-acting (clear) •Onset: 30 minutes •Peak: 2-3 hours •Duration: 6.5 hours	Humulin®-R Novolin®ge Toronto	Taken about 30 minutes before eating, or to lower high blood glucose
•Intermediate-acting (cloudy) •Onset: 1-3 hours •Peak: 5-8 hours •Duration: up to 18 hours	Humulin®-N Novolin®ge NPH	Often taken at bedtime, or twice a day (morning and bedtime)
•Long-acting analogue (clear and colourless) •Onset: 90 minutes •Peak: none •Duration: up to 24 hours	Lantus® (insulin glargine) Levemir® (insulin detemir)	Usually taken once or twice a day
Premixed (cloudy) A single vial or cartridge contains a fixed ratio of insulin (rapid- or fast-acting insulin to the percent of intermediate-acting insulin)	•NPH Humulin® (30/70) •Novolin®ge (30/70, 40/60, 50/50) •Humalog® Mix25 and Mix50 •NovoMix 30	Depends on the combination



Injected Insulin

- Syringes
- Pens
- Jet injectors
- Pumps

Insulin - Syringes

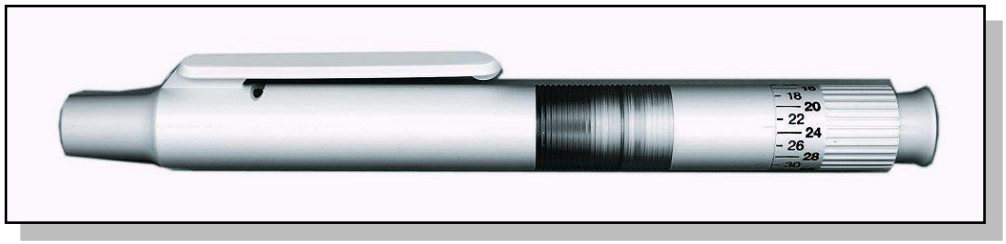
- A new U100 syringe is used for each injection
- Insulin is drawn into syringe, some people mix two insulin type in one syringe.
- People are trained in techniques for drawing and injecting insulin

Insulin Syringes

- Insulin in injected into:
  - Arms, Legs, Abdomen, Buttocks, and Calves (children)
- Repetitive injection in one area will cause a buildup of scar tissue and cause the insulin to not be absorbed.
  - Therefore rotation of injection sites in necessary.

Insulin Pens

- Look like an oversized writing pen
- Insulin comes in two sized vials that are inserted into the pen and remain there until the insulin in used.
- A short needle is attached to the end of the pen for injection, and replaced for the next use.



Insulin Pens

- Many people find insulin pens more convenient.
- Good for multiple does regimes.

Insulin jet injectors

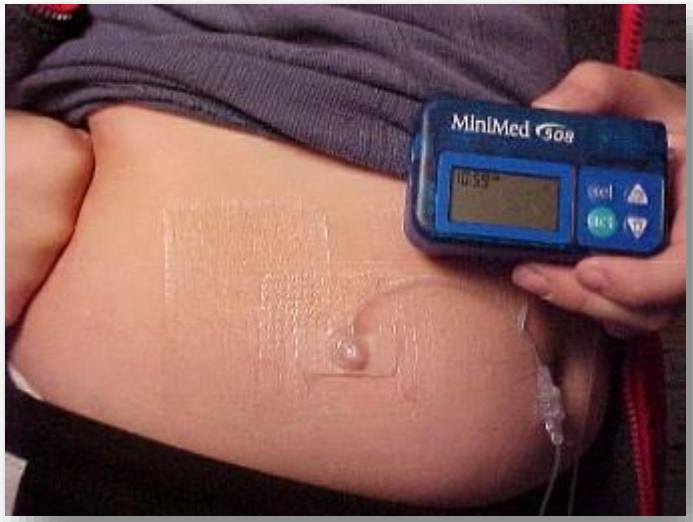
- Jet injectors have no needle at all
- A tiny stream of insulin is forced through the skin by pressure.
- Many people find bruising occur at injection site
- Not as popular due to bruising and pain

Inhaled Insulin

- Clinical trial being run through QEII in Halifax
- Insulin inhaled into lungs and absorbed into blood stream
- Capsules of 2 units insulin crushed into chamber and inhaled

Insulin Pumps

- Quickly becoming more popular, especially with children
- Offer greater flexibility and improvement in glucose control



Insulin Pumps

- A microcomputer
- Size of a pager
- Deliver insulin in units as little as 1/10th of a unit.
- Syringe reservoir filled with insulin and placed in the pump

Insulin Pumps

- Thin tube (infusing set) is connected to reservoir
- Small needle which is inserted into the fatty tissue and left there
- Infusion set is changed ever 3 – 4 days

Insulin Pumps

- Pump is programmed to deliver a continuous infusion or background insulin (basal) that is meant to mimic what would naturally occur with a functioning pancreas.
- Whenever food is eaten, an appropriate ratio of insulin is given via the pump to accommodate the grams of carbohydrate in the food eaten.

Insulin Pumps

- A malfunction can set a patient into Hyperglycemia/DKA within hours. Frequent glucose testing in required.
- In a diabetic emergency, do not disconnect the insulin pump. Call the 1-800 number on the back of the insulin pump and a nurse on the line will tell you exactly what to do.

Medication NIDDM

- Medication for type II diabetes are typically pills used to control hyperglycemia of stable, mild, nonketosis prone maturity onset diabetes.
- Type II diabetes is initially attempted to be controlled by diet and exercise, medications are a second choice.
- If diet, exercise and pills do not work, some type II patients are put on insulin to control their blood sugar levels.

Type II Diabetes

- Oral Hypoglycemic Agents
  - Are not "insulin pills“
    - At the present time, insulin cannot be given in pill form
    - Like any other protein, it would be digested before the body could absorb it
- Groups of medications:
  - Sulfonylureas
  - Biguanides
  - Alpha-Glucosidase Inhibitors
  - Meglitinides
  - Thiazolidinediones

Sulfonylureas

- Includes:
  - Glyburide (DiaBeta, Euglucon, etc…)
  - Gliclazide (Diamicron)
  - Glimepiride (Amaryl)
  - Tolbutamide
- Act on the ß-cells to stimulate pancreatic insulin release
- They also enhance glucose uptake by peripheral tissues
- Hypoglycemic episodes are possible on this medication.
- Very important to eat three regular meals a day on this medication

Biguanides

- Includes: Metformin (Glucophage)
  - Decreases hepatic glucose production
  - Delays glucose absorption
  - Enhances insulin-mediated glucose uptake
  - Additive Effect with sulfonylureas

Alpha-Glucosidase Inhibitors

- Includes: Prandase (Acarbose)
  - Delay the absorption of glucose from the GI tract by inhibiting the enzyme responsible for breaking down complex carbohydrates
  - These carbohydrates are still absorbed but further down the G I tract so there is a more gradual and delayed rise in postprandial blood glucose concentration
  - You must eat for this medication to work.

Meglitinides

- Includes: Repaglinide (Gluconorm), Nateglinide (Starlix)
  - Increases pancreatic insulin output
    - but in a more physiological way than do the sulfonylureas
  - Its action is faster and has a shorter duration
    - Allows for greater insulin availability after eating, when it is most needed, and less insulin when it is not

Thiazolidinediones

- Includes: Pioglitazone (Actos), Rosiglitazone (Avandia)
  - This class of drugs is known as Insulin Sensitizers
  - Act by increasing overall insulin sensitivity, with evidence of effects in both liver and skeletal muscle.
    - Act on the cell nucleus to affect messages from DNA
    - Dramatically improve the action of insulin on the muscle cell

Medication – Type 3

- Typically gestational diabetes is controlled with a specifically followed diet regime along with exercise and reduction of stress.
- Some cases require insulin to ensure blood glucose level control for safety of mother and baby.

Blood Glucose Testing

- Blood glucose levels should be tested before every meal, typically 3 – 4 times per day minimum.
- With an increase in number of daily injections, the number of tests per day will increase.

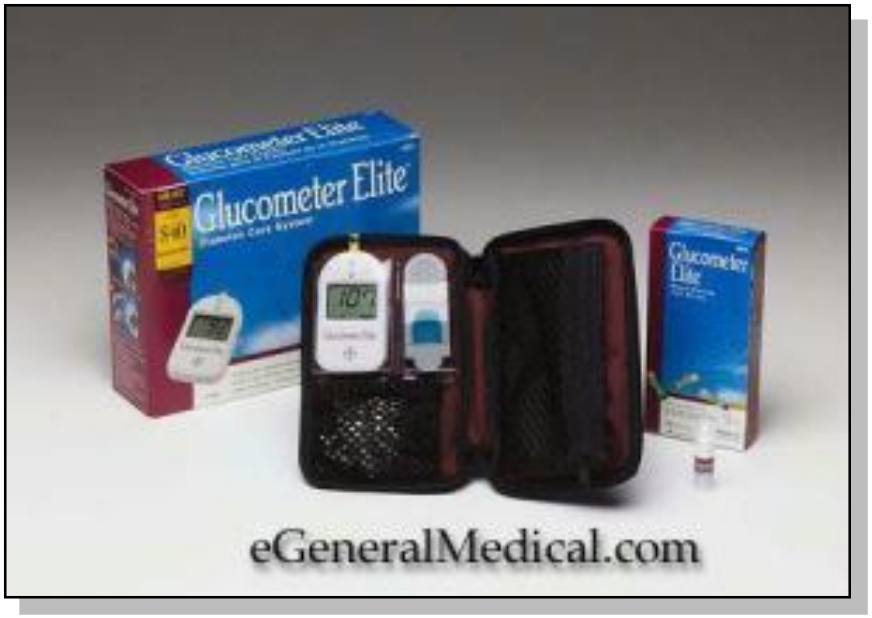
Glucose control ranges

	Target Zone	Suboptimal	Inadequate
Glucose level before meal	4 – 7 mmol/L	7.1 – 10 mmol/L	Over 10 mmol/L
Glucose 1 – 2 hours after eating	5.0 – 11 mmol/L	11.1 – 14 mmol/L	Over 14 mmol/L
HbAIc*	Less than 0.07%	0.07 – 0.084%	Over 0.084%

*measurement of glycated hemoglobin which in turns tells how much glucose was in the blood stream. Increased glucose caused increased glycated hemoglobin. Acts like an average glucose test.

Blood Glucose Testing Techniques

- Blood glucose meters can be purchased from your local pharmacy.
- Blood glucose testing requires a small drop of blood to place on a blood glucose testing strip



Blood Glucose Testing

- Canada – mmol/L measurement
- US – mg/dl

- Conversion: (mmol/L) x 18 = (mg/dl)

Glucose in Urine

- When blood glucose levels rise, glucose will be dumped from the body through urine.
- Patients can tell that there is glucose in their urine by carrying out urine glucose test.
- Ketones can be detected in the same matter (urine test)

Complication of Diabetes

- Atherosclerosis
  - Build up of fatty deposits and damage to the arteries and blood vessels from high blood glucose levels

- Avoided/controlled by:
  - Maintain good blood glucose levels
  - don’t smoke
  - Eat well, Keep healthy weight
  - Exercise regularly
  - Check Blood Pressure
  - Have cholesterol and blood fat levels checked

Complications of Diabetes

- Retinopathy
  - Microaneurysms of and damage to the blood vessels of the retina
  - Will cause progressive loss of vision.
- Early detection and laser treatment can help save vision.
- Avoided/controlled by:
  - Maintain good blood glucose levels
  - have eyes examined by eye specialist once a year
  - Check Blood Pressure (very important)
  - and notify your doctor if there are changes in your vision..

Complications of Diabetes

- Neuropathy
  - damage to the filtering units in the kidneys caused by high blood glucose levels.
  - Once the kidney is damaged, it can not be repaired.
- Eventually, dialysis will be needed.
- Avoided/controlled by:
  - Maintain good blood glucose levels
  - blood pressure checked regularly
  - know signs of urinary tract infections
  - have urine tested for protein at least 1 per year
  - have blood tested for creatinine
  - eat a well balanced diet.

Complication of Diabetes

- Peripheral nerve damage from high blood glucose levels.
- Causes desensitization of nerves causing loss of feeling/constant tingling in arms/legs

Complication of Diabetes

- Decrease in healing time for wounds
- Causes ulcers
- If untreated or treatment unsuccessful, can lead to necrosis/gangrene resulting in need for amputation.
- High blood glucose level provides a great environment for bacteria to grow. This stops the wound from healing.

Diabetes Insipidus

- What is it?
  - A rare disorder of water metabolism where the balance between how much intake fluid is not balanced with the outtake fluid.
  - Is caused by a lack of, or non-response to, the anti-diuretic hormone vasopressin.
  - This hormone controls water balance by concentrating urine.
  - Patients with diabetes insipidus urinate too much, so they need to drink a lot to replace the fluid they lose.
  - Vasopressin is made by the cells of the hypothalamus and is stored and secreted the posterior pituitary gland.
  - When vasopressin is present at normal levels, more water is reabsorbed and less urine is formed.
  - Do not confuse diabetes insipidus with the diabetes mellitus.

Diabetes Insipidus

- Types of Diabetes Insipidus?
  - While the symptoms of these two disorders are similar, the causes are different.
    - Central diabetes insipidus
    - Nephrogenic diabetes insipidus

Diabetes Insipidus

- Central diabetes insipidus
  - Vasopressin is either missing or present at a low level.
  - This low level or lack of vasopressin is due to a malfunction in the part of your brain, the posterior pituitary gland
  - Caused by:
    - Tumour of the pituitary gland.
    - Head injury, with damage to pituitary gland.
    - Brain tumour.
    - Infections, such as meningitis or encephalitis.
    - Haemorrhage in the pituitary gland or in adjacent structures. Aneurysm.

Diabetes Insipidus

- Central diabetes insipidus
- S/S
  - excessive urination (polyuria)
  - excessive thirst (polydipsia)
  - are often extremely tired because they cannot get enough sleep uninterrupted by the need to urinate.
  - urine is very clear and odourless.
  - If this disorder is untreated, they could become seriously dehydrated

Diabetes Insipidus

- Nephrogenic diabetes insipidus
  - Is much less common than central diabetes insipidus.
  - May be caused by kidney diseases that make the kidneys unable to respond to vasopressin.
  - While there is enough vasopressin in the body (unlike in central diabetes insipidus), the kidneys cannot respond to the hormone's signal to reabsorb water.
  - The disease may be acquired or inherited by male children.

- S/S
  - excessive urination (polyuria)
  - excessive thirst (polydipsia).

Diabetes Insipidus

- Treatment
  - The first step in treating this disease is correct diagnosis.
  - Medications to balance fluid intake with your urine output
  - Central diabetes insipidus
    - Is usually corrected by giving synthetic vasopressin
      - It is administered by placing some in a small plastic tube and gently blowing the liquid into the nostril where it is readily absorbed by the tissue of the nose lining. It can also be taken orally, but the dose required is higher.
  - Nephrogenic diabetes insipidus
    - Thiazide diuretics
      - Thiazide diuretics have been shown to stimulate the production of a hormone that helps your body retain salt. This added amount of salt keeps you from losing too much water.
- It is possible to overdose on synthetic vasopressin
  - become drowsy and do not pass urine

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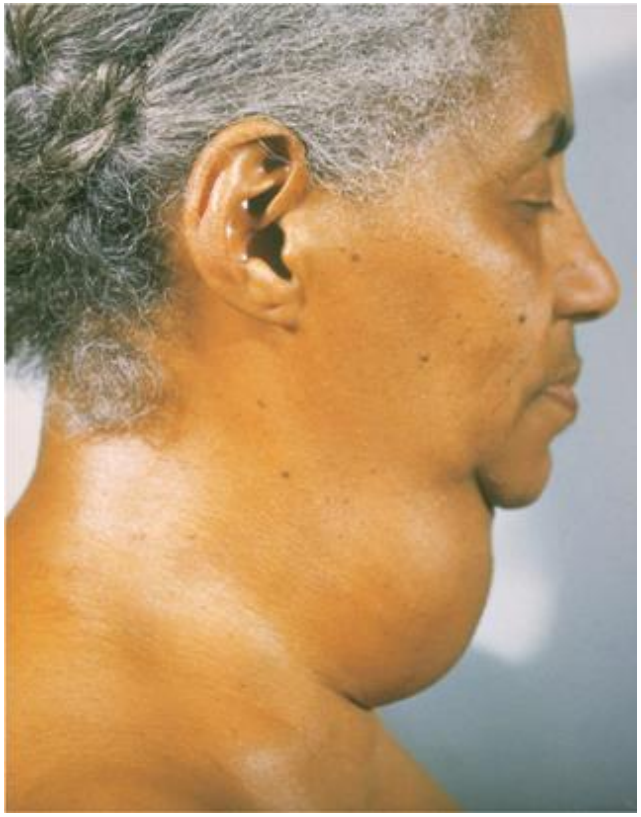
Disorders of the Thyroid Gland

Thyroid Disorders

- Causes
  - Congenital defects
  - Genetic disorders
  - Infection
  - Tumors
  - Autoimmune disorders
  - Hormonal disorders (puberty and pregnancy)
  - Nutritional disorders

Grave's Disease



- Probably hereditary in nature.
- Much more common in women.
- Autoantibodies are generated that stimulate thyroid tissue to produce excessive hormone
- Enlargement (goiter) of the thyroid gland
  - Autoimmune response
- Leads to swollen neck and protruding eyes

Grave's Disease

- Presentation
  - Agitation, emotional changeability, insomnia, poor heat tolerance, weight loss, weakness, dyspnea.
  - Tachycardia and new-onset atrial fibrillation.
  - Protrusion of the eyeballs or goiters.
- Assessment and management
  - Usually arise from cardiovascular signs/symptoms.
  - Manage signs and symptoms.

Thyrotoxic Crisis

- Thyroid storm
- Life-threatening emergency
  - Usually associated with severe physiologic stress or overdose of thyroid hormone.
- Thyroid hormone
  - Moves from bound state to free state within the blood.

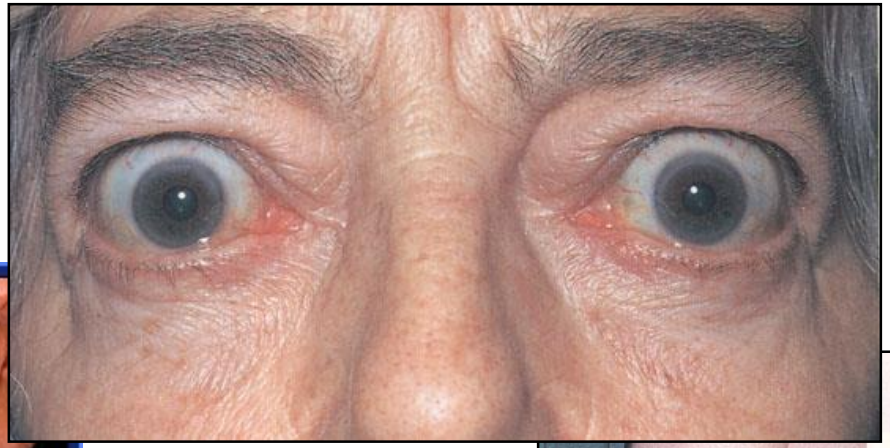
Thyrotoxic Crisis

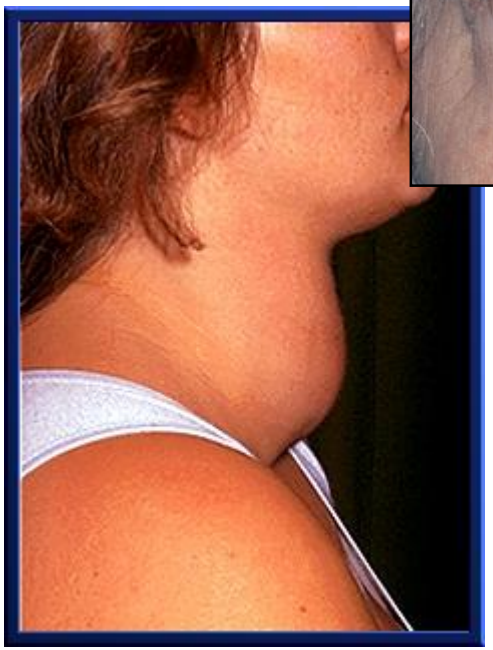
- Presentation
  - High fever (>41º C)
  - Increased activity of sympathetic nervous system.
  - Irritability, delirium or coma
  - Tachycardia and hypotension
  - Vomiting and diarrhea
- Assessment and management
  - Support airway, breathing, and circulation.
  - Monitor closely and expedite transport.

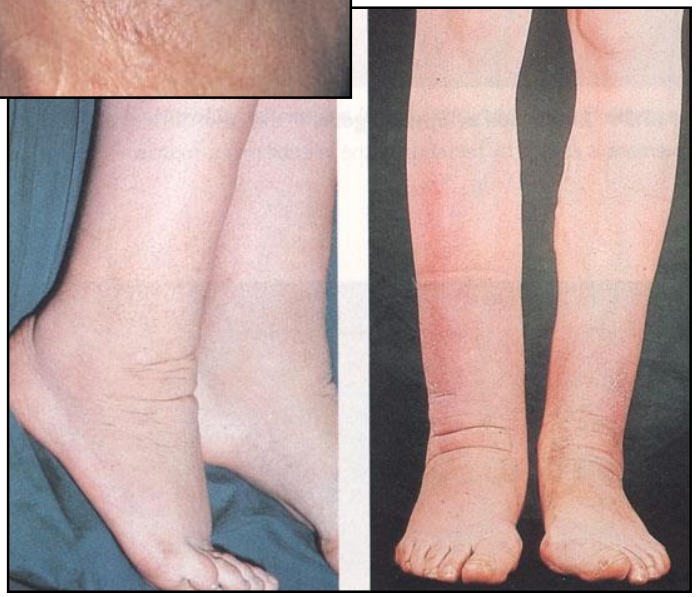
Hyperthyroid

- Increased production of thyroid hormone
- S/S
  - Protruding eyes
  - Skin Changes
    - Thickening of the epidermis
    - Fast nail growth
    - Soft velvety dry skin
    - Increased pigmentation
    - Increased skin temperature
    - Diffuse thinned scalp hair
    - Red palms
    - Pruritus and hives (rare)
    - Flushed face
    - Pretibial myxedema
    - Increased sweating palms, soles
    - Acropathy (thick fingers and toes)

Hyperthyroid







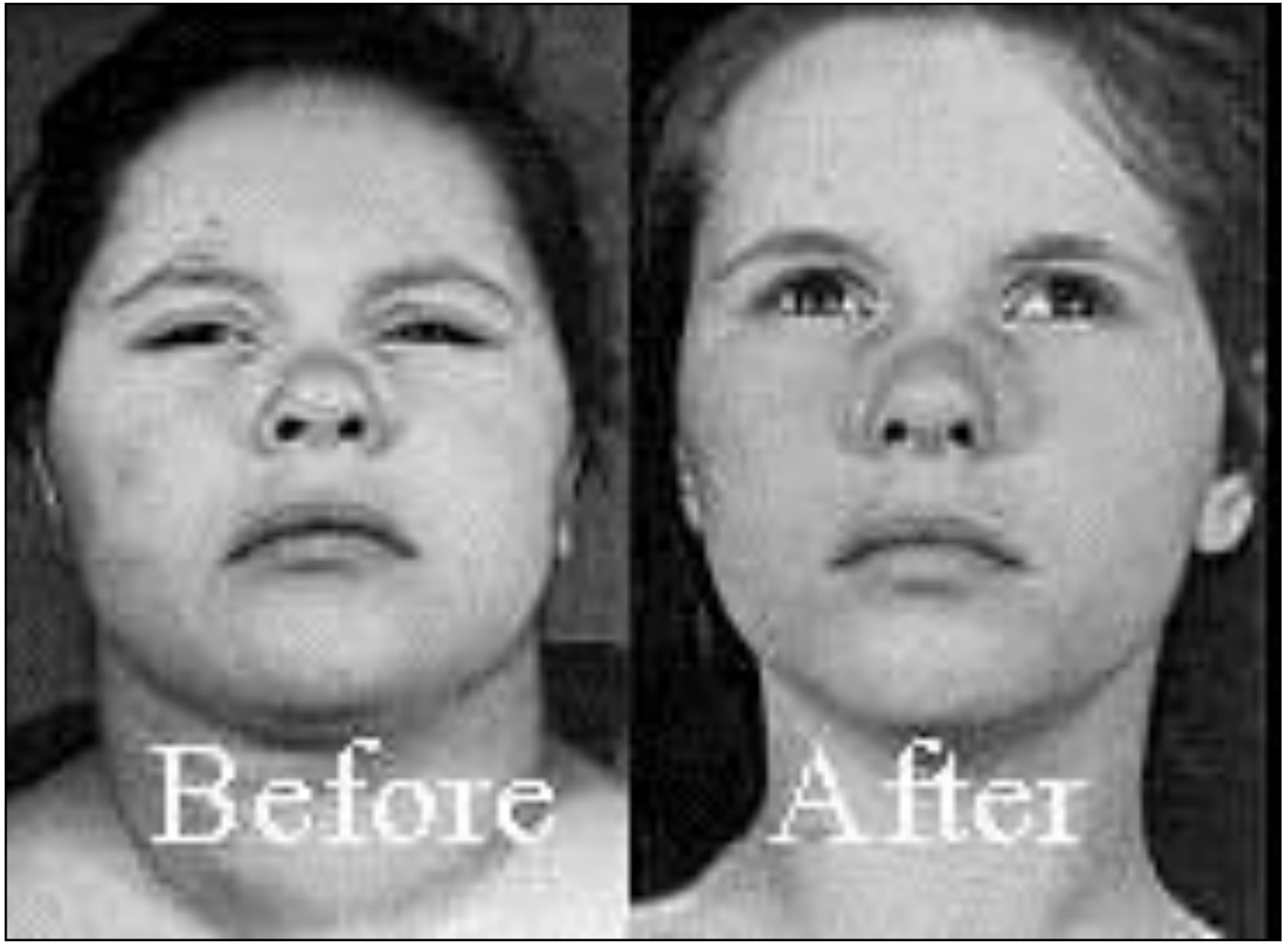
Hypothyroidism and Myxedema

- Can be inherited or acquired.
- Chronic untreated hypothyroidism creates myxedema.
- Thickening of connective tissue in skin and other tissues.
- Triggers for progression to myxedemic coma
  - Infection
  - Trauma
  - CNS depressants
  - Cold environment

Hypothyroid

- Slow metabolic rate due to alteration of glandular function
  - May be due to an autoimmune disease or secondary failure due to tumor, infection or trauma
- S/S
  - Insidious onset of tiredness, fatigue, leg cramps
  - Generalized myxedema
  - Skin Changes
    - Pale, cold, scaly, wrinkled skin
      - can slowly develop a broad nose, swollen lips and puffy eyelids
    - Coarse, dry scalp and hair
    - Absence of sweating
    - Hair loss - scalp groin, lateral eyebrows, etc.
    - Skin colour - ivory - yellow (due to alteration in Vitamin A metabolism – carotenemia)
    - Puffy edema (hands, face, eyelids)
    - Brittle thick nails
    - Eczema craquele
    - Bruising
    - Poor wound healing

Hypothyroid



Myxedema

- Thyroid hormone deficiency
- Associated with
  - Inflammation of thyroid
  - Atrophy of thyroid
  - Consequence of hyperthyroid treatments
- Causes accumulation of mucinous (albuminoid substance in mucous) material in the skin
  - Thickens and coarsens the skin and other tissues (lips and nose)

Presentation

- Fatigue, slowed mental function
- Cold intolerance, constipation, lethargy
- Absence of emotion, thinning hair, enlarged tongue
- Cool, pale doughlike skin
- Coma, hypothermia, and bradycardia

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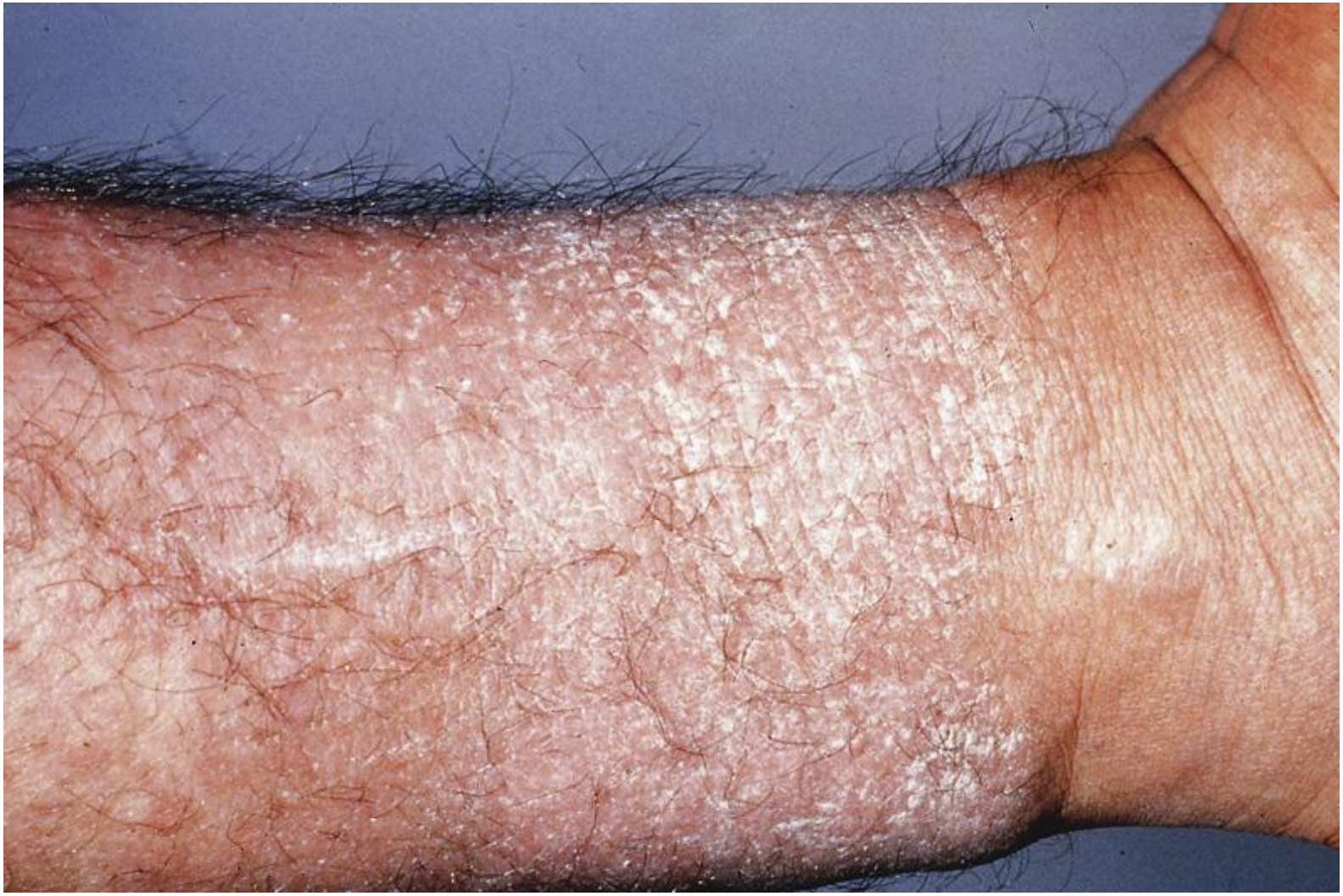
Doughy, edematous skin classic of myxedema



Management

- Focus on maintaining ABCs.
- Closely monitor cardiac and pulmonary status.
- Establish IV access, but limit fluids.
- Expedite transport.

Hyper vs Hypo

Hyperthyroid

- Protruding eyeballs
- Goiter
- Warm flushed skin
- Fever
- Agitation/psychosis
- Hyperactivity
- Weight loss
- Meds
- Iodine
- Tapazole
- Propacil

Hypothyroid

- Facial edema
- JVD
- Cool skin
- Exposure to cold
- Coma
- Weakness
- Weight Gain

Meds

- Synthroid
- Cytomel
- Euthroid

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Disorders of the Adrenal Glands

Hyperadrenalism

- Cushing's syndrome
  - Often due to abnormalities in the anterior pituitary or adrenal cortex.
  - May also be due to steroid therapy for nonendocrine conditions such as COPD or asthma.
  - Rare condition (Usually women 30 – 50 y/o)
- Long-term cortisol elevation causes many changes.
  - Atherosclerosis, diabetes, hypertension
  - Increased response to catecholamines
  - Hypokalemia and susceptibility to infection

Cushing's Syndrome

- S/S
  - Face is round (Moon faced) and red
  - Trunk is Obese
  - Limb muscles atrophy
  - Acne
  - Purple stretch marks on ABD, thighs and breasts
  - Skin is thin and bruises easily
  - Weak bones
  - Increased body and facial hair
  - Hump on back of neck
  - Supraclavicular fat pads
  - Weight gain
  - Hypertension
  - Depression, paranoia
  - Insomnia
  - Diabetes mellitus

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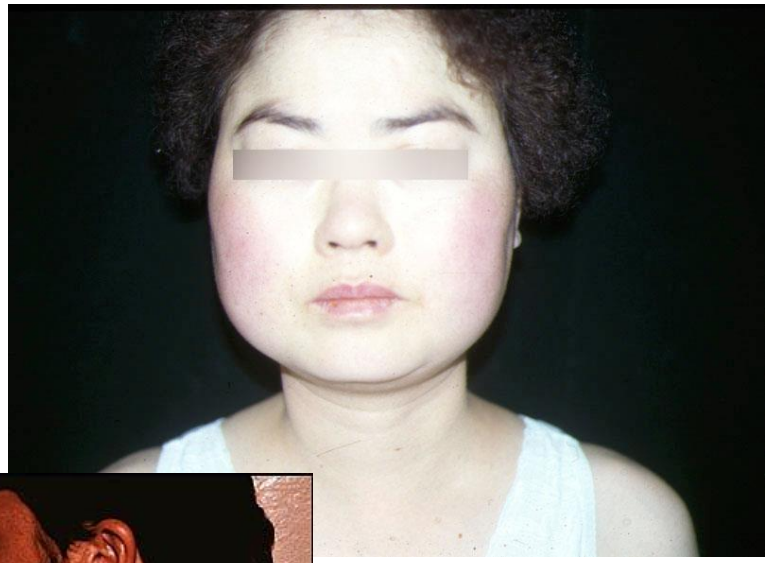
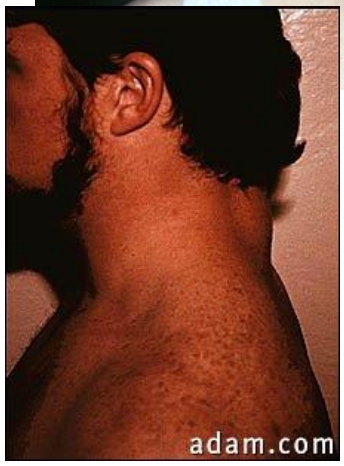
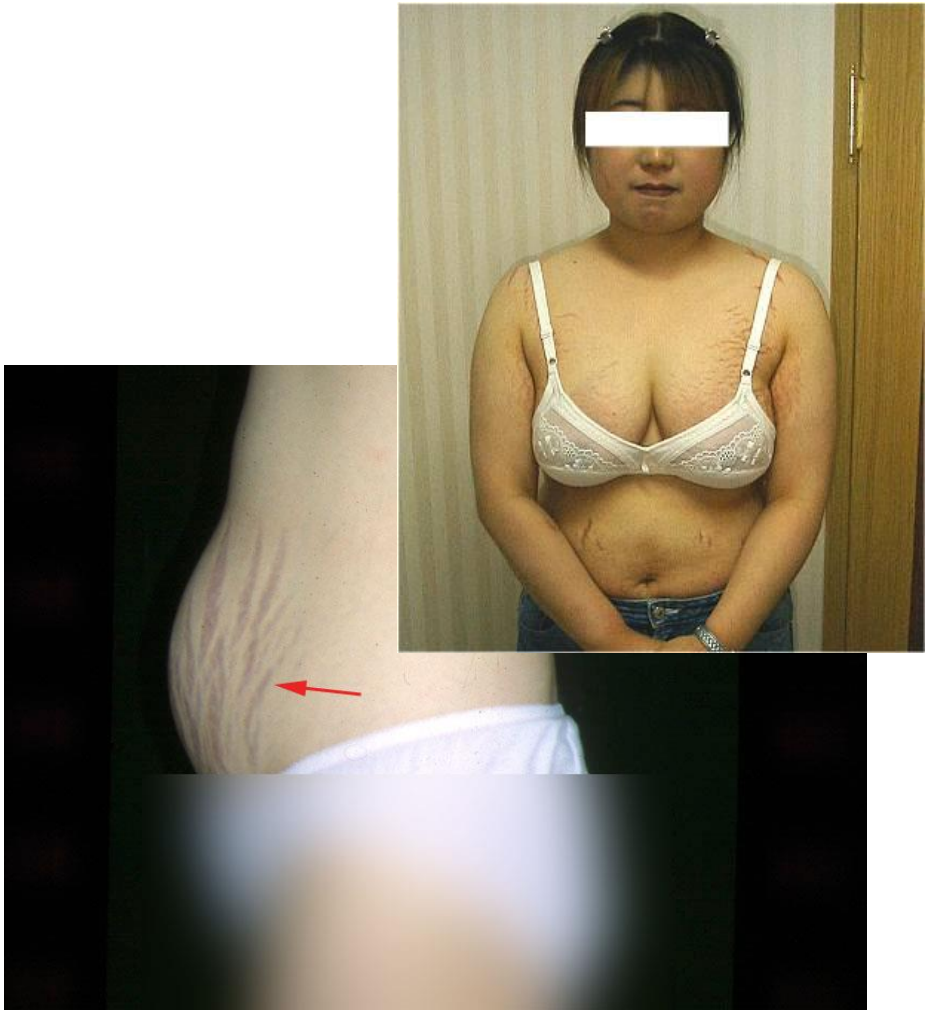
Cushing's Syndrome







Adrenal Insufficiency

- Addison's disease
  - Due to destruction of the adrenal cortex.
    - Deficiency of cortisol and aldosterone
  - Often related to heredity.
  - Result from
    - Stress may trigger Addisonian crisis.
    - May be related to steroid therapy
      - Sudden withdrawal can trigger Addisonian crisis
    - A result of any disease that affects the adrenal cortex (ie. Idiopathic atrophy, infarct, TB or autoimmune)
  - Rare, potentially life threatening

Addison's Disease

- S/S
  - Progressive weakness
  - Progressive weight loss
  - Anorexia
  - Skin hyperpigmentation (increased work of the pituitary gland which stimulates melanin)
  - Hypotension
  - Hyponatremia
  - Hyperkalemia
  - N/V, diarrhea

Addison's Disease

- Disease has slow onset
- Symptoms develop over period of time (months to years)
- Acute episodes (Addisonian crisis)
  - Precipitated by stress (physiological and emotional)
  - Adrenals are unable to meet the needs to help the body cope with stress
  - This results in
    - Drops in blood glucose levels
    - Unable to regulate Na, K and H20 concentrations in blood
    - Hypotension and low blood volume

Cushing vs Addison

Corticosteroid Excess (Cushing's Syndrome)

- Weight Gain
- Weakness
- Buffalo Hump
- Slow healing
- Increased body/facial hair

Meds

- Cytadren (blocks adrenal steroids)
- Metopirone (affects cortisol levels)

Adrenal Insufficiency (Addison's Disease)

- Weight Loss
- Weakness
- Hypotension
- GI Disorders
- Hyperpigmentation

Meds

- Dexamethasone
- Fludrocortisone

Pheochromocytoma

- A tumor of the core of the adrenal glands that secretes excessive amounts of the hormones epinephrine and norepinephrine, resulting in high blood pressure.
- Is a rare disease (Occurs primarily in adults 30-40 years of age)
- This overproduction of hormones causes high blood pressure and increased metabolism and may elevate blood sugar.
- Life threatening if untreated, and can cause stroke, or damage to the kidneys, brain, or heart.
- Is usually benign and does not spread to other organs. It may be associated with other endocrine gland tumors.
- The cause of pheochromocytoma is not known, but scientists suspect a genetic link.

Pheochromocytoma

- Persistent or recurrent high BP
- Excessive sweating
- Palpitations
- Fast pulse
- Headache
- Pallor
- Weight loss
- Constipation
- Warmth or flushing
- Numbness and tingling
- Tremor
- Nervousness
- Feelings of doom
- Rapid breathing
- Abdominal pain, nausea, and vomiting.

Pheochromocytoma

- Diagnosis
  - based on a number of factors including
    - a physical examination
    - blood and urine lab tests, which measure urinary catecholamines
    - imaging, including computed tomography scan (CT scan) and magnetic resonance imaging (MRI).
- Treatment
  - Laparoscopic surgical removal of the tumor is the treatment of choice for pheochromocytoma.
  - Before surgery, medications such as alpha-adrenergic blockers are given to block the effect of the hormones and normalize blood pressure.
  - A beta-blocker, such as Inderal, may also be used.
- During an acute attack or a hypertensive crisis, intravenous Regitine or Nipride are administered to bring the blood pressure down to normal.