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Endocrinology

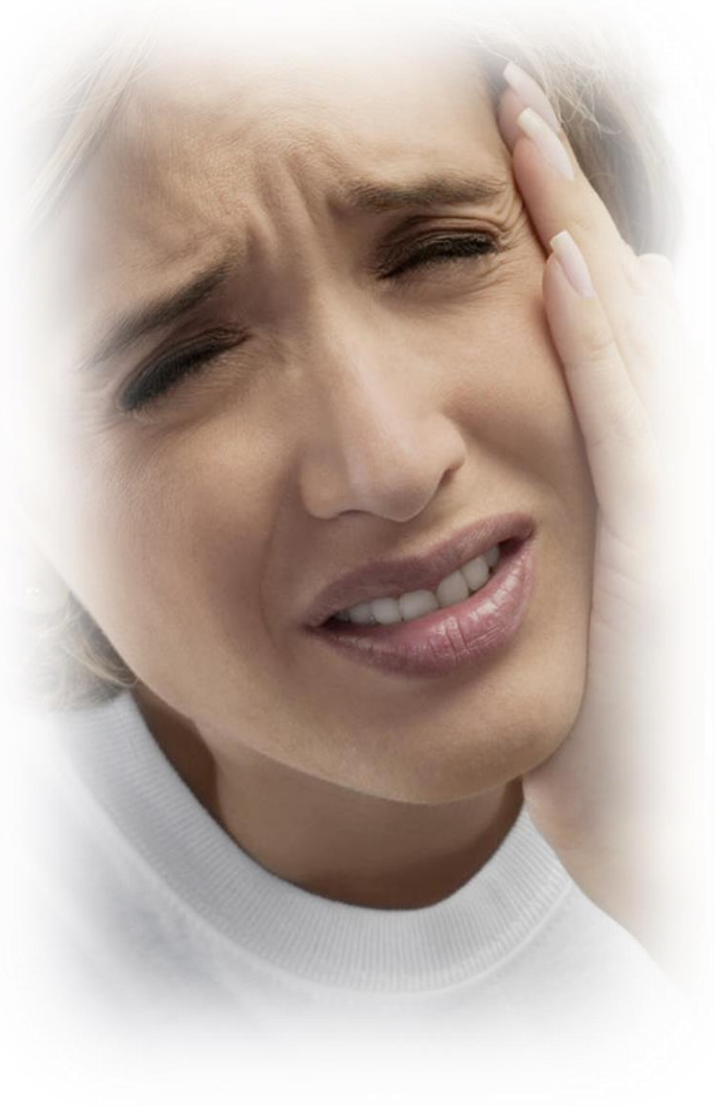
Primary Care Paramedicine

Module: 13

Section: 07

Case Study

- You are dispatched to an arena for a 47 y/o F conscious and breathing, altered level of consciousness



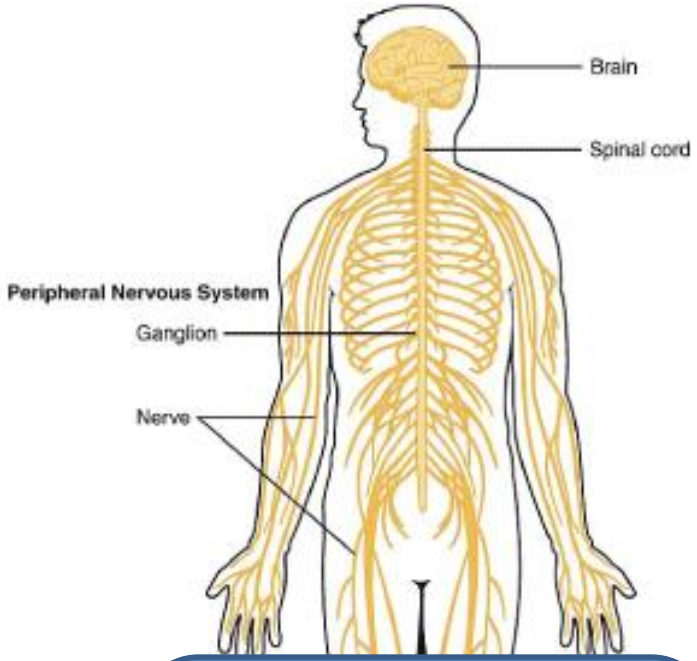
Case study

- On arrival you witness many concerned bystanders offering you information, “her pulse is fast”, “I think she is having a stroke.”
- You find a 47 y/o F sitting upright pale warm and clammy, not answering questions appropriately, point to her mouth

Endocrine System

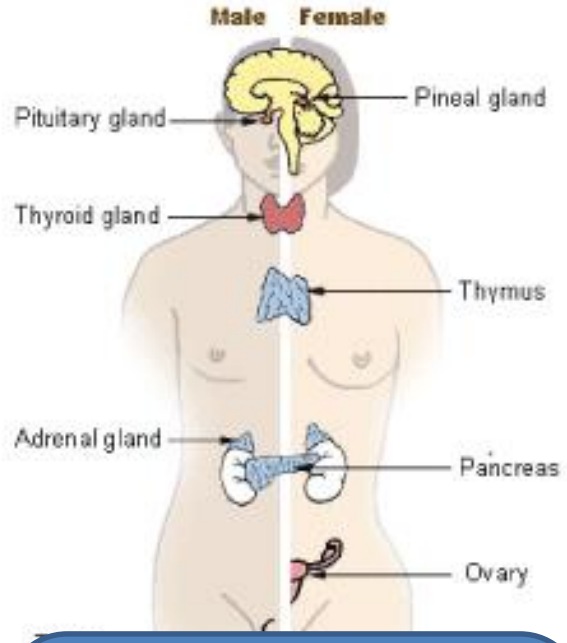
- The purpose of the endocrine system is to control, coordinate and integrate cellular activity by regulating cellular and organ function throughout life to maintain homeostasis
  - Homeostasis is the maintenance of a relatively constant internal environment

Remember



Nervous System

- Electrical impulses are used to transmit signals
- Quick and short acting



Endocrine System

- Hormones are chemical messengers that target cells through the bloodstream
- Delayed but long acting

Endocrinology

DISORDERS OF THE ENDOCRINE SYSTEM

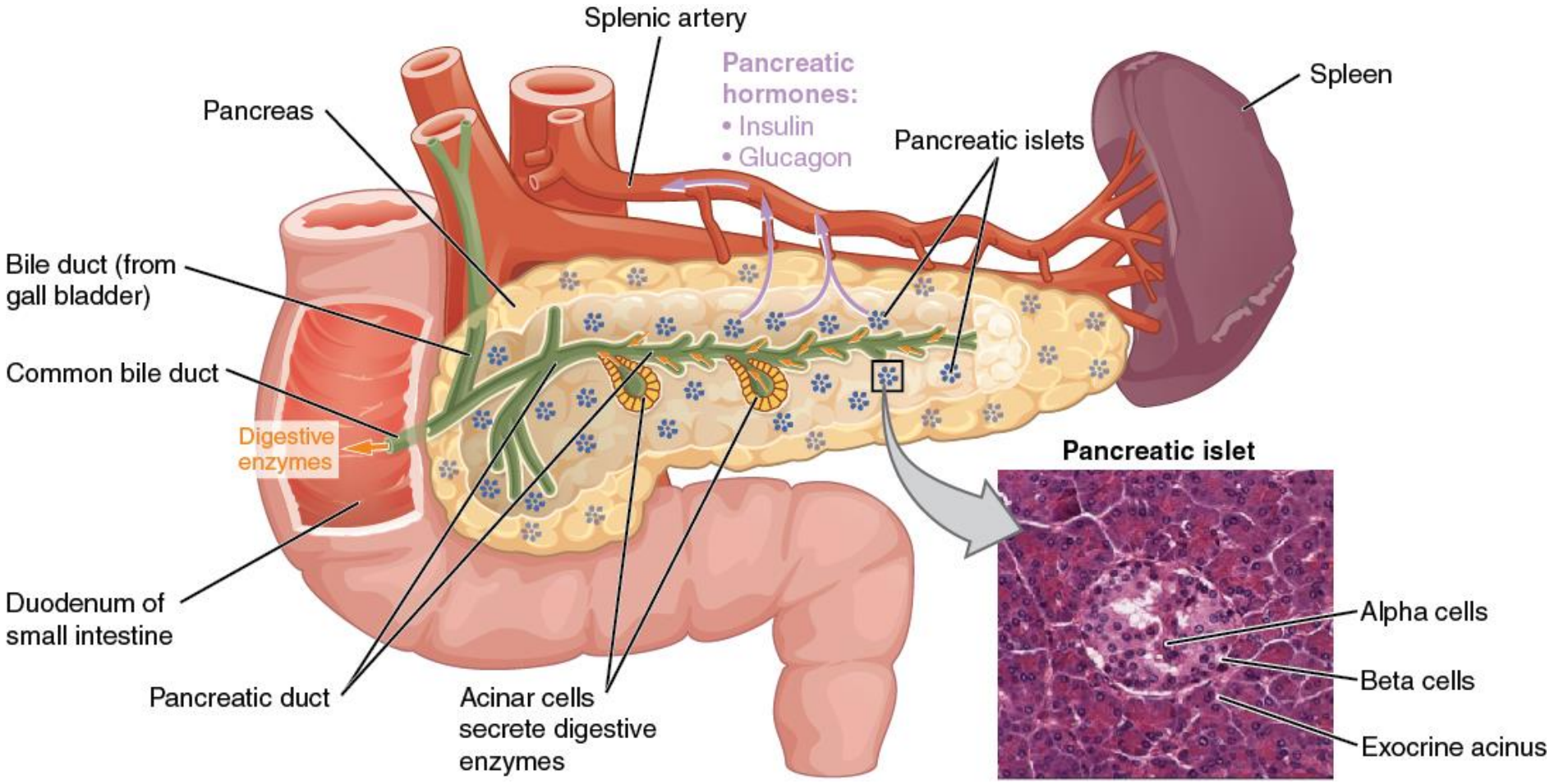
Endocrine Disorders

- **Causes**
  - Hormone hypersecretion, leading to excessive target-gland activity
  - Target-organ underactivity can be caused by insufficient secretion of a hormone by a gland or resistance to a hormone at the target organ.
- **Effects**
  - Determined by the degree of dysfunction of the gland
  - Age and sex of the patient also determine the effects.
  - Range from barely detectable variations to extreme dysfunction

Endocrinology

DISORDERS OF THE PANCREAS

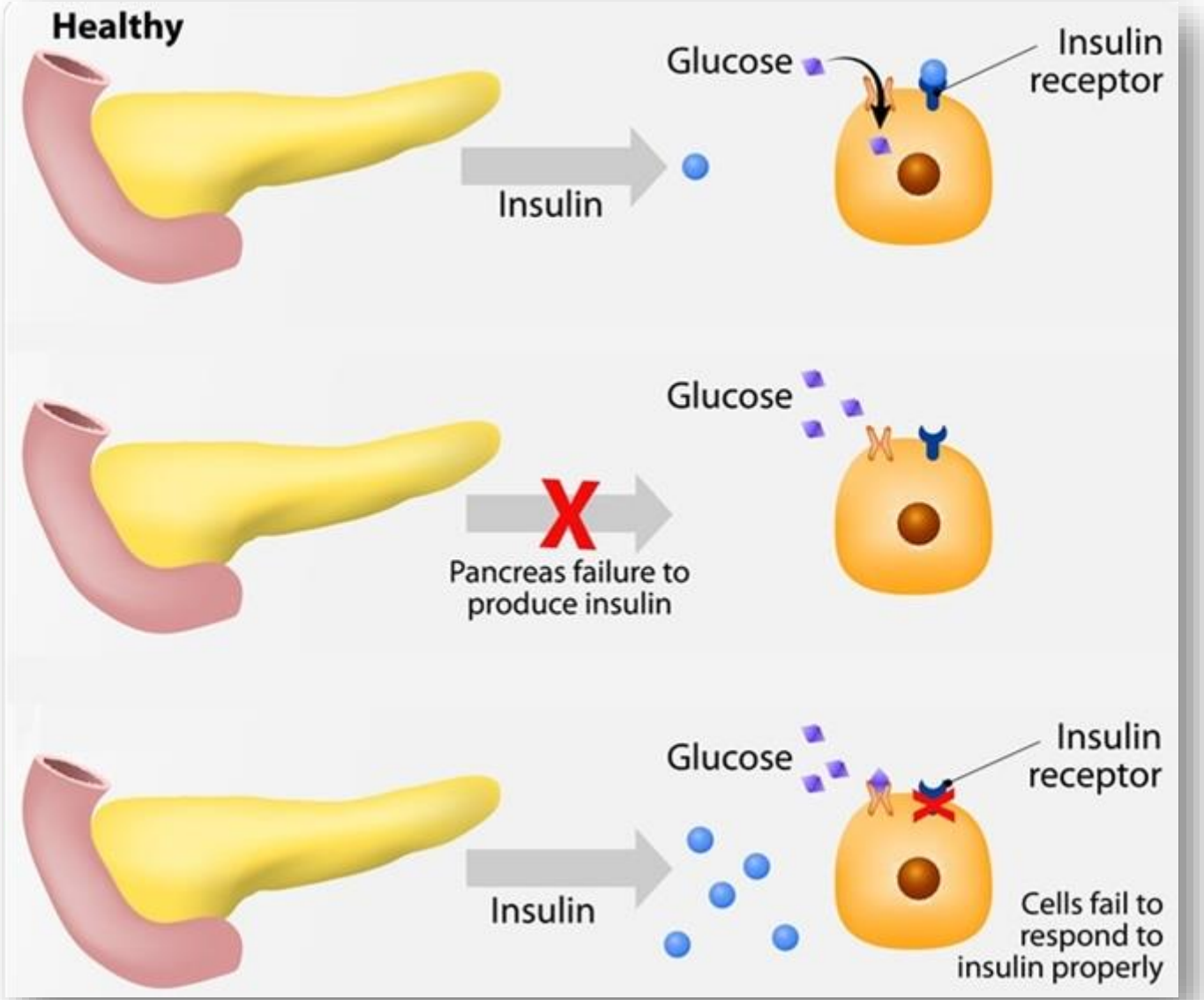
Pancreas


Splenic artery
Pancreatic hormones:
• Insulin
• Glucagon
Spleen
Pancreas
Pancreatic islets
Bile duct (from gall bladder)
Common bile duct
Digestive enzymes
Pancreatic islet
Duodenum of small intestine
Alpha cells
Beta cells
Exocrine acinus
Pancreatic duct
Acinar cells secrete digestive enzymes

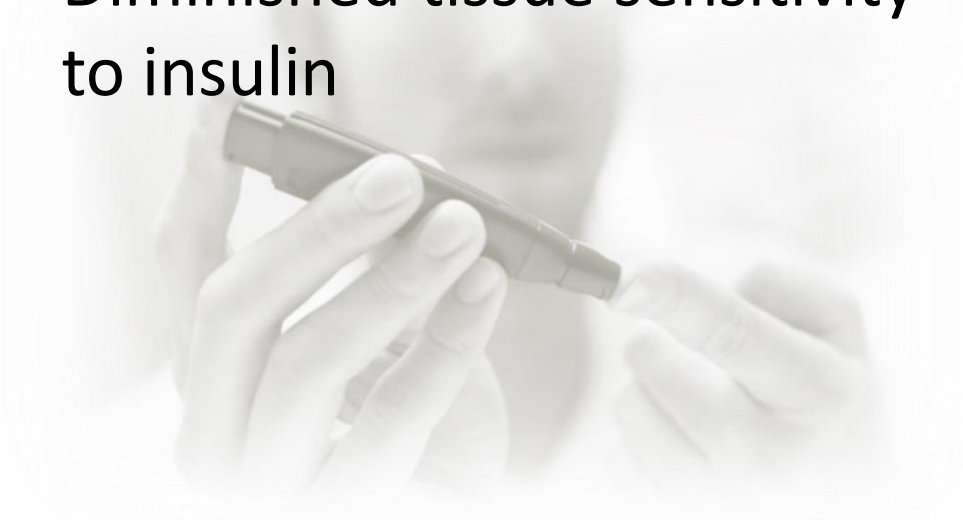
Diabetes Mellitus

- Body's ability to metabolize simple carbohydrates (glucose) is impaired
- Characterized by:
  - Passage of large quantities of urine containing glucose
  - Significant thirst
  - Deterioration of body function
  - Inability to sufficiently metabolize glucose

Diabetes Mellitus



- A result of:
  - No insulin production
  - Inadequate insulin production
  - Diminished tissue sensitivity to insulin



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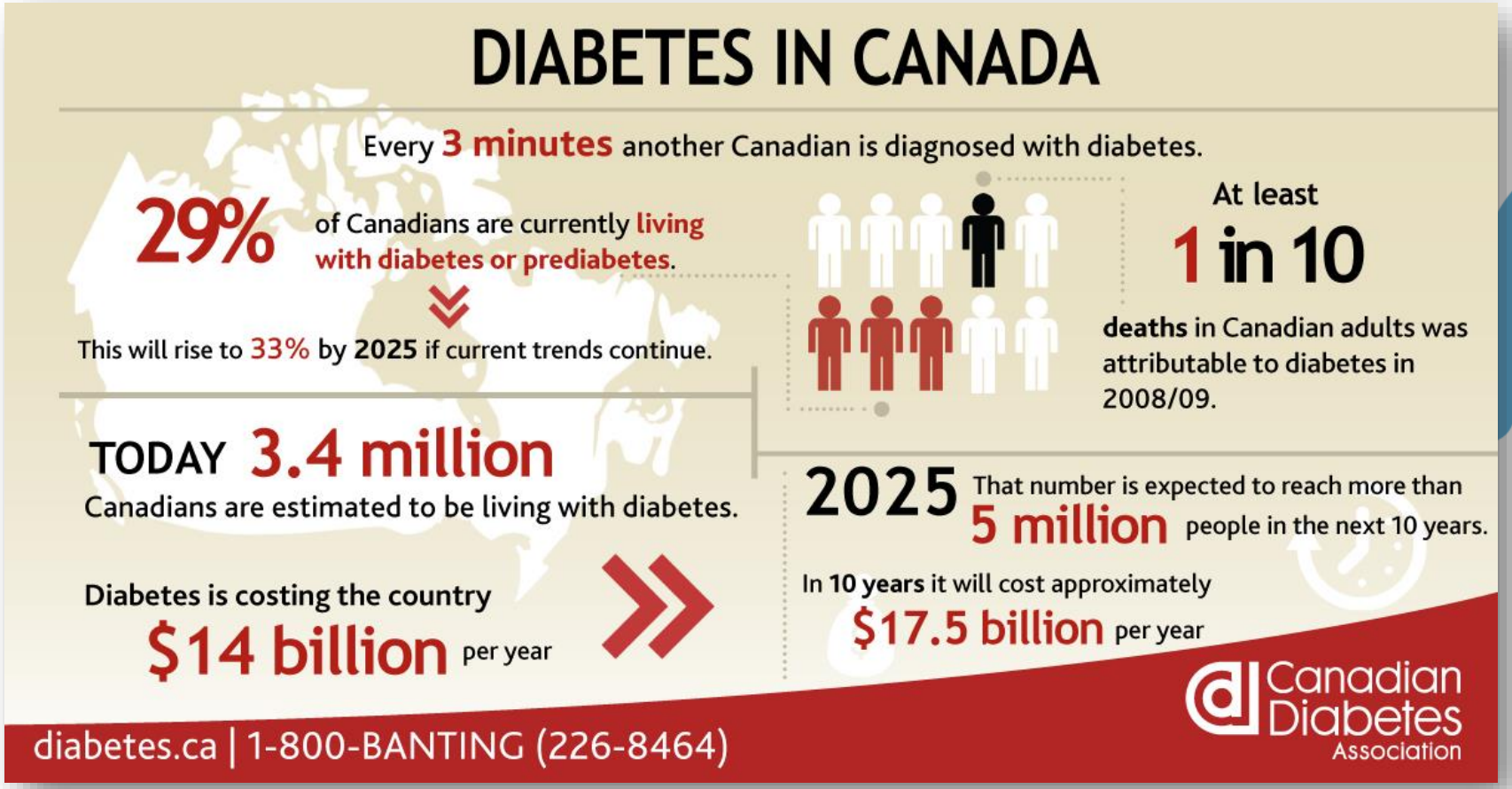
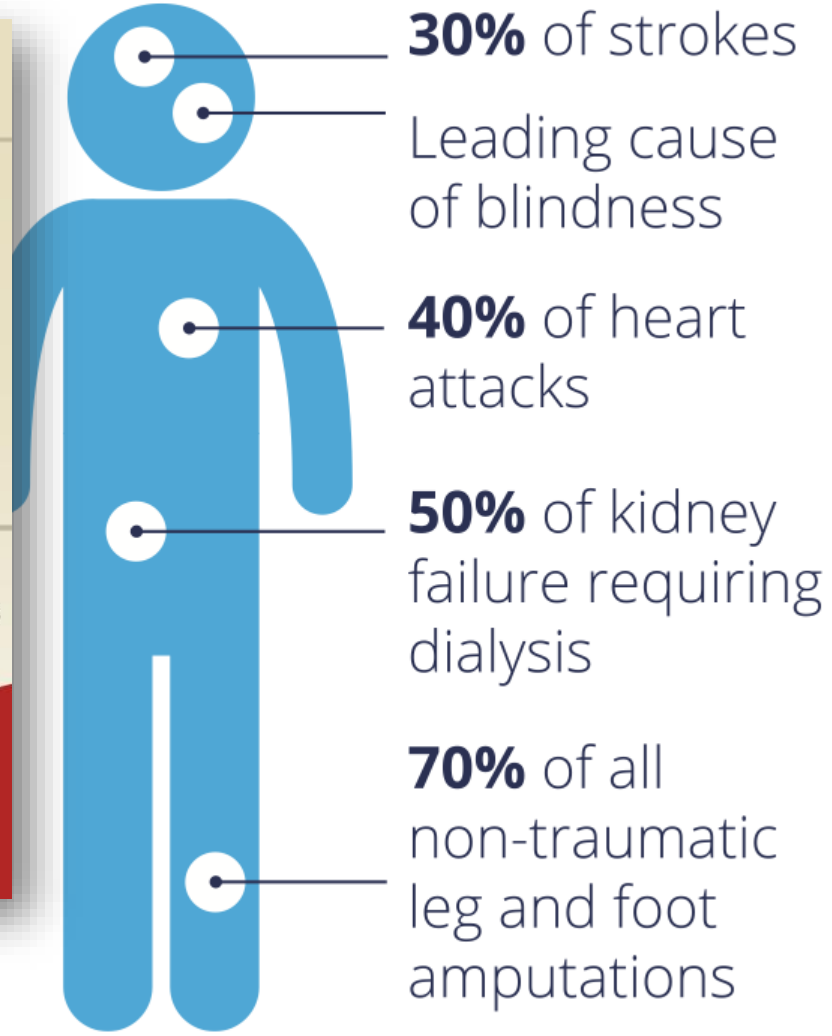
Diabetes Mellitus

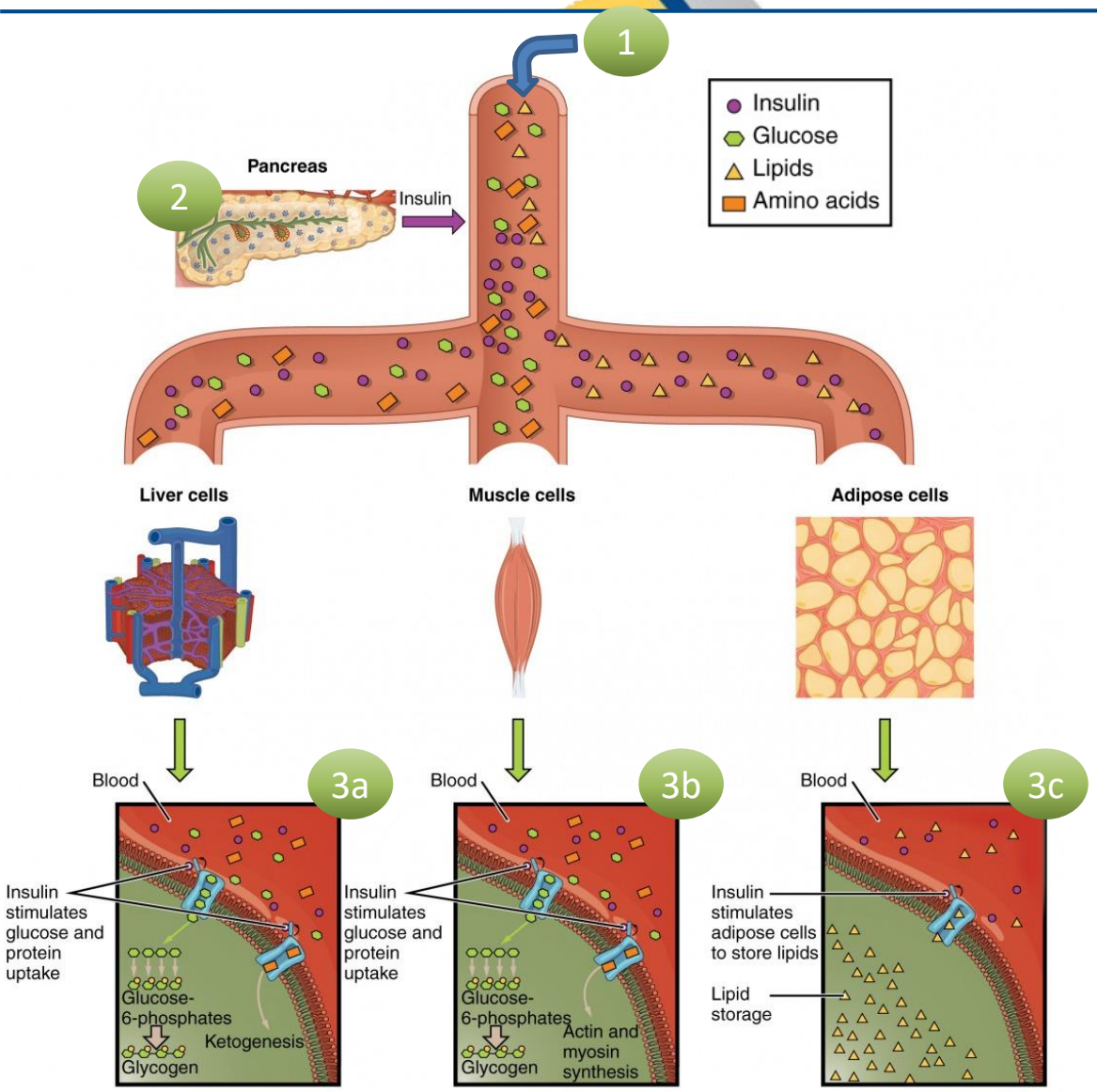




Diabetes Pathophysiology

- Although plasma glucose is high, cellular uptake is low
  - Body counterintuitively attempts to increase plasma glucose
- Muscle begins to metabolize glycogen stores and fatty acids for fuel
- Liver in response to epinephrine metabolizes free fatty acids
  - ketonemia, ketonuria, ketoacidosis
- Occasionally the body cannot use glucose as a primary energy source
  - Fat metabolism

Fed State (Absorptive state)



1. Digested food enters the blood stream from the intestines. Blood sugar concentration rises

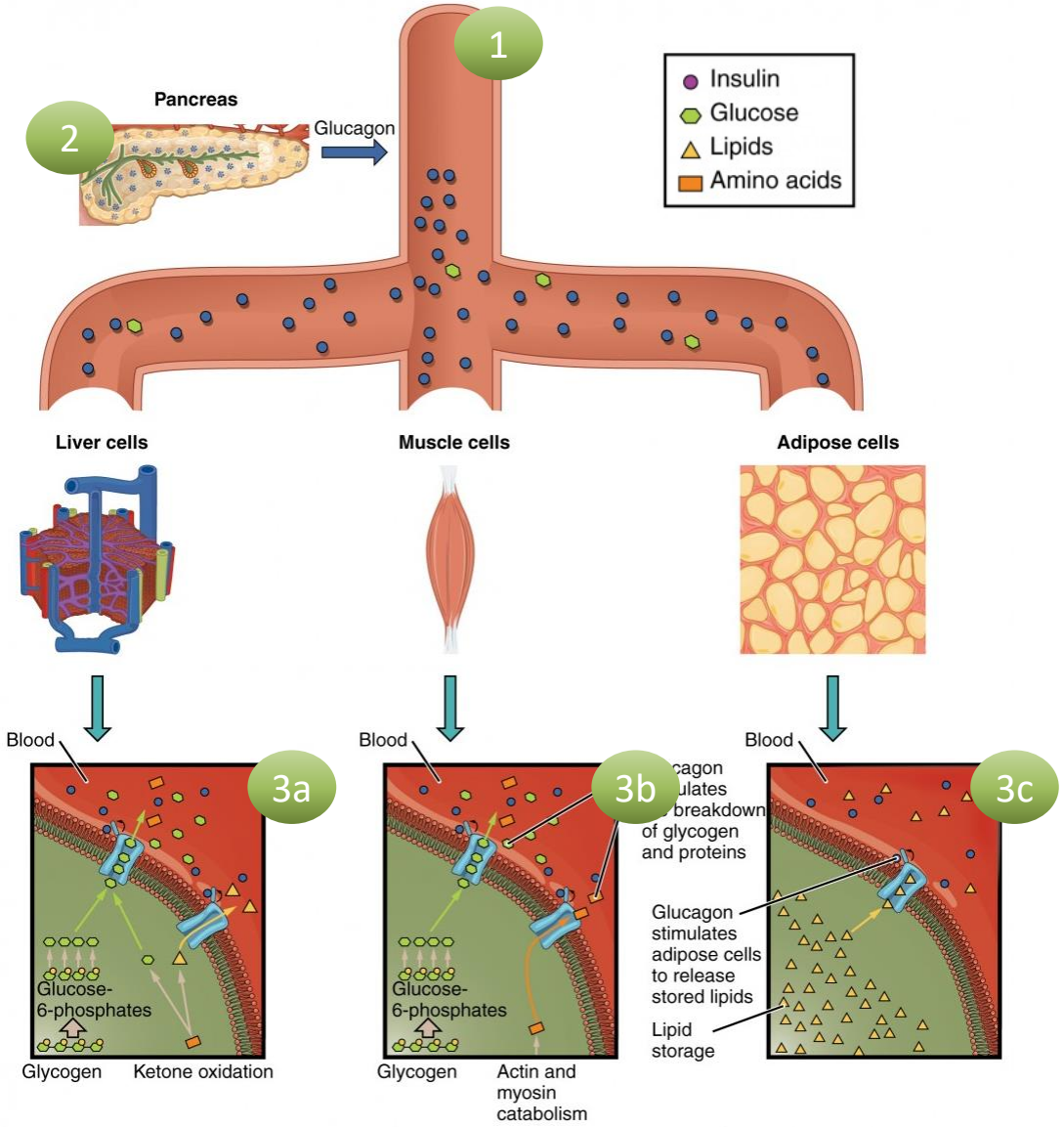
2. Release of digested nutrients into the blood stimulates insulin release by the pancreas

3a. Liver cells convert excess glucose to glycogen for storage. Amino acids are converted to ketones that can later be converted to acetyl CoA when needed.

3b. Muscles convert excess glucose to glycogen for storage. Amino acids are used to synthesize actin and myosin for muscle rebuilding.

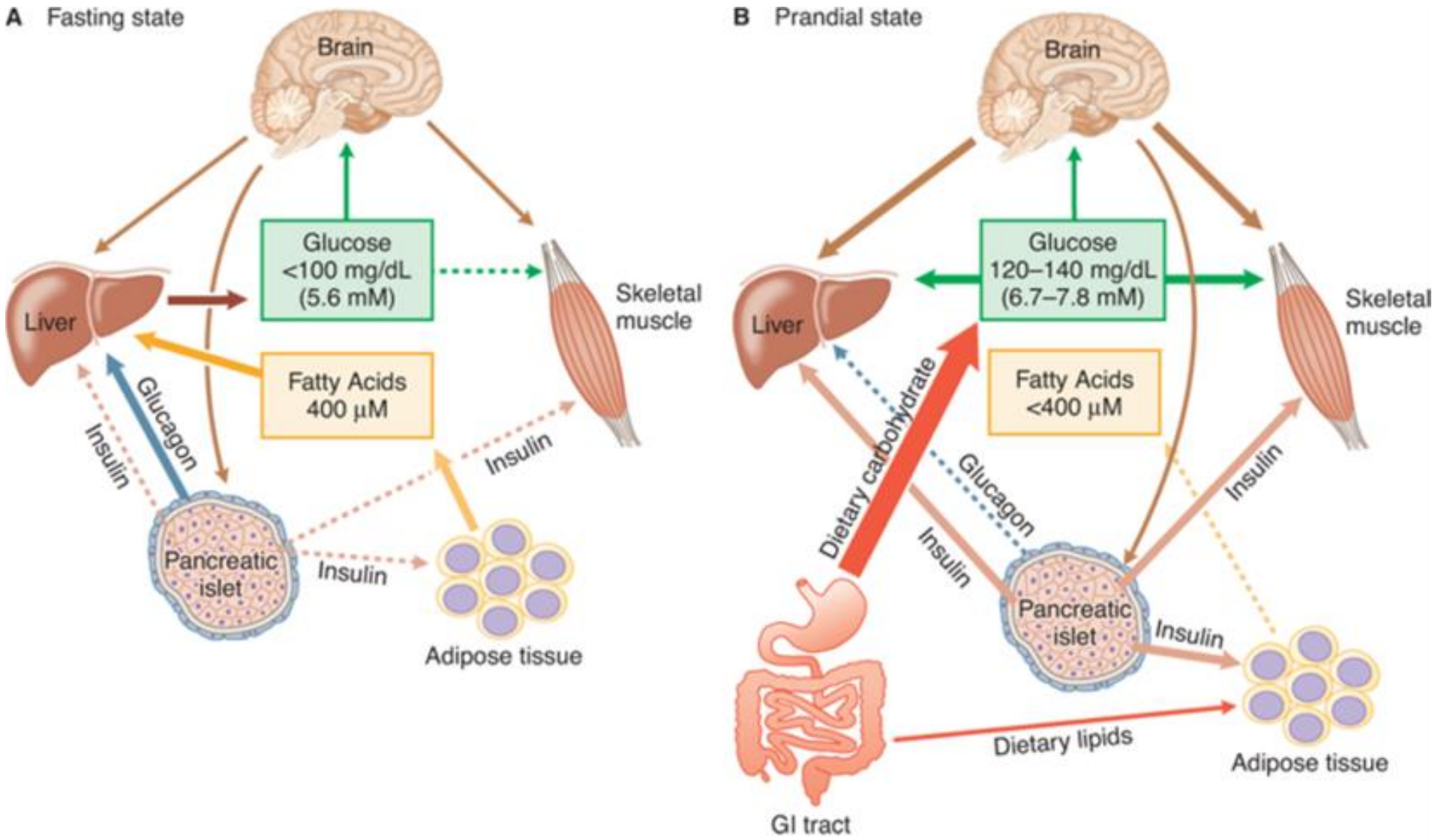
3c. Adipose cells store excess lipids increasing fat reserves.

Fasting State (Postabsorptive state)

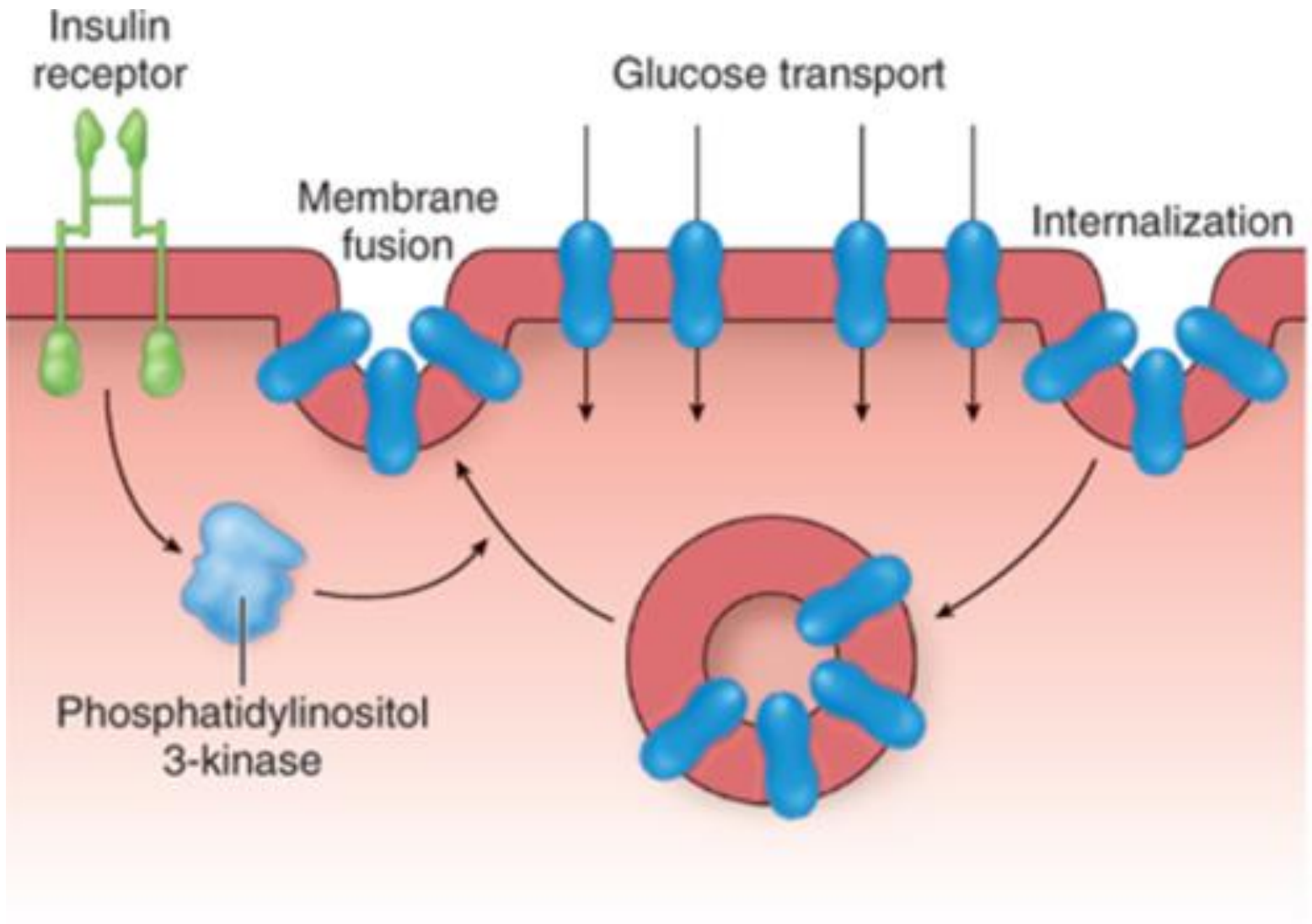


1. No nutrients enter the blood stream from the digestive system
2. Sugar concentrations in blood drop, stimulating the pancreas to stop releasing insulin and instead releases glucagon.
3a. Glyconeolysis releases glucose into the blood to increase glucose levels. Ketone oxidation also releases lipids and additional glucose. (can be uses to generate ATP)
3b. Muscles releases glucose into blood. Catabolized amino acids can be used to generate ATP through ketogenesis and ketone oxidation in the liver.
3c. Adipose cells release stored lipids which can be used to generate glucose, ketones, or ATP.

Control


A Fasting state
Brain
Glucose
<100 mg/dL
(5.6 mM)
Liver
Fatty Acids
400 µM
Skeletal
muscle
Glucagon
Insulin
Insulin
Pancreatic
islet
Insulin
Adipose tissue
B Prandial state
Brain
Glucose
120–140 mg/dL
(6.7–7.8 mM)
Liver
Fatty Acids
<400 µM
Skeletal
muscle
Dietary carbohydrate
Glucagon
Insulin
Insulin
Pancreatic
islet
Insulin
Dietary lipids
Adipose tissue
GI tract

Insulin


Insulin receptor
Glucose transport
Membrane fusion
Internalization
Phosphatidylinositol 3-kinase

Glucose Transporters (GLUT)

Transporter	Found	Function
GLUT-1	Everywhere, highest concentrations on RBC and endothelial tissue, also found on skeletal muscle and adipose tissue	Uptake of glucose by skeletal muscle and adipose tissue
GLUT-2	Pancreatic β-cells, liver, intestine, and kidney	Glucose sensor for endocrine system
GLUT-3	Neurons	Allows glucose to cross BBB
GLUT-4	Predominantly in skeletal muscle and adipose tissue	The predominant transporter of glucose to maintain homeostasis, predominant receptor of insulin
GLUT-5	Sperm and small intestine	Fructose transporter

Storage of Glucose

Glycogenolysis

- Liver and Muscle
- Breakdown of glycogen into glucose
  - Liver for blood levels
  - Muscle for itself

Gluconeogenesis

- Liver and Kidney
- Production of glucose from non-carbohydrates (pyruvate, lactate, glycerol and fatty acids)

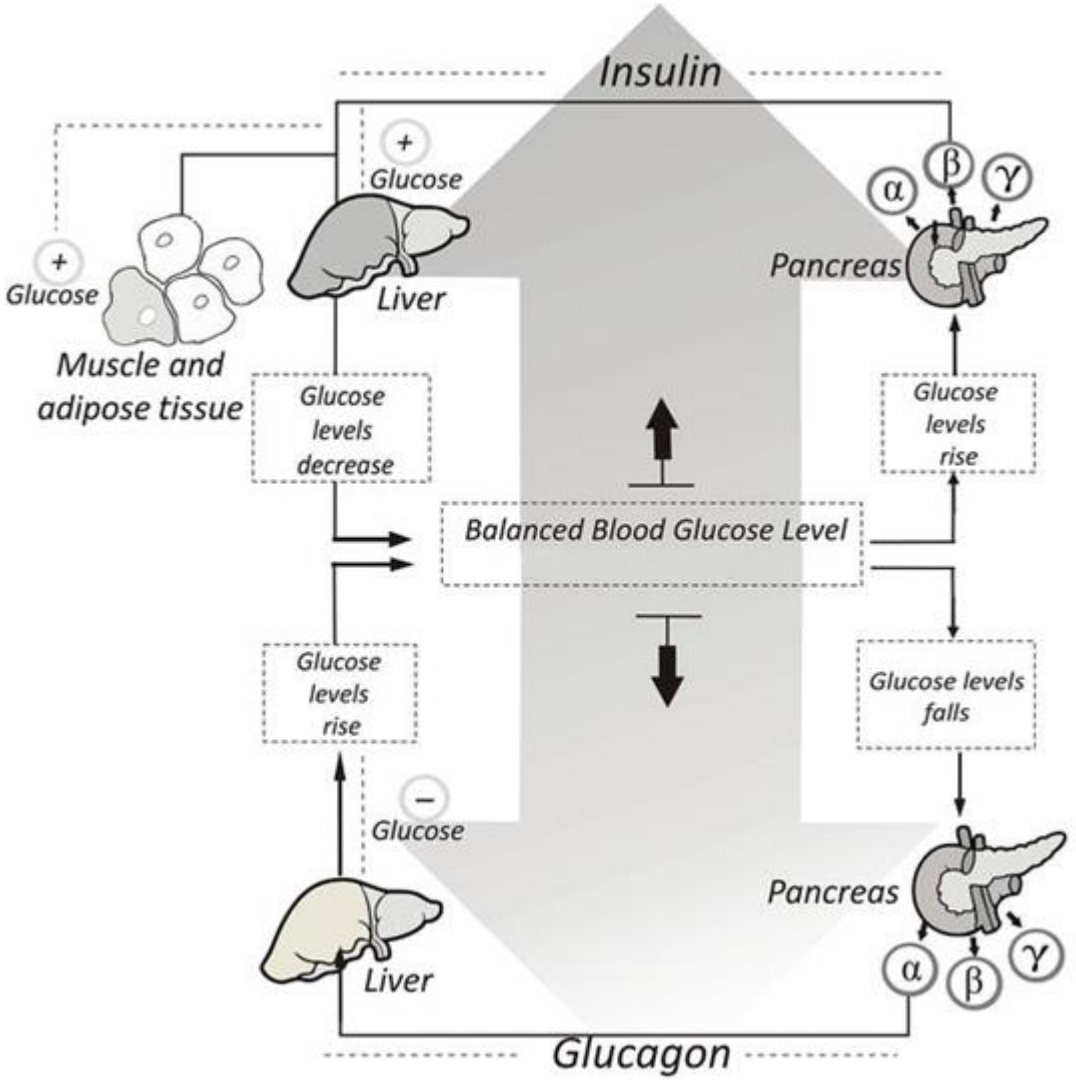


Table 30-2 Summary of Glucose Metabolism

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)

Diabetes Mellitus

- Left untreated
  - Leads to wasting of body tissues
  - Some patients will die relatively young from one or more complications.
  - Severity is related to the average blood glucose level and the onset of diabetes.
  - No cure for the disease
  - Treatment focuses on maintaining blood glucose levels within the normal range.

Diabetes Classifications

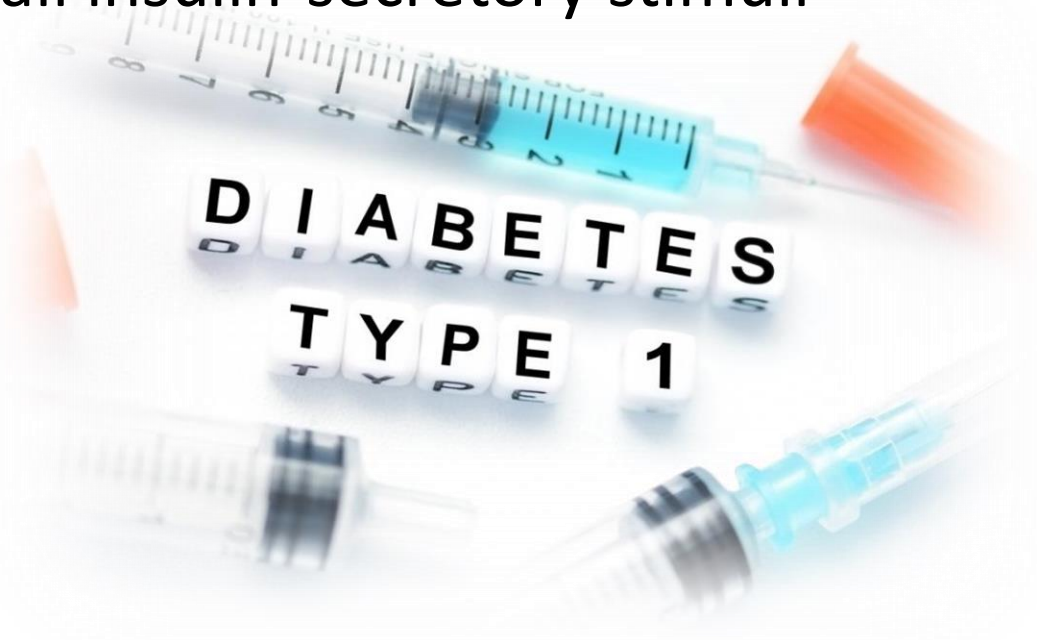
- Diabetes, Type 1
- Diabetes, Type 2
- Maturity Onset Diabetes of the Young (MODY)
- Diabetes insipidus
- Gestational diabetes

Type 1 DM

- 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 DM

- 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
- An autoimmune disease



Type 1 DM

- 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

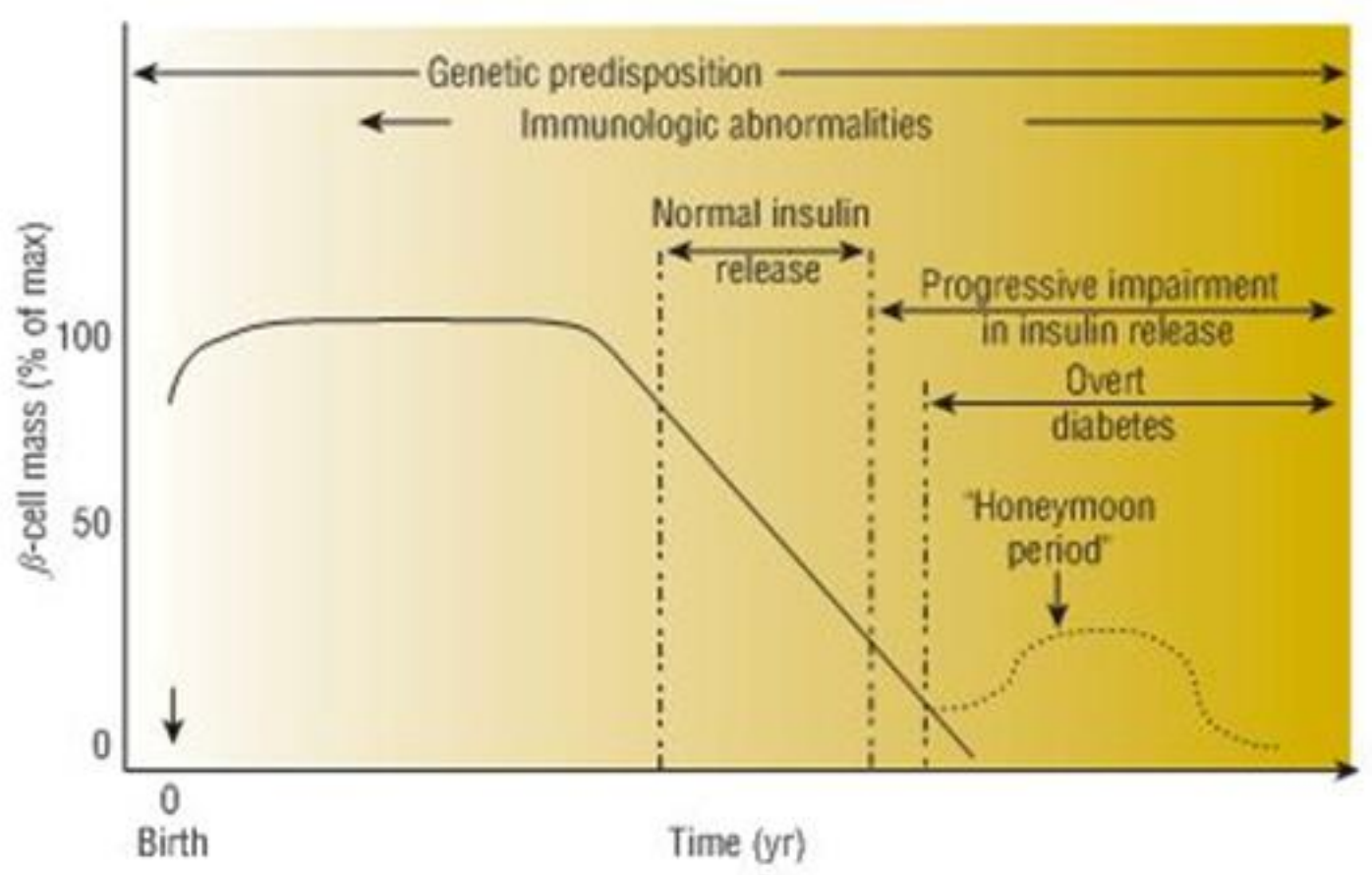
Hallmark Diabetes S/S (The 3 P's)

- Results in pronounced hyperglycemia.

- Also
  - Weight loss
  - Cachexia

Type 1 DM

- Insulin is the cornerstone of therapy



Type 2 DM

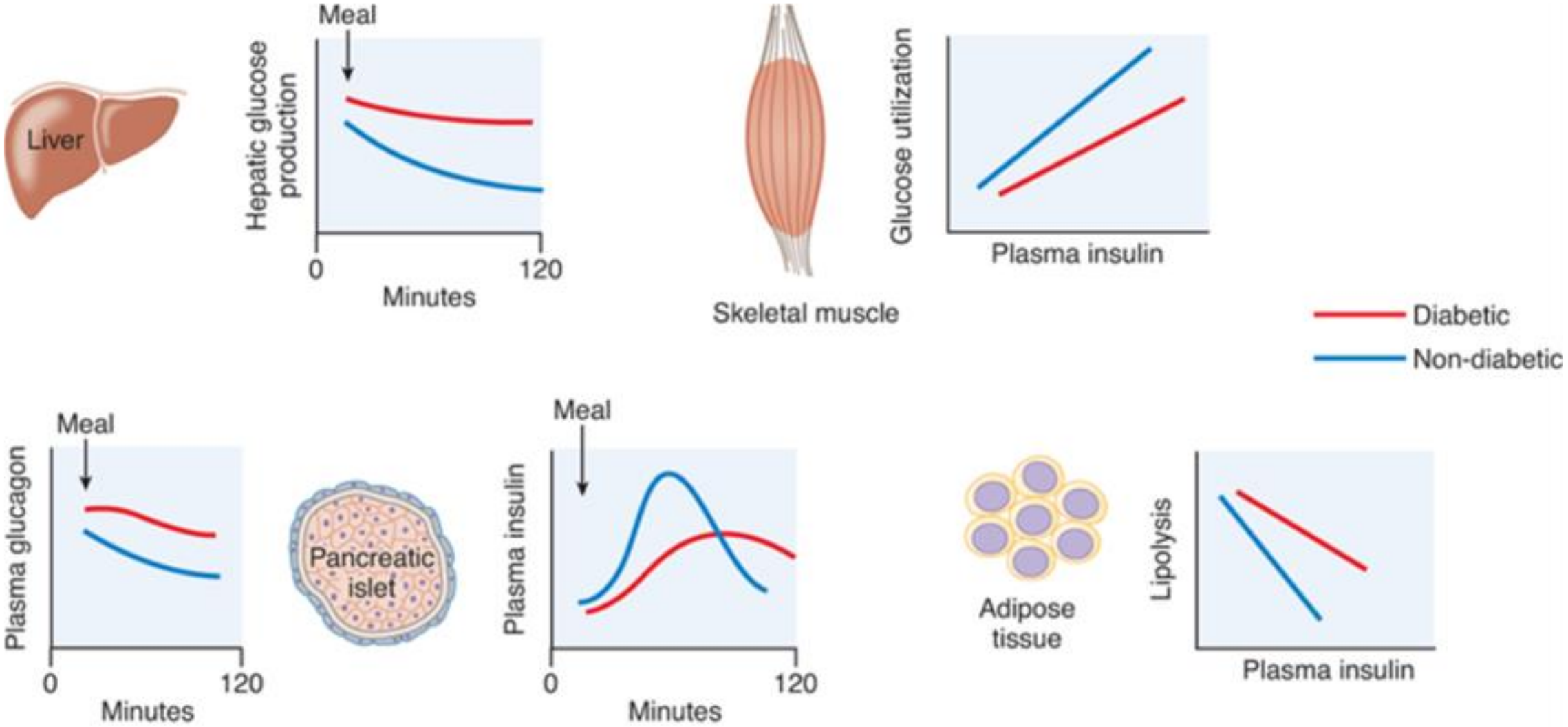
- Characterized by:
  - Decrease in insulin production
  - Diminished tissue sensitivity to insulin
- Results in less-pronounced hyperglycemia
  - Less risk of fat-based metabolism.
  - Managed with dietary and lifestyle changes with oral drugs to stimulate insulin production and increase receptor effectiveness.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

Most common form; most people with diabetes in Canada have type 2 diabetes

Type 2 DM

- 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 2 Diabetes



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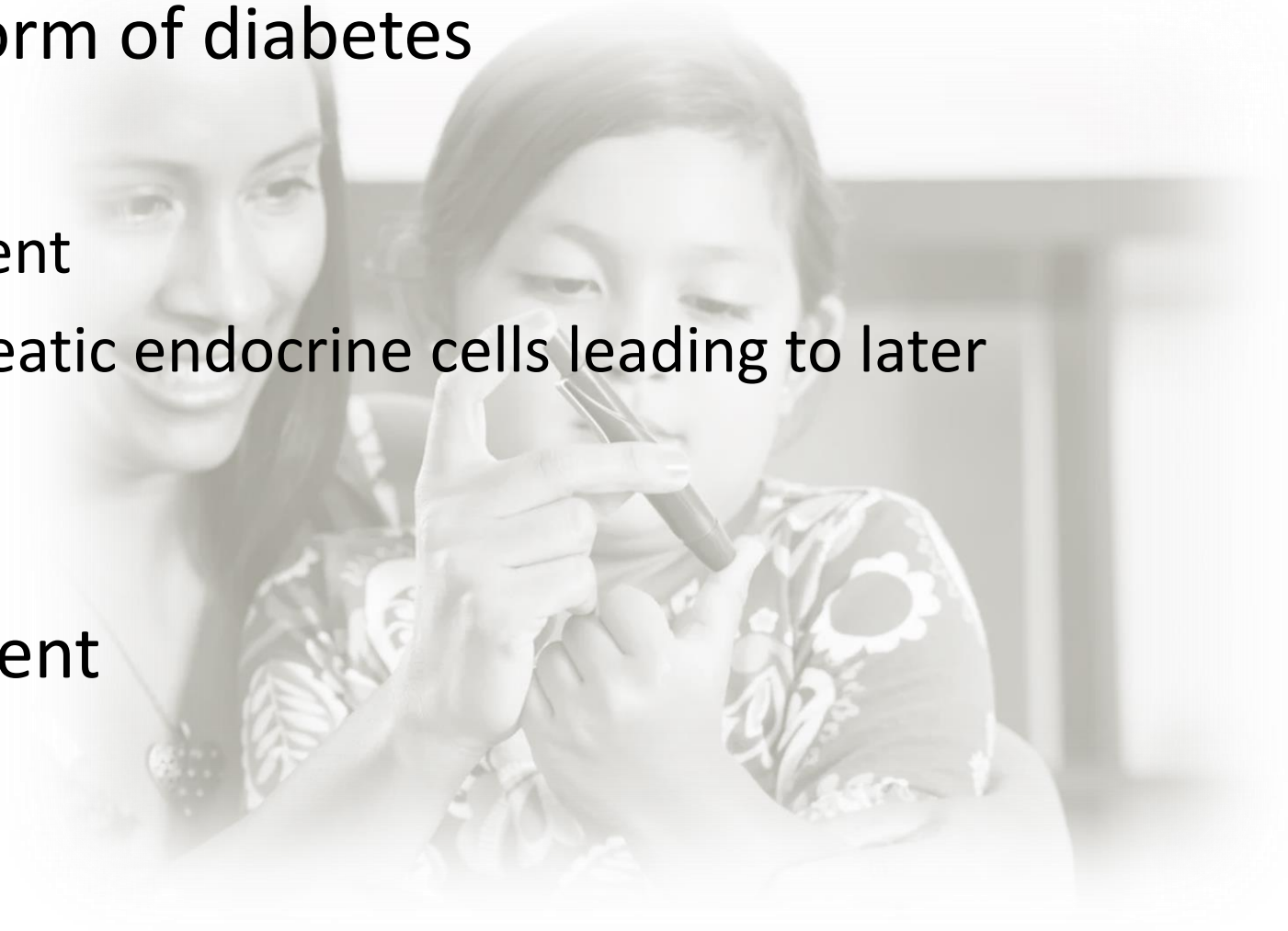
Contrasting Type 1 and Type 2

Characteristic	Type 1 DM	Type 2 DM
Age	< 30 years	> 30 years
Onset	Abrupt	Gradual
Body habitus	Lean	Obese or history of obesity
Insulin resistance	Absent	Present
Autoantibodies	Often present	Rarely present
Symptoms	Symptomatic	Often asymptomatic
Ketones at diagnosis	Present	Absent
Need for inulin therapy	Immediate	Years after diagnosis
Acute complications	DKA	HHNK
Microvascular complications at diagnosis	No	Common
Macrovascular complications at or before diagnosis	Rare	Common

Maturity Onset Diabetes of the Young (MODY)

- A non-insulin-dependent form of diabetes
- Autosomal disease
  - Lacks autoimmune component
  - Slower destruction of pancreatic endocrine cells leading to later onset
- African and Asian ancestry
- Significant genetic component



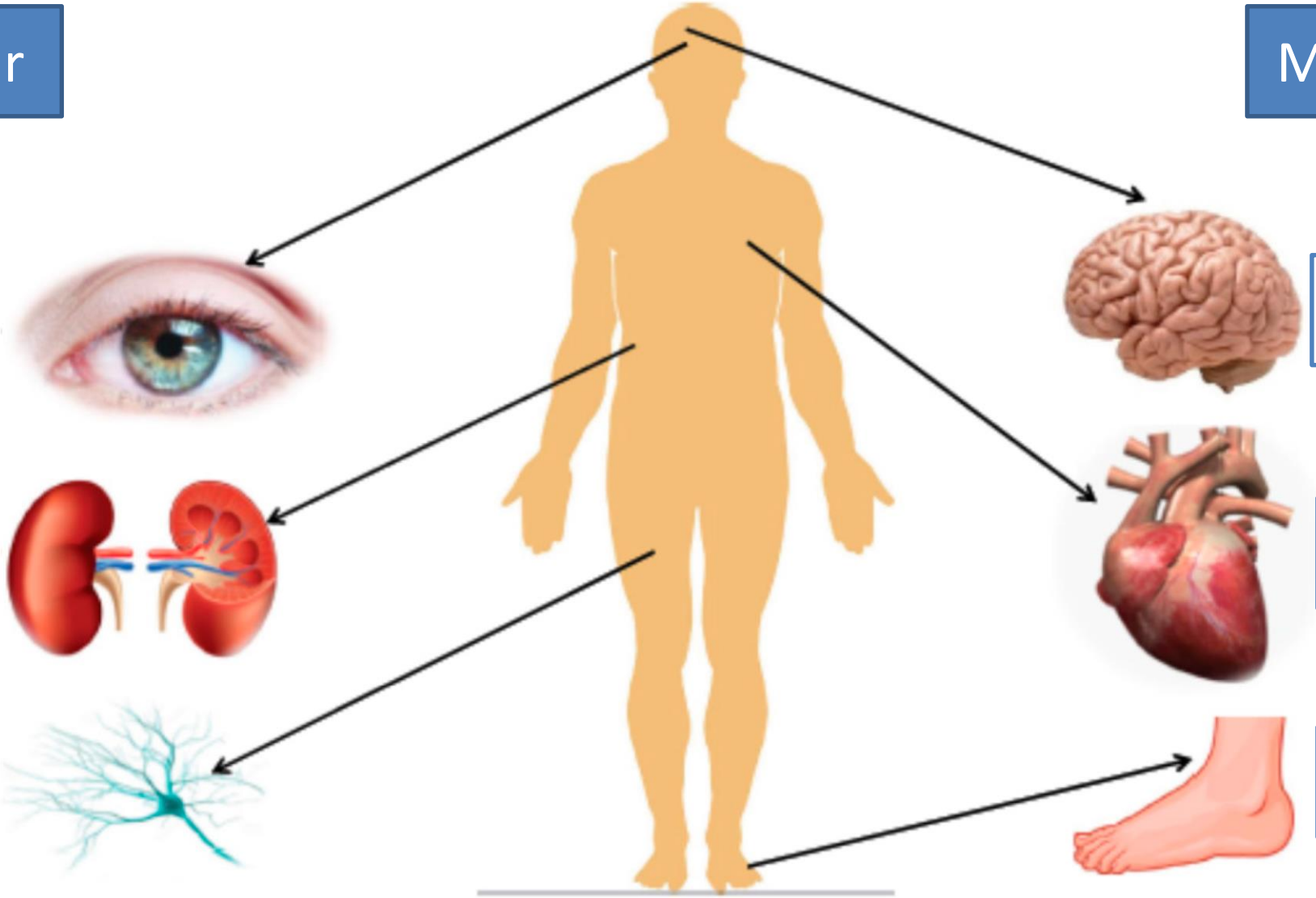
Gestational diabetes

- A transient form of diabetes and occurs in pregnant women as a result of insulin resistance during pregnancy
- Untreated GDM leads to increased maternal and perinatal morbidity

Diabetic Complications

Microvascular

- Retinopathy
- Nephropathy
- Neuropathy



Macrovascular

- Stroke
- Coronary vascular disease
- Peripheral vascular disease

Treatment Plans

- Type 1 Diabetes
  - Requires insulin to sustain life
- Type 2 Diabetes
  - Generally do not require insulin at time of diagnosis
  - Lifestyle modification
  - PO medications

Treatment Plans

- Type 2 diabetes patients and hypoglycemia
  - Less common than with type 1
  - Paramedics should assess and determine association with medications, diseases
  - Medico-legal concern when not transporting

Endocrinology

DIABETIC EMERGENCIES

Emergency Management

- Diabetes
  - Hypoglycemia
  - Hyperglycemia

Hypoglycemia

- Low blood sugar
  - Over administration of insulin or Oral diabetic medications
  - Over activity, stress, alcohol ingestion
  - Rarely seen outside diabetics
- Brain cells
  - Require adequate circulating levels of glucose
  - Rapid cerebral impairment
- By the time signs and symptoms develop, most of the body's stores have been used
- Diabetics with kidney failure are predisposed to hypoglycemia

Hypoglycemia

- BGL <4.0 mmol/L
- Rapid onset

Signs and Symptoms

- Trembling
- Palpitations
- Diaphoresis
- Anxiety
- Hunger
- Nausea
- Tingling

Neurogenic (Autonomic)

- Difficulty concentrating
- Confusion
- Weakness
- Drowsiness
- Vision changes
- Difficult speaking
- Headache
- Dizziness

Neuroglucopenic

Treatment Options

Oral Glucose

IV Dextrose

Glucagon

Oral Glucose

Classification

- Monosaccharide (glucose)

Mechanism of Action

- Provides glucose content for regular cell metabolism
- Usually absorbed through the mucous membranes

Indications

- BGL < 4.0 mmol/L in a conscious patient who can maintain their own airway
- Confusion with recorded BGL < 4.0 mmol/L (An altered level of awareness)

Oral Glucose

Contraindications

- Unable to maintain their own airway
- Altered LOC (Level of consciousness that affects the patient's ability to maintain their own airway)

Dosage

- 1 tube (15 g) orally/buccal PRN

Treatment of Hypoglycemia

- “Evidence suggests that 15 g glucose (monosaccharide) is required to produce an increase in BG of approximately 2.1 mmol/L within 20 minutes, with adequate symptom relief for most people”
- The diabetes drug acarbose will prevent patient from absorbing fructose (juice, fruit etc) and oral glucose will only suffice in hypoglycemia

Dextrose 50% ($D_{50}W$)

Classification

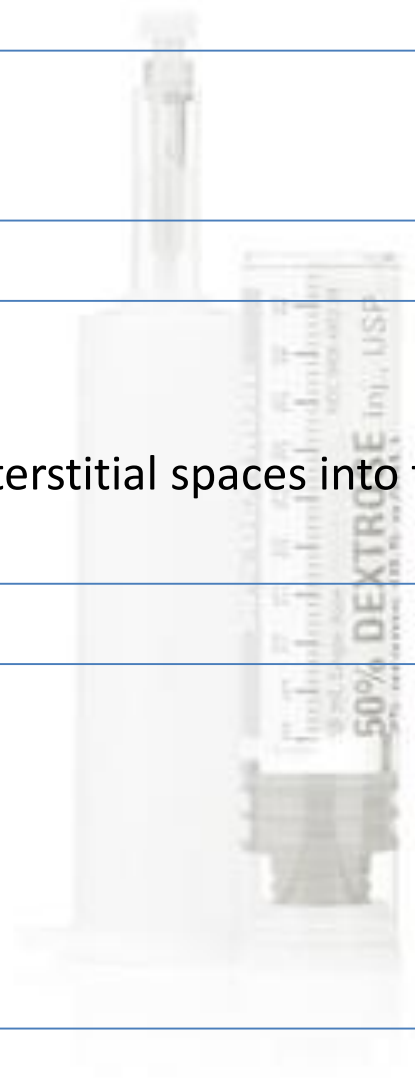
- Carbohydrate

Mechanism of Action

- Increases blood glucose levels
- Hypertonic solution producing a transient movement of water from interstitial spaces into the venous system

Indications

- Hypoglycemia (BGL < 4.0 mmol/L)



Dextrose 50% ($D_{50}W$)

Contraindications

- Unable to maintain their own airway
- Altered LOC (Level of consciousness that affects the patient's ability to maintain their own airway)

Dosage

- 1 tube (15 g) orally/buccal PRN

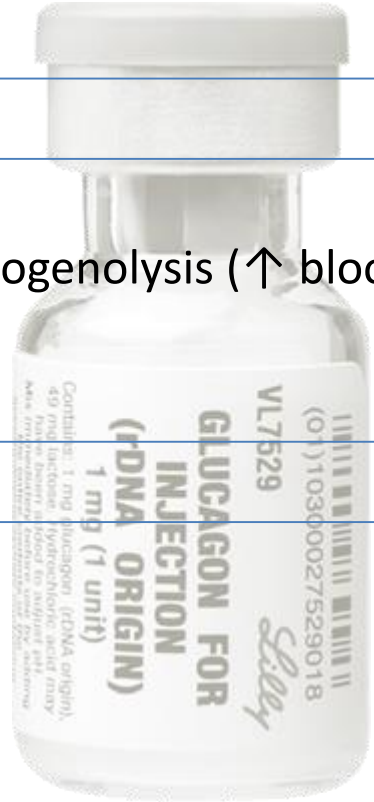
Glucagon

Classification

- Anti-hypoglycemic (Endogenous Pancreatic Hormone)

Mechanism of Action

- Stimulates the release of glycogen (gluconeogenesis) from the liver, for glycogenolysis (↑ blood glucose levels)
- Smooth muscle relaxation



Indications

- Hypoglycemia (BGL < 4.0 mmol/L and unable to take oral glucose or IV D_{50})

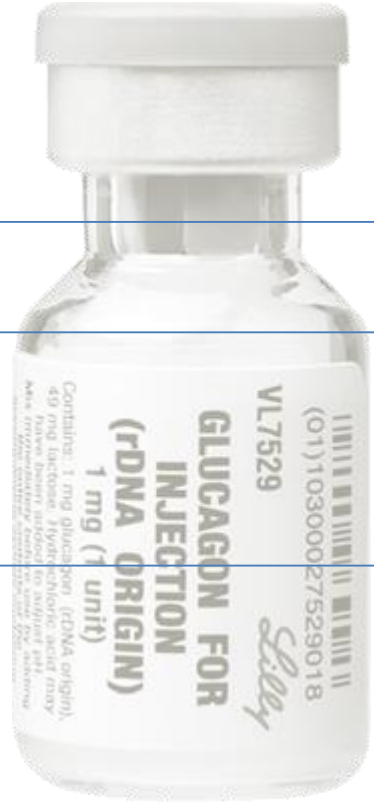
Glucagon

Contraindications

- Hypersensitivity
- Pheochromocytoma

Dosage

- Adult
  - 1.0 mg IM, SQ
  - May repeat q 20 min if required
- Pediatric
  - 0.5 mg IM, SQ (< 20 kg)



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Glucagon Prep

1

2

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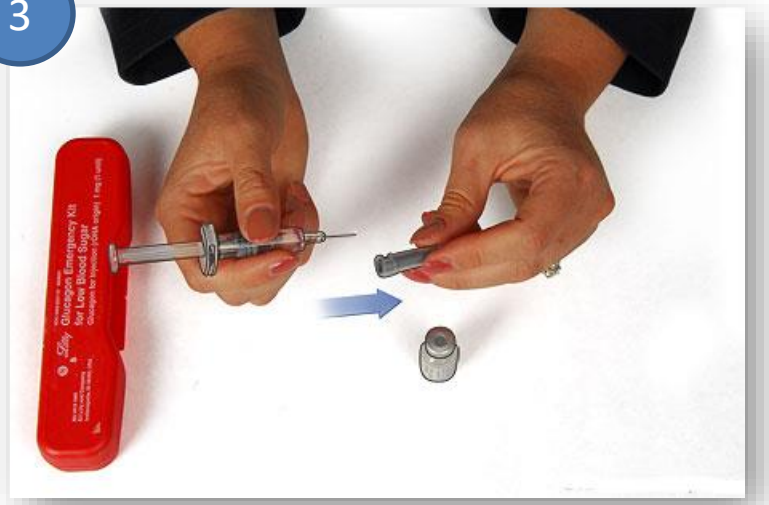

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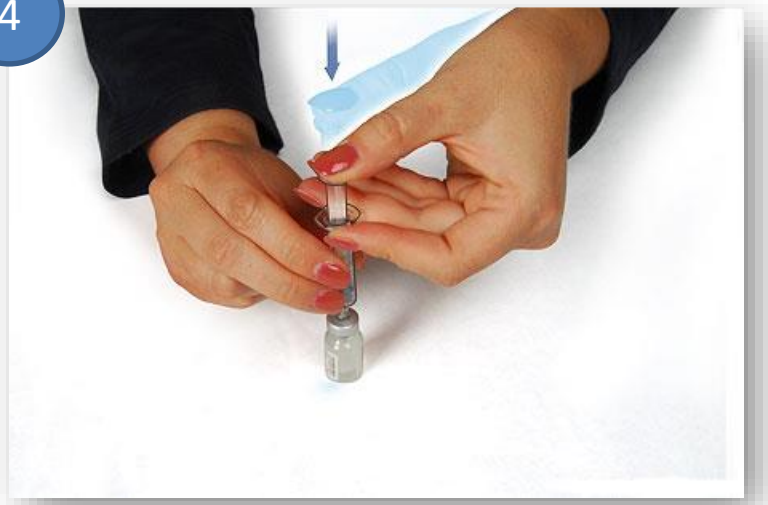

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Glucagon Prep

5

6

7

8

Hyperglycemic Emergencies

- Diabetic Ketoacidosis (DKA)
- Hyperosmolar Hyperglycemic State (HHS)

Hyperglycemia and Diabetic Ketoacidosis

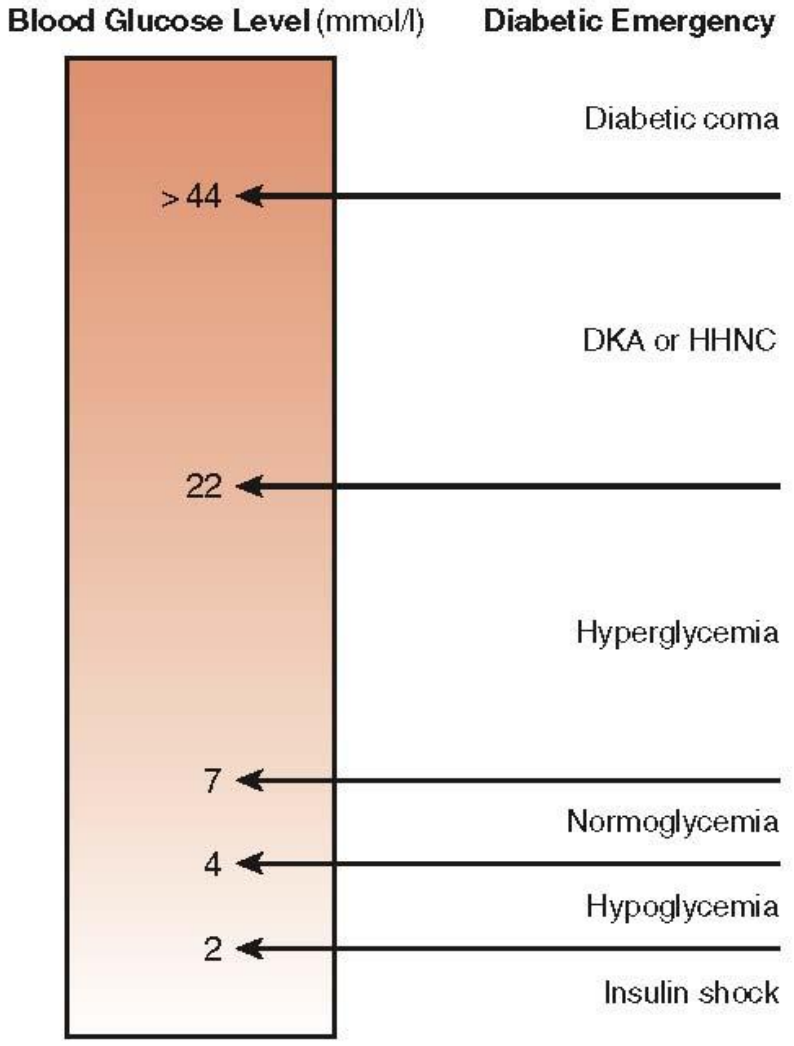
- High blood glucose level is one of the classic symptom of diabetes mellitus.
- Common early signs
  - Frequent and excessive thirst
  - Frequent and excessive urination
- Hyperglycemia is associated with worse neurologic outcome after a stroke.
- Occurs when levels of glucose in the blood exceed normal range
  - Physicians try to keep glucose levels of diabetic patients at less than 8 mmol/L.

Hyperglycemia and Diabetic Ketoacidosis

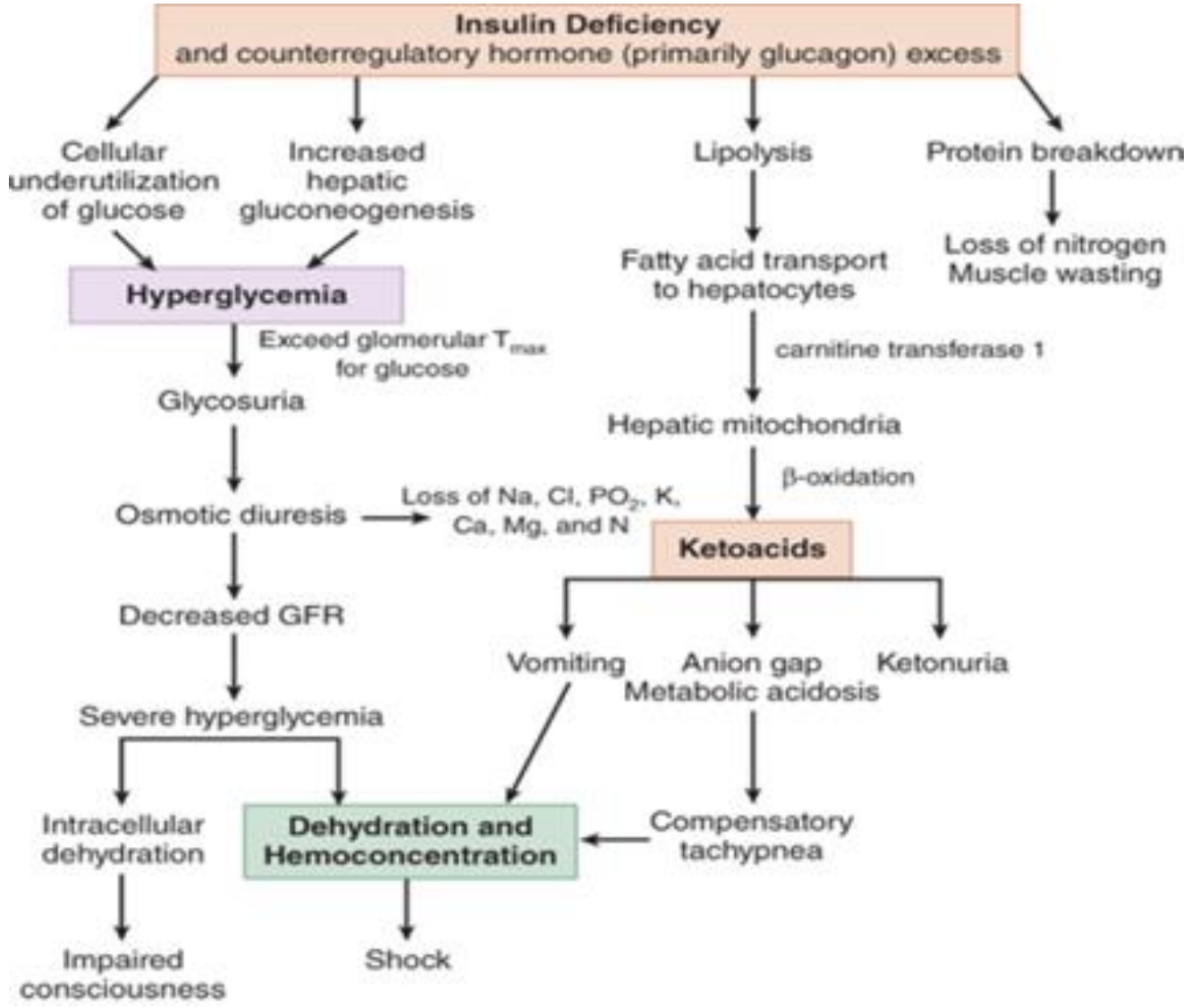
- Onset may be rapid or gradual, depending on the cause.
- When serum glucose levels rise above tolerable levels, other physiologic changes occur.
- These changes represent actual pathologic conditions called:
  - Diabetic ketoacidosis (DKA)
    - BGL > approximately 18 mmol/L
  - Hyperosmolar hyperglycemic syndrome (HHS)
    - BGL > approximately 33 mmol/L

Hyperglycemia and Diabetic Ketoacidosis

- If left untreated, hyperglycemia will progress to DKA.
  - Life-threatening condition
  - Certain acids accumulate in the body.
  - Patients tend to be young.
  - Deficiency of insulin prevents cells from taking up the extra glucose.
  - Metabolism of fat generates acids and ketones as waste products.
  - Patient undergoes massive osmotic diuresis.



DKA Pathophysiology

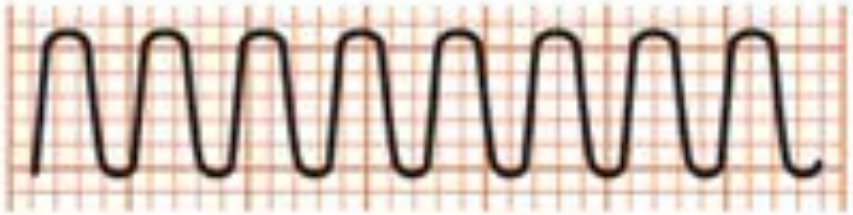
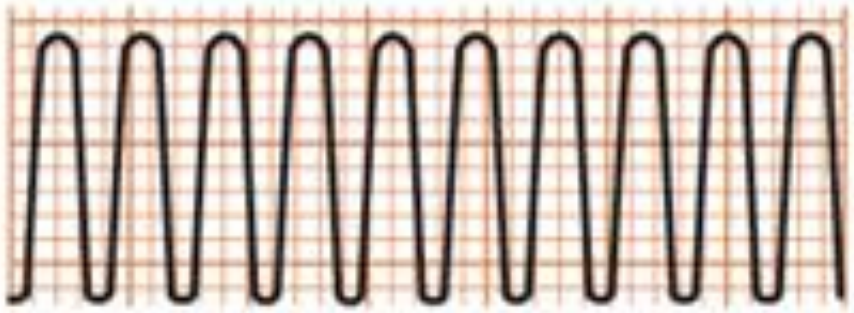


- BGL > 18.0 mmol/L
- Gradual onset

Signs and Symptoms

- Polyuria
- Polydipsia
- Polyphagia
- Nausea and vomiting
- Tachycardia
- ALOC (Coma)
- Anorexia
- Kussmaul's respiration and acetone breath
- Volume depletion can lead to:
  - Dry mucous membranes
  - Tachycardia
  - Hypotension
- Abdominal Pain
- Hypotension

Kussmaul's Respirations

Type of respiration	Diagram	Discussion
Normal		16–20/min; regular in rhythm; ratio of respiratory rate to pulse rate is 1:4
Kussmaul's respiration		Increase in both rate and depth. Associated with diabetic ketoacidosis

DKA management

- The order of therapeutic priorities is **volume first and foremost**, correction of potassium deficits, and then insulin administration to rectify acidosis
  - Difficult to do on ground ambulance
  - Correcting DKA is a lengthy and heavily monitored process
    - 24-36 hours
- Treat volume, monitor EKG (watch for K^+ disturbances), monitor BGL
- In individuals with DKA, IV 0.9% sodium chloride should be administered initially at 500 mL/h for 4 hours, then 250 mL/h for 4 hours (Diabetes Canada guidelines)
  - With consideration of a higher initial rate (1–2 L/h) in the presence of shock states

Hyperosmolar Hyperglycemic Syndrome (HHS) Pathophysiology

- Also called hyperosmolar nonketotic coma (HONK)
- Metabolic derangement principally in patients with type 2 diabetes
- Characterized by
  - Hyperglycemia
  - Hyperosmolarity
  - Absence of significant ketosis

Coma in fewer than 10% of cases

Acute MI is frequently associated

Often develops in patients with diabetes

Infection is the most common cause.

- BGL > 33.0 mmol/L
- Gradual onset

Signs and Symptoms

- Polyuria
- Volume depletion
- Poor turgor
- Dry mucus membranes
- Weight loss
- Confusion, lethargy

- **No ketones**
- Physical examination
- Orthostatic fall in blood pressure and rise in pulse, supine tachycardia

Management

- Follows the pathway for dehydration and altered mental status
- Airway management is the top priority.
- Fluid resuscitation with 0.9% normal saline at a rate of 15 to 20 mL/kg/h during the first hour, followed by rates from 4 to 14 mL/kg/h
- Monitor EKG (watch for K^+ disturbances)
- Monitor BGL
- Patient will need insulin

Patient Monitoring

- Self monitoring of blood glucose (SMBG)
  - Individualized
  - Depends on if targets being met
- Treatment with insulin
  - SMBG, 4-5x/day when giving 3-4 injections/day
  - SMBG, 1-2x/day when gibing 3-4 injections/day
- Treatment with oral antihyperglycemics
  - Possibly weekly once stabilized
  - More often when initially diagnosed
- During periods of rapid change in BG levels (e.g. after meals, after exercise and during hypoglycemia), fingertip testing has been shown to more accurately reflect glycemic status than forearm or thigh testing

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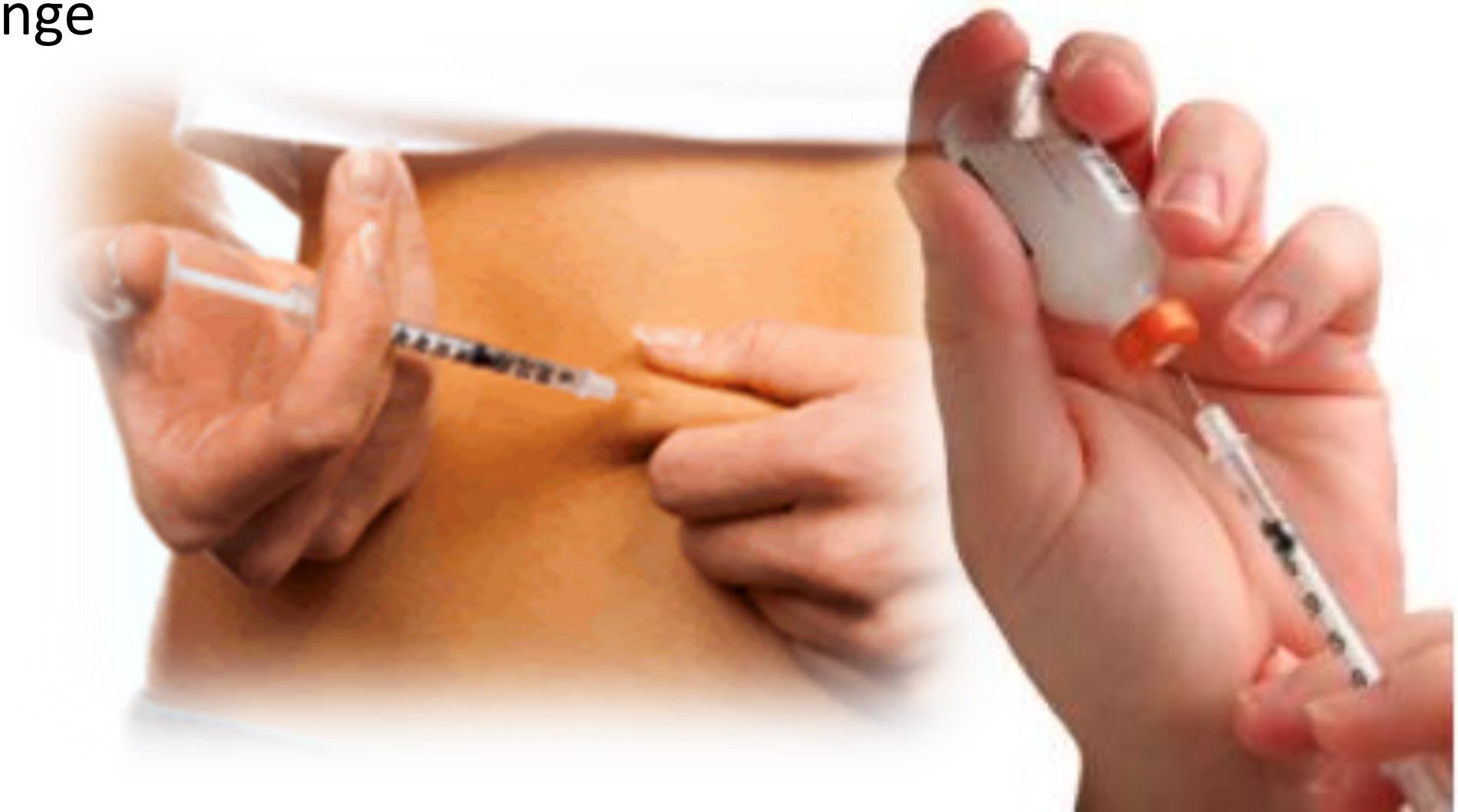
Insulin Therapy

Insulin Type (trade name)	Onset	Peak	Duration
Bolus (prandial) Insulins			
Rapid-acting insulin analogues (clear): • Insulin aspart (NovoRapid®) • Insulin glulisine (Apidra™) • Insulin lispro (Humalog®) • Insulin lispro U200 (Humalog® 200 units/mL)	 10 - 15 min 10 - 15 min 10 - 15 min 10 - 15 min	 1 - 1.5 h 1 - 1.5 h 1 - 2 h 1 - 2 h	 3 - 5 h 3 - 5 h 3.5 - 4.75 h 3.5 - 4.75 h
Short-acting insulins (clear): • Insulin regular (Humulin®-R) • Insulin regular (Novolin®geToronto)	30 min	2 - 3 h	6.5 h
Basal Insulins			
Intermediate-acting insulins (cloudy): • Insulin NPH (Humulin®-N) • Insulin NPH (Novolin®ge NPH)	1 - 3 h	5 - 8 h	Up to 18 h
Long-acting basal insulin analogues (clear) • Insulin detemir (Levemir®) • Insulin glargine (Lantus®) • Insulin glargine U300 (Toujeo®) • Insulin glargine (Basaglar™)	 90 min 90 min Up to 6 h 90 min	Not applicable	 Up to 24 h (detemir 16-24 h) Up to 24 h (glargine 24 h) Up to 30 h Up to 24 h (glargine 24 h)

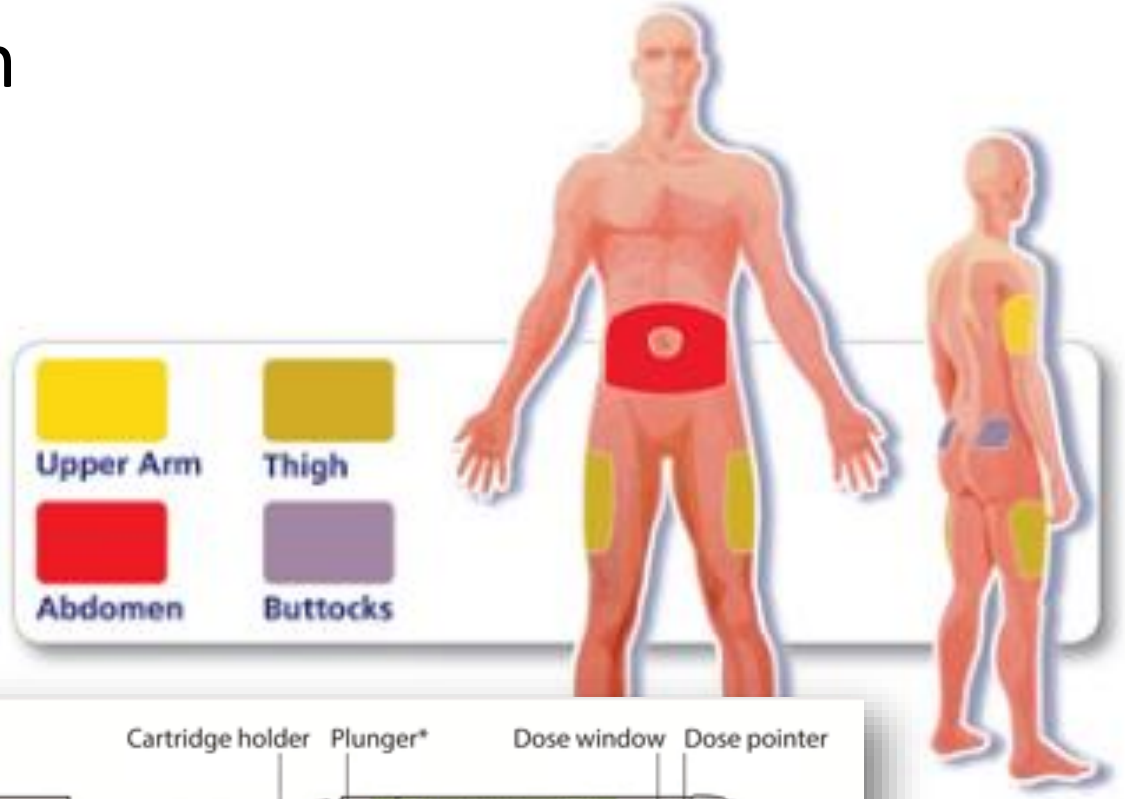
Insulin Administration

- Syringe



Insulin Administration

- Pen



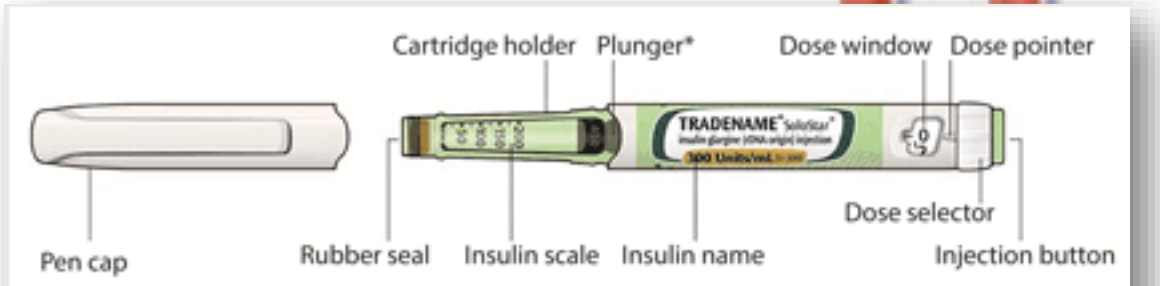
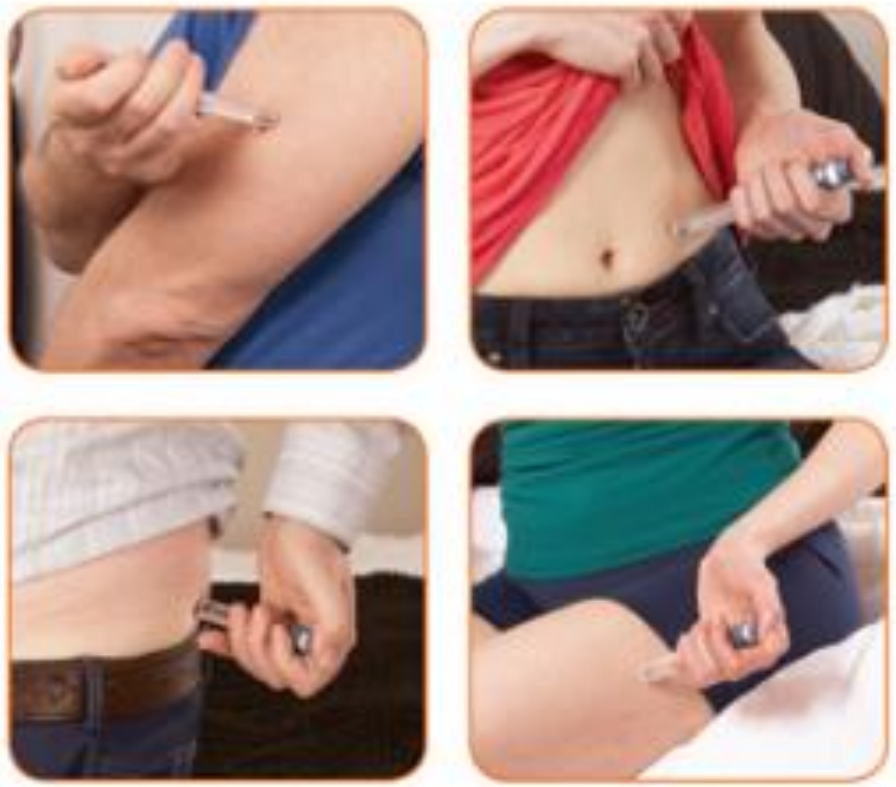


Figure 1 : TOUJEO SoloSTAR Pre-Filled Pen



Insulin Administration

- Pump

Pump Emergencies

- If patient is hypoglycemic, do not discontinue the pump
  - diabetic ketoacidosis can rapidly develop.
- If a patient on an insulin pump is to be made NPO, the insulin pump should stay in place and remain on
  - glucose levels should be checked every 30 to 60 minutes. If the patient has hypoglycemic episodes while NPO, the pump basal rate can be reduced
- If patient is hyperglycemic due to inadequate daily BGL management, pt can bolus themselves with “correction”
- If pt is DKA, pump should be suspended, pt will need IV insulin and further treatment for DKA

Health Promotion and Diabetes

- BP control
  - Target <130/80
- Smoking cessation
- Weight control
  - Proper nutrition
- Exercise
- Foot care
- Blood glucose monitoring
- Testing for micro/macrovascular complications
- Discuss signs and symptoms of hypoglycemia and hyperglycemia

Health Promotion and Diabetes

- Alcohol ingestion may mask the symptoms of hypoglycemia, reduce hepatic production of glucose and increase ketones.
- Moderate alcohol consumption (6 to 18 g/day) is associated with a 25% to 66% lower risk of total and fatal CHD in persons with type 2 diabetes

American's..

- You respond to Pearson International Airport for an unknown patient having a "diabetic emergency"
  - On arrival BGL reading 38 mg/dl and patient acting very inappropriate?
    - Thoughts?

Conversion

- Divide by 18 (mg/dL to mmol/L)

1 mmol/L = 18 mg/dL

Example 1

$$\frac{180\,\frac{\text{mg}}{\text{dL}}}{18} = 10\,\frac{\text{mmol}}{\text{L}}$$

Example 2

$$\frac{38\ mg/dL}{18} = 2.1\ mmol/L$$

Hi vs Low

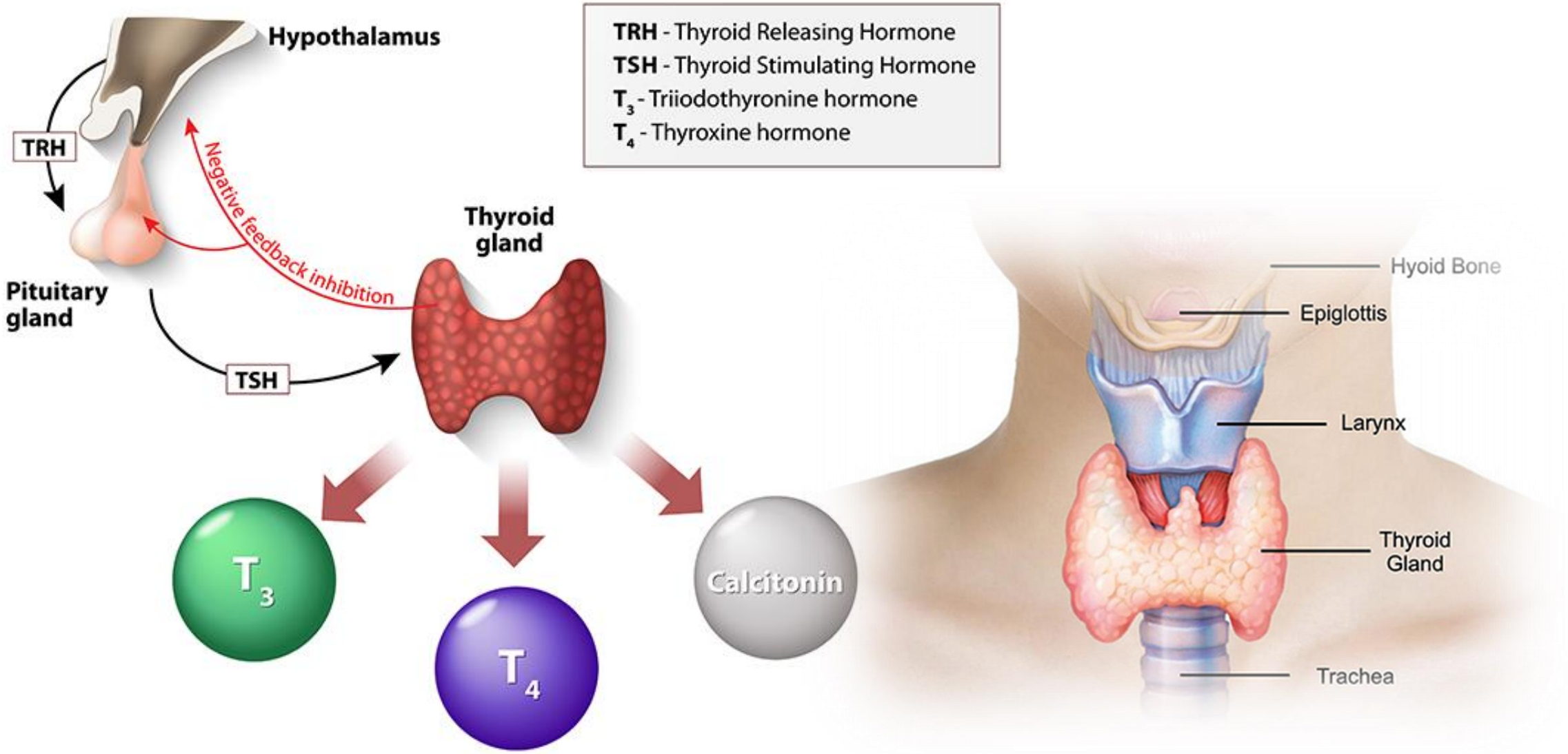
Table 30-4 Diagnostic Signs by System for Diabetic Emergencies

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)

Endocrinology

DISORDER OF THE THYROID GLAND

Thyroid Gland

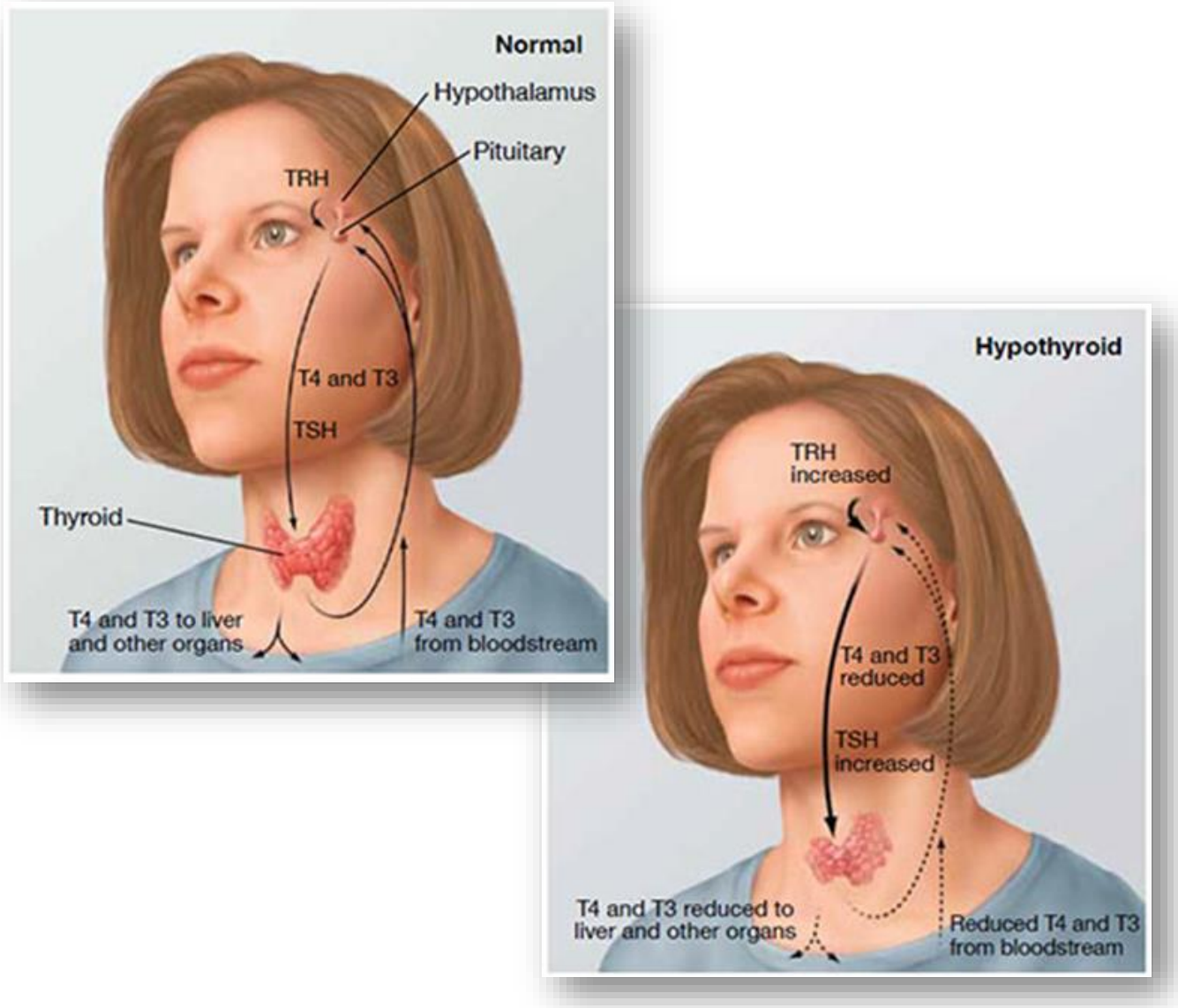


Thyroid Pathophysiology

- Primary
  - Dysfunction of the thyroid gland
- Secondary
  - Deficiency in TSH or Thyrotropin-releasing hormone (pituitary or hypothalamus)

Hypothyroid

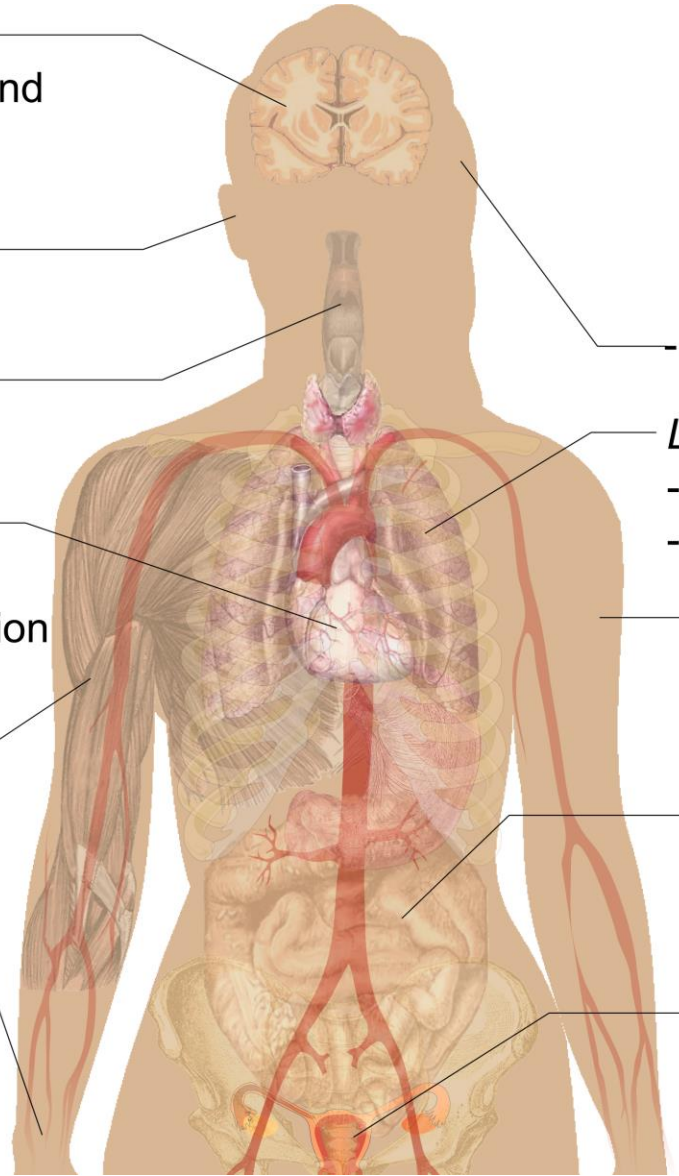
- Slow metabolic rate due to alteration of glandular function



Hypothyroidism

Primary	Secondary
Hashimoto's	Pituitary tumour
Iodine deficiency	Hypothalamic tumour
Thyroiditis	Lack of TSH secretion
Drugs	
Idiopathic	

Hypothyroidism
Signs and Symptoms



Psychological
- Poor memory and concentration

- Poor hearing

Pharynx
- Hoarseness

Heart
- Slow pulse rate
- Pericardial effusion

Muscular
- Delayed reflex relaxation

Extremities
- Coldness
- Carpal tunnel syndrome

General
- Fatigue
- Feeling cold
- Weight gain with poor appetite

- Hair loss

Lungs
- Shortness of breath
- Pleural effusion

Skin
- Paresthesia
- Myxedema

Intestines
- Constipation
- Ascites

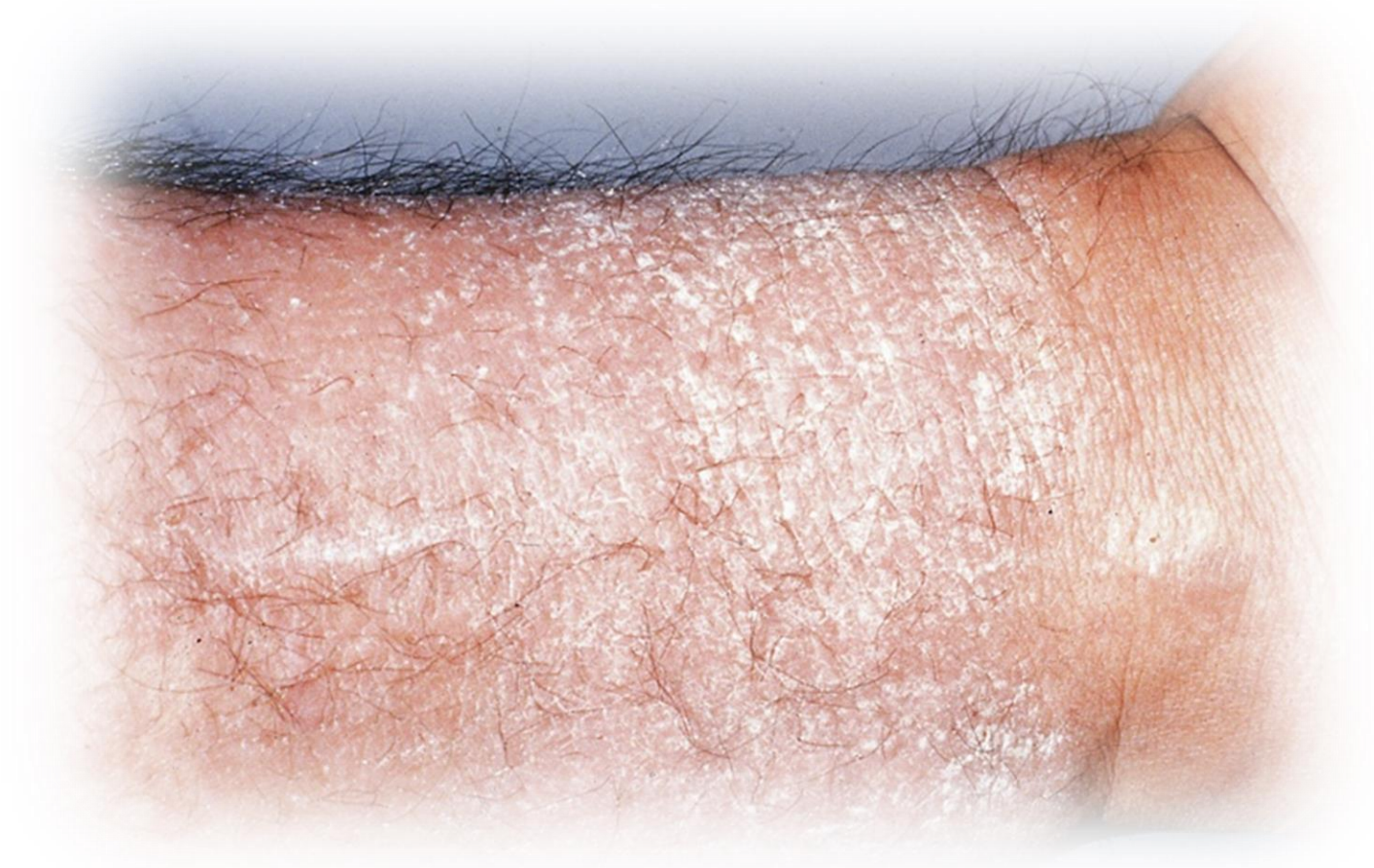
Reproductive system
- Menorrhagia

Late

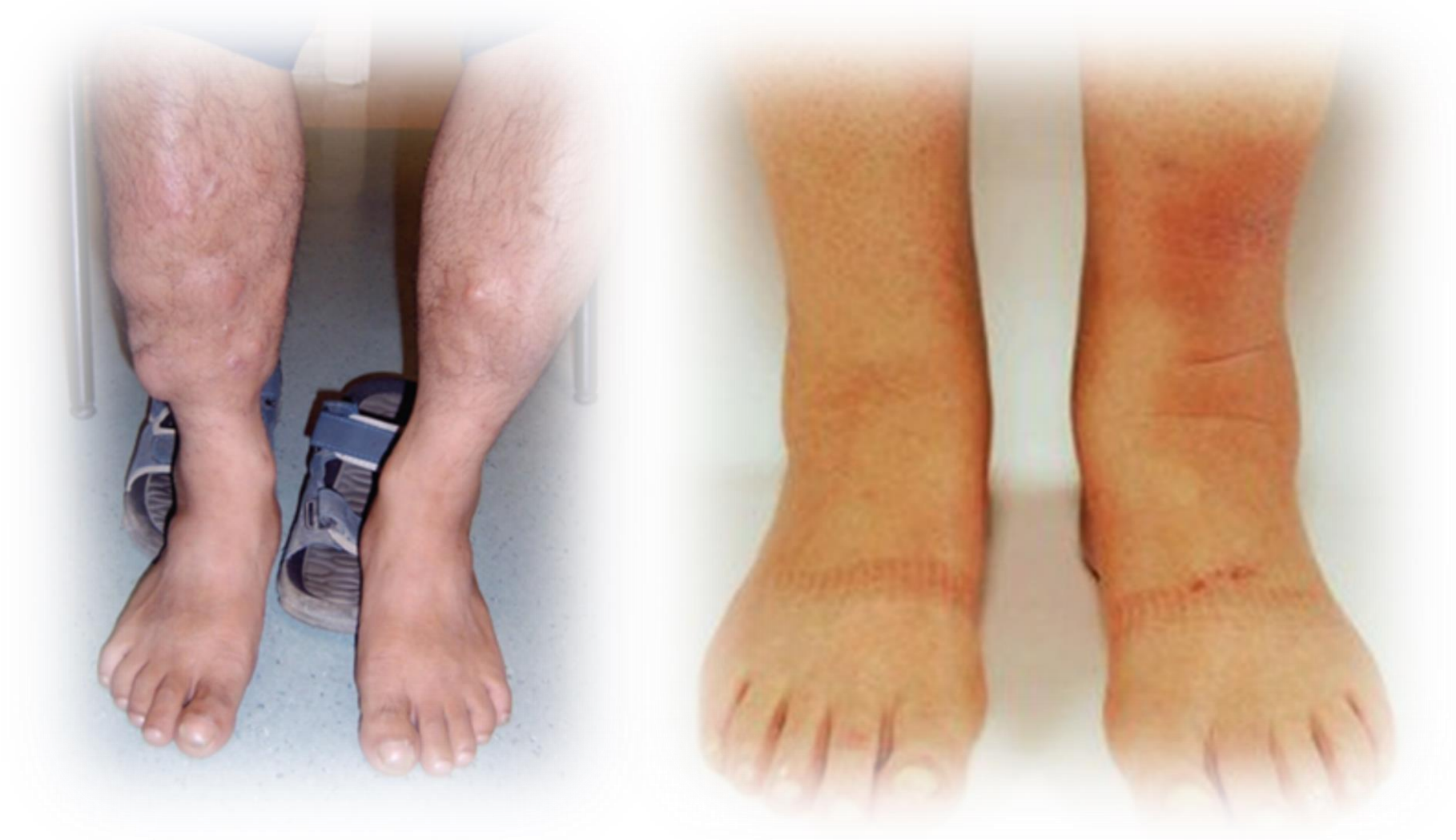
- Myxedema coma
- Decrease HR & SV
- Hypothermia
- Confusion
- Stupor
- Coma
- Carbon dioxide retention
- Hypoglycemia
- Hyponatremia
- Bowel obstruction

Myxedema

- Doughy, edematous skin classic of myxedema

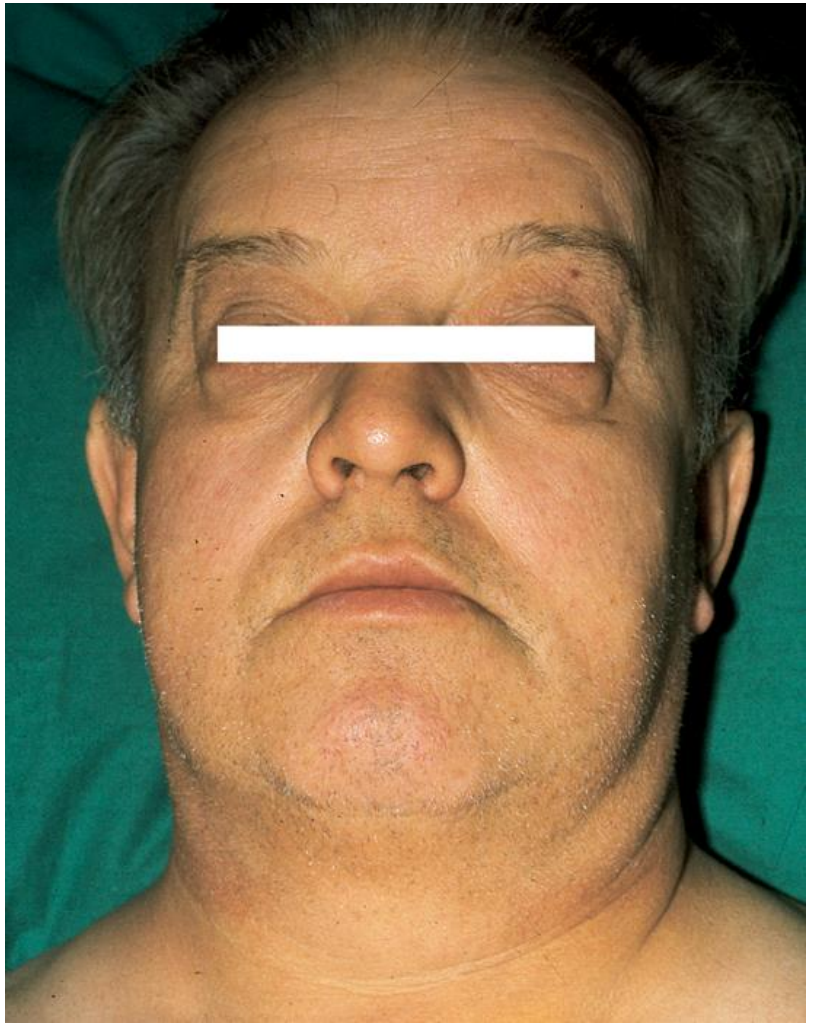


Pretibial Myxedema



Hypothyroidism
Myxedema Coma

- Myxedema coma
  - If thyroid hormones become inadequate
    - Organ tissues do not grow or mature.
    - Energy production declines.
    - Actions of other hormones are affected.
- Severe hypothyroidism (Myxedema crisis)
- Rare life-threatening clinical condition that represents severe hypothyroidism with physiological decompensation (Mortality up to 75%)



Hypothyroidism
Myxedema Coma

- Manifests by a general slowing of the body's metabolic process
- Symptoms include:
  - Fatigue
  - Feeling cold
  - Weight gain
  - Dry skin
  - Sleepiness

Hypothyroidism
Myxedema Coma

- Triggers
  - Infection
  - Exposure to cold
  - Trauma
  - Surgery
  - Certain medications
- Hallmark is deterioration of the patient's mental status.
- Metabolic and cardiovascular emergency
- Administer supplemental oxygen therapy.
- Monitor cardiac status.
- Treat hypothermia with passive rewarming methods.

Management

- Consider
  - Assisted ventilation
  - Monitor BGL frequently and treat hypoglycemia
  - Restriction of fluids
  - Avoid narcotic medications as they can exacerbate condition

Hypothyroidism

- Chronic management
  - Synthroid (levothyroxine T4)
    - Very common medication you will encounter
    - Good to be familiar with it and some questions to ask
    - 20% of adverse drug reactions due to over treatment
      - Standard dose 12.5-25 mcg for elderly
      - 50 mcg for those under 50
      - Dose should be taken the same time each day spaced out from meals and other medications as it interacts with many drugs
      - Bloodwork q 6-8 wks until stable and then q 6-12 months
      - What questions would you want to ask a patient on this medication?

Hyperthyroidism

- Overactive thyroid
- A condition in which your thyroid gland produces too much of the hormone thyroxine.
- Hyperthyroidism can accelerate your body's metabolism significantly, causing sudden weight loss, a rapid or irregular heartbeat, sweating, and nervousness or irritability

Hyperthyroidism

Signs and Symptoms

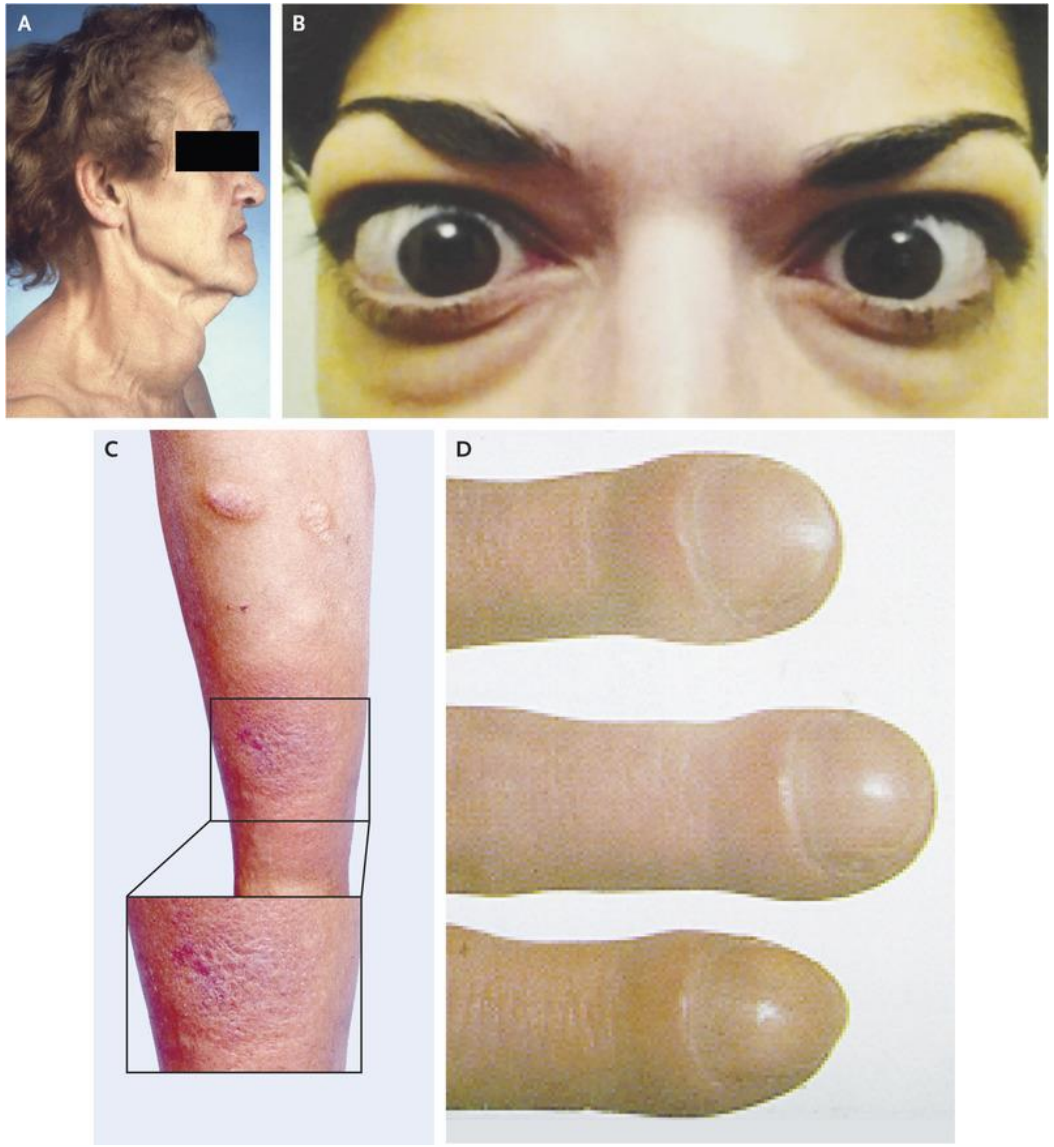
- Early
  - Insomnia
  - Proximal muscle weakness
  - Frequent bowel movements
  - Amenorrhea
  - Emotional lability
- Late or Acute
  - Tachycardia
  - Pulmonary edema
  - HTN
  - Shock
  - Tremor
  - Confusion
  - Psychosis
  - Diarrhea
  - Liver enlargement,
    - Jaundice (↑bilirubin)
  - Hyperglycemia

Hypothyroidism and Hyperthyroidism

Table 32-5 Comparison of Major Effects of Hypothyroidism and Hyperthyroidism

	Hypothyroidism	Hyperthyroidism
Cardiovascular effects	Slow pulse, reduced cardiac output	Rapid pulse, increased cardiac output
Metabolic effects	Decreased metabolism, cold skin, weight gain	Increased metabolism, skin hot and flushed, weight loss
Neuromuscular effects	Weakness, sluggish reflexes	Tremor, hyperactive reflexes
Mental, emotional effects	Mental processes sluggish, personality placid	Restlessness, irritability, emotional lability
Gastrointestinal effects	Constipated	Diarrhea
General somatic effects	Cold, dry skin	Warm, moist skin

Grave's Disease

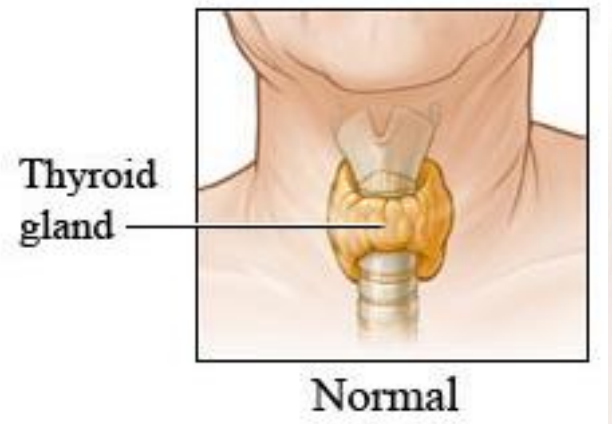


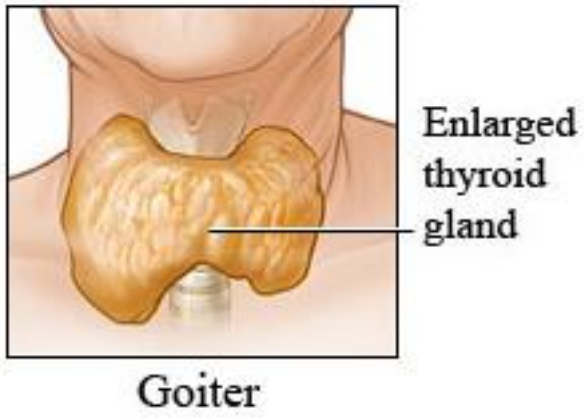
- Autoantibodies are generated that stimulate thyroid tissue to produce excessive hormone
  - Autoimmune response
  - Most common cause of hyperthyroidism

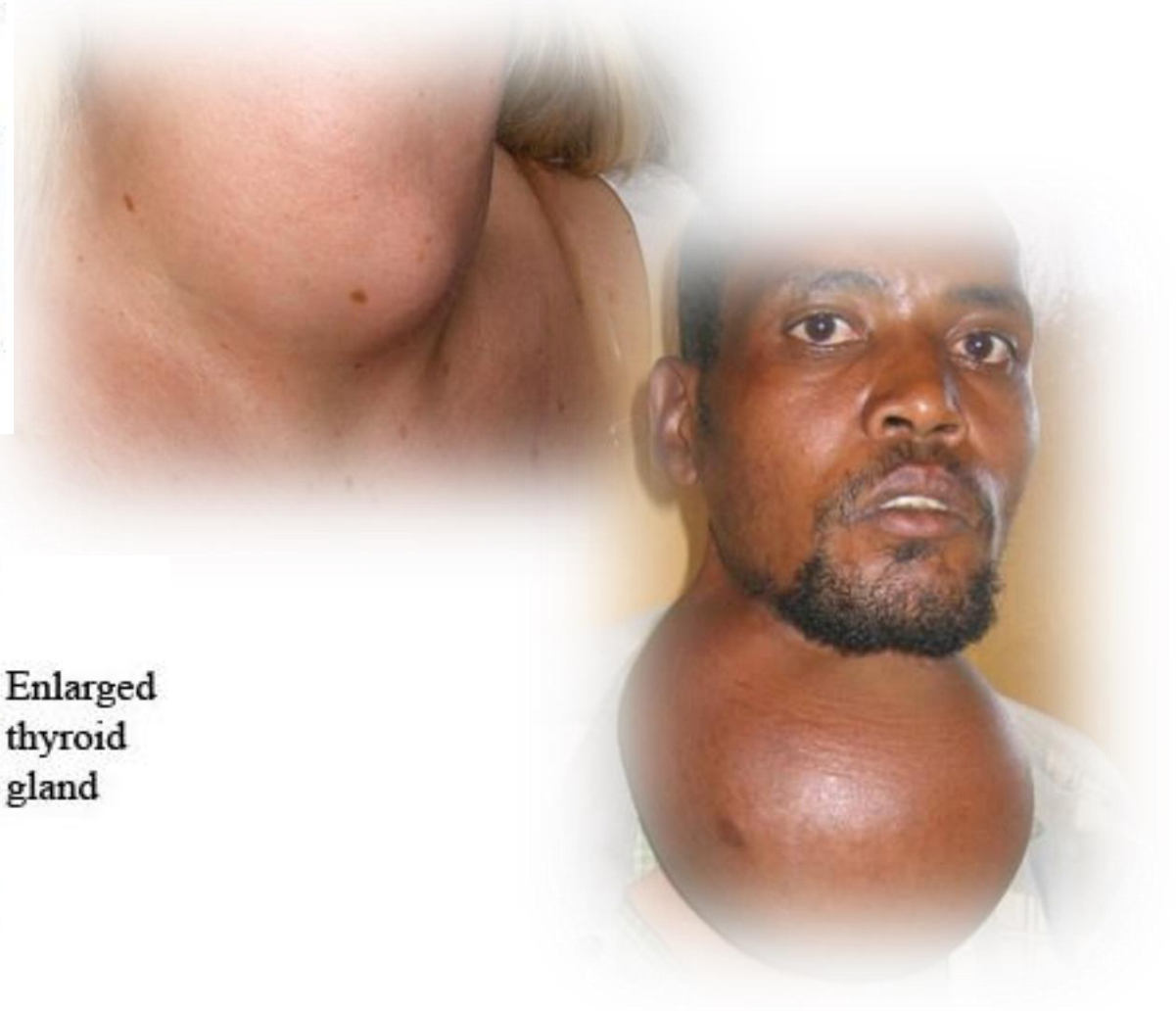
Grave's Disease

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

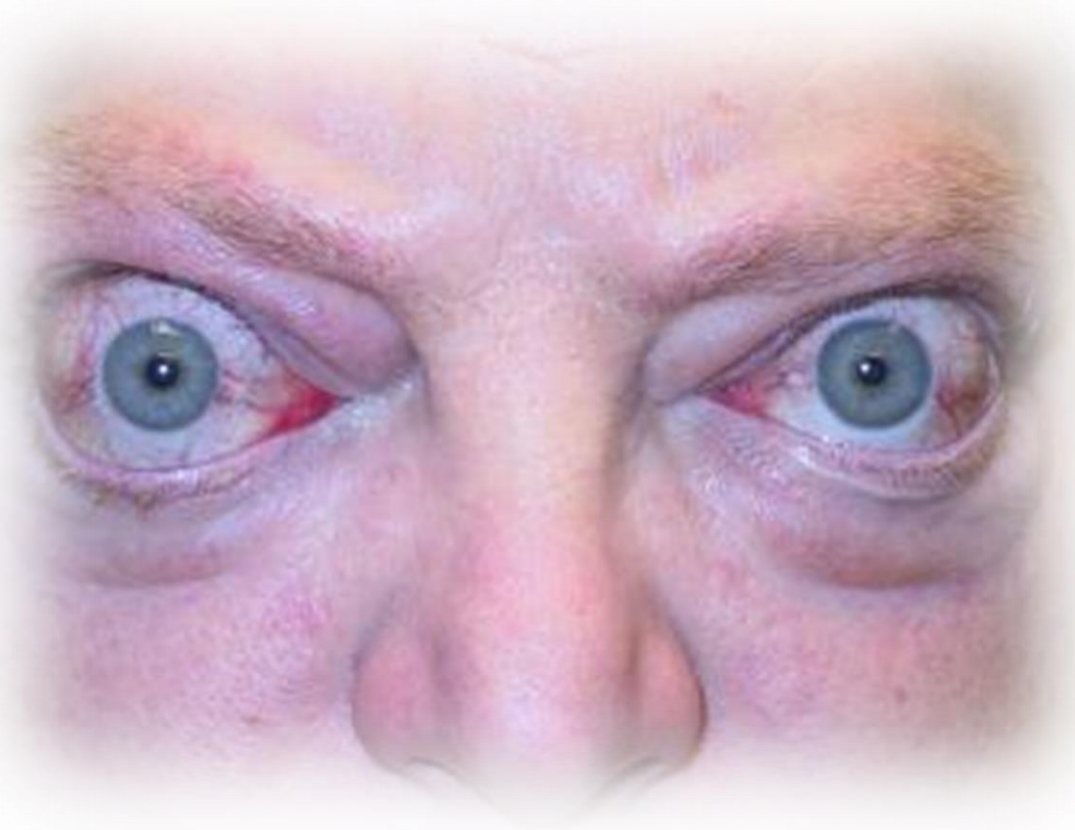
Goiter

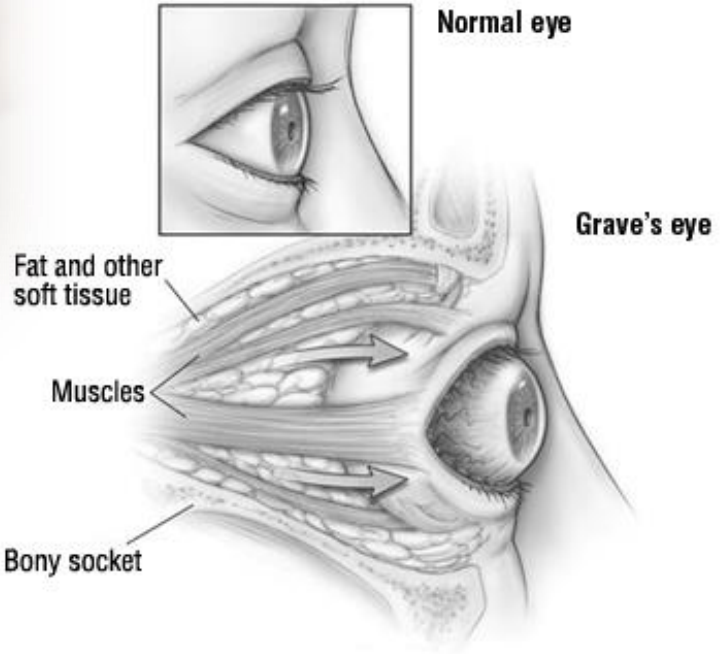






Graves Ophthalmopathy





Thyrotoxicosis

- Thyroid storm
  - Life-threatening emergency
  - Usually associated with severe physiologic stress or overdose of thyroid hormone
  - Manifests as high fever tachycardia, tachypnea
    - Can lead to mortality due to uncontrolled HTN
    - Mortality up to 50% even when treated

Thyrotoxicosis

Signs and Symptoms	Management
• High fever (>41°C) • Increased activity of sympathetic nervous system. • Irritability, delirium or coma • Tachycardia and hypotension • Vomiting and diarrhea	• Fluids • Antipyretics (acetaminophen) • Maintain HR <100 • Monitor for arrhythmia's • 12-lead to identify ischemia

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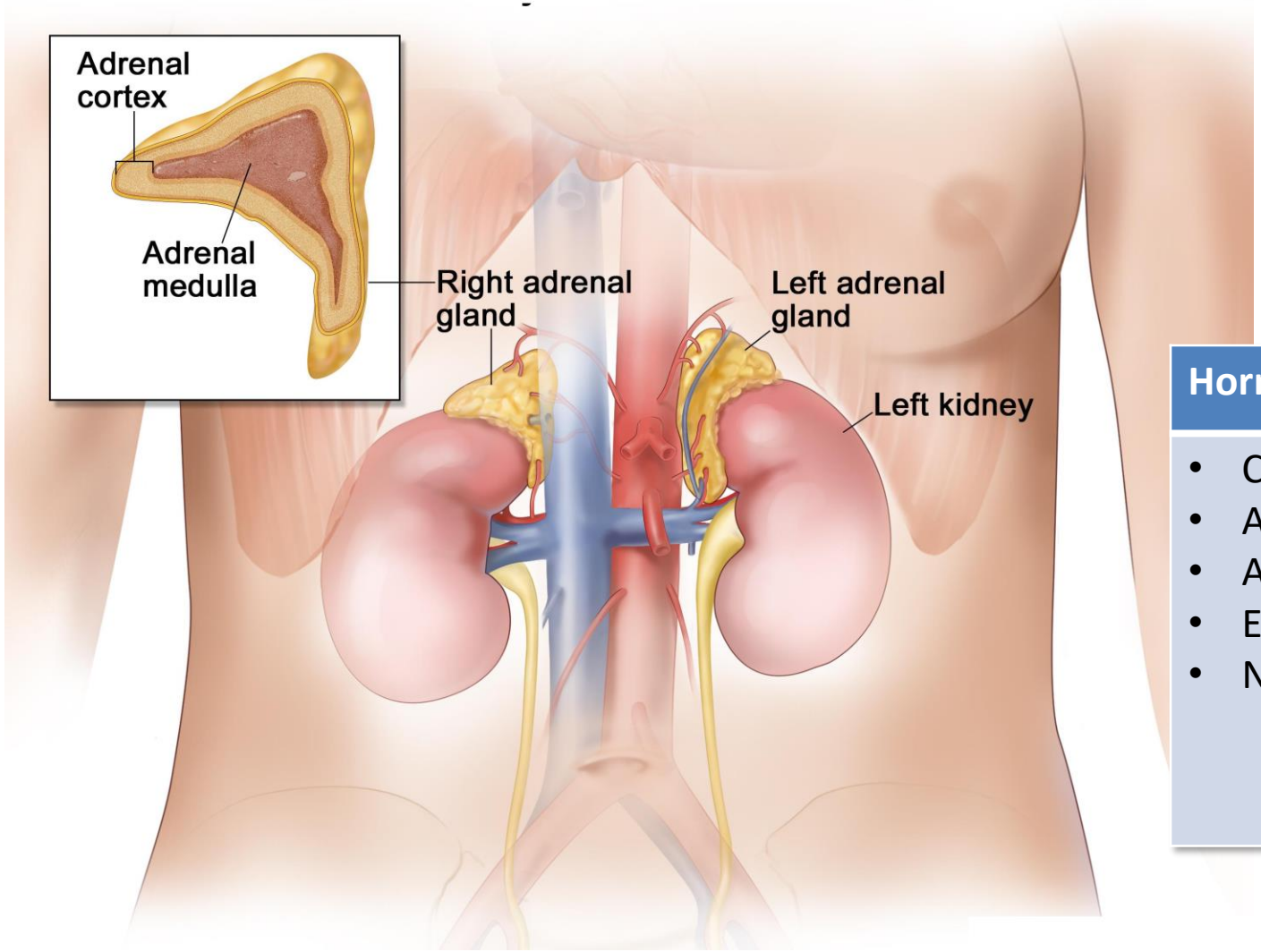
Endocrinology

DISORDERS OF THE ADRENAL GLAND

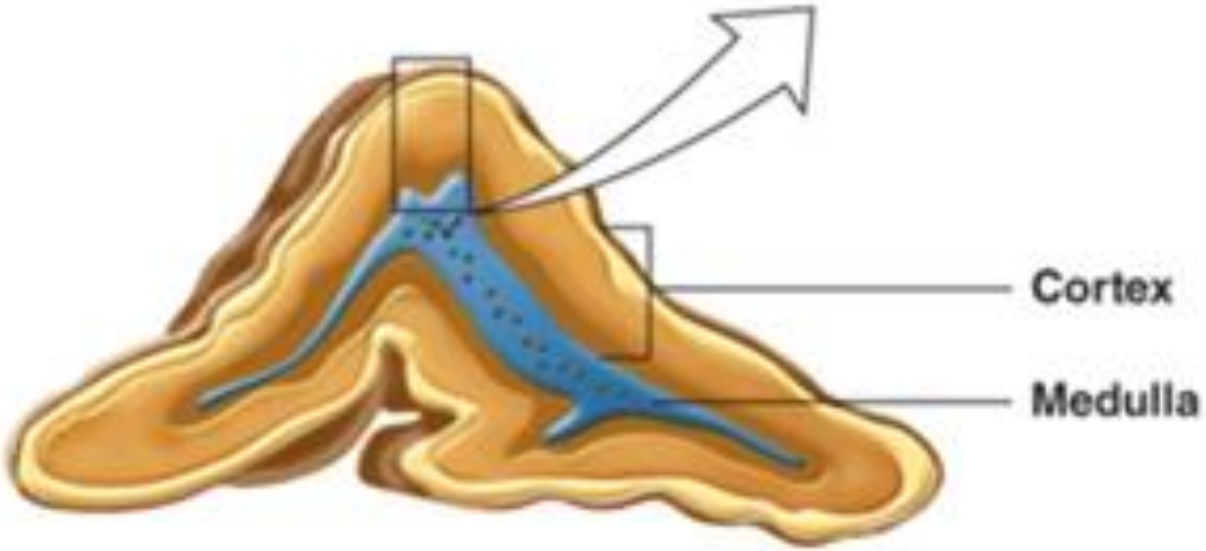
Adrenal Glands

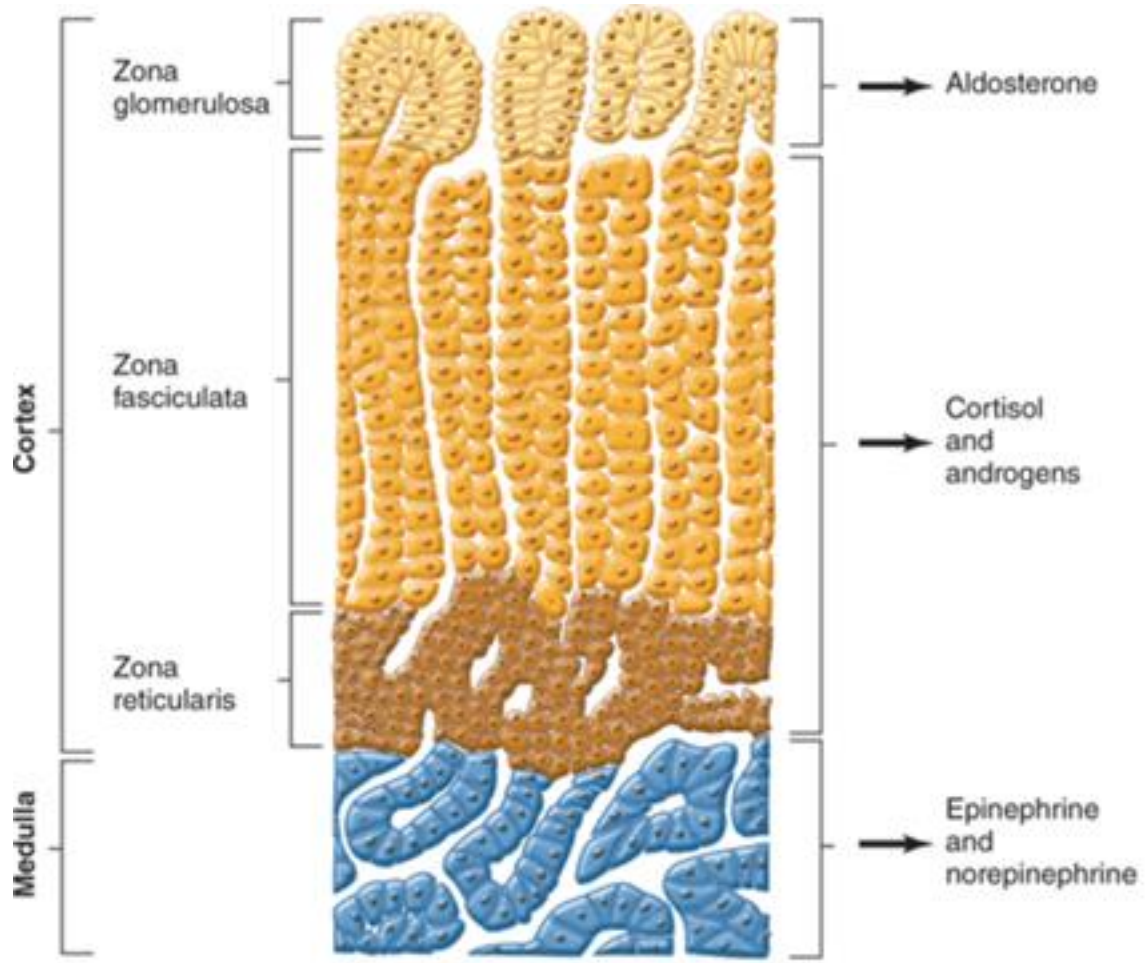


Hormones

- Cortisol
- Aldosterone
- Adrenal androgens
- Epinephrine
- Norepinephrine

Adrenal Gland Anatomy

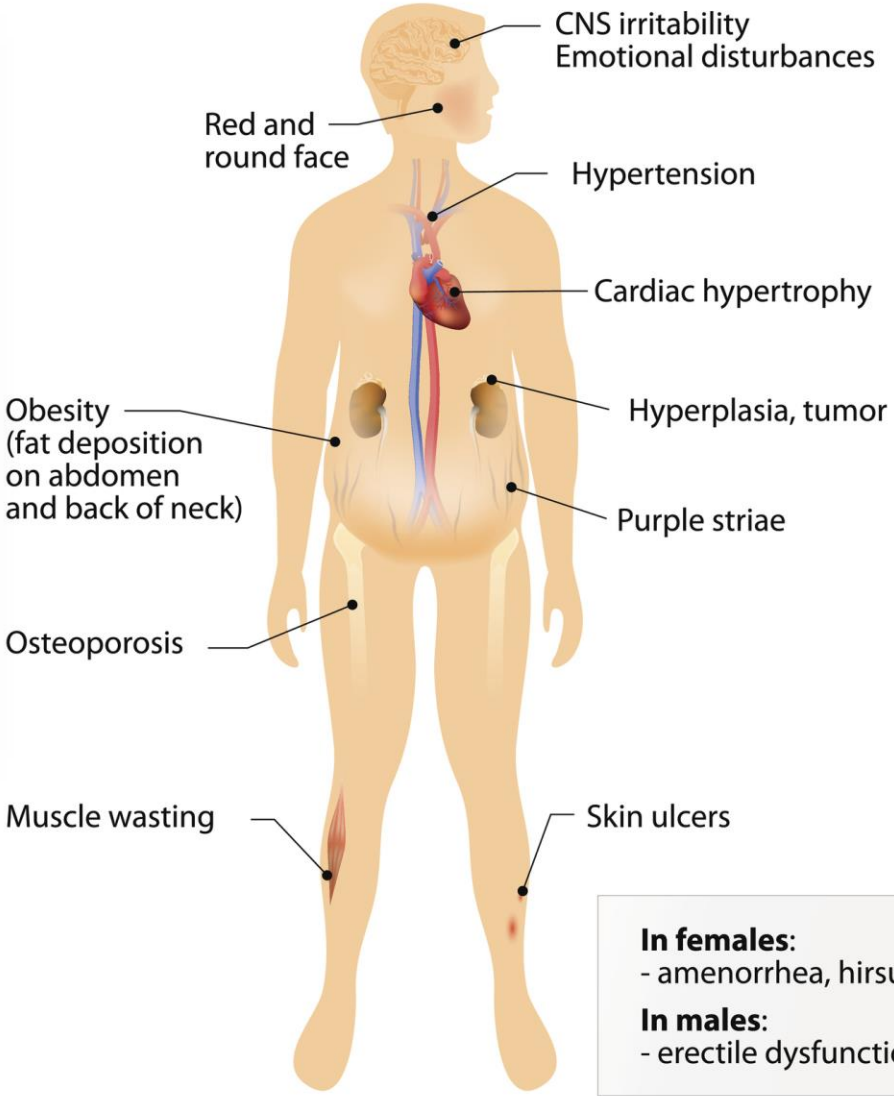




Hyperadrenalism

- Cushing's syndrome
  - Body exposed to excess glucocorticoids of any etiology
    - Excess of cortisol production or excessive use of cortisol
    - Tumours of the pituitary gland or adrenal cortex can stimulate the production of excess hormone
  - Characteristic changes in many body systems
    - Metabolism of carbohydrate, protein, and fat is disturbed.
    - Blood glucose elevates.
    - Body proteins are broken down.
    - Bones become weaker and more susceptible to fracture.

Cushing's Disease



In females:
- amenorrhea, hirsutism

In males:
- erectile dysfunction

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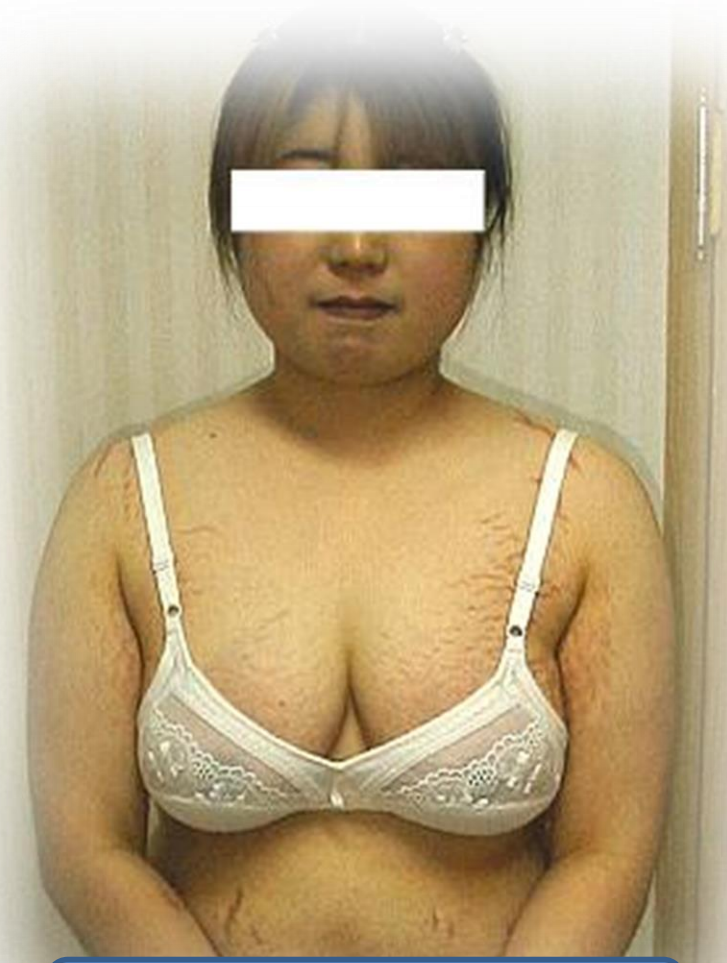
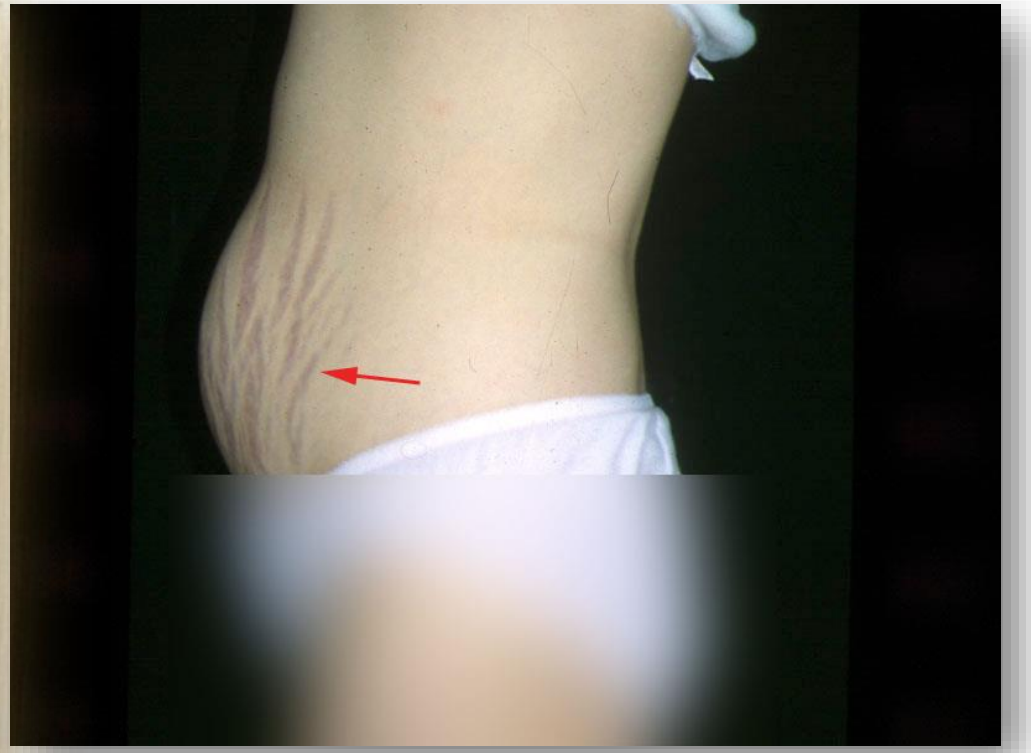
Cushing's Disease

Moon Face

Buffalo Hump

Cushing's Disease





Striations

Adrenal Insufficiency

Management

- Support ABCs.
- Use caution when establishing IV access.
- Obtain a glucose level.
- Monitor the patient's blood pressure.
- Treat abnormalities as they present.

Adrenal Insufficiency

Primary Adrenal Insufficiency

- Also known as Addison disease
- Caused by atrophy or destruction of glands
- Affects glucocorticoids and mineralcorticoids
  - Rare disease
  - Most frequently result of idiopathic atrophy
  - Normal life expectancy when treated
  - Frequently exhibit increased pigmentation of the skin

Secondary Adrenal Insufficiency

- Relatively common condition
- Defined as a lack of ACTH secretion from the pituitary gland
- ACTH stimulates the adrenal cortex to manufacture and secrete cortisol.
- Patients who abruptly stop taking corticosteroids

Adrenal Insufficiency

Signs and Symptoms (Chronic adrenal insufficiency)

- Unexplained weight loss
- Fatigue
- Vomiting
- Diarrhea
- Anorexia
- Salt craving
- Muscle and joint pain
- Abdominal pain
- Postural dizziness
- Increased pigmentation in the extensor surfaces, palm creases, and oral mucosa

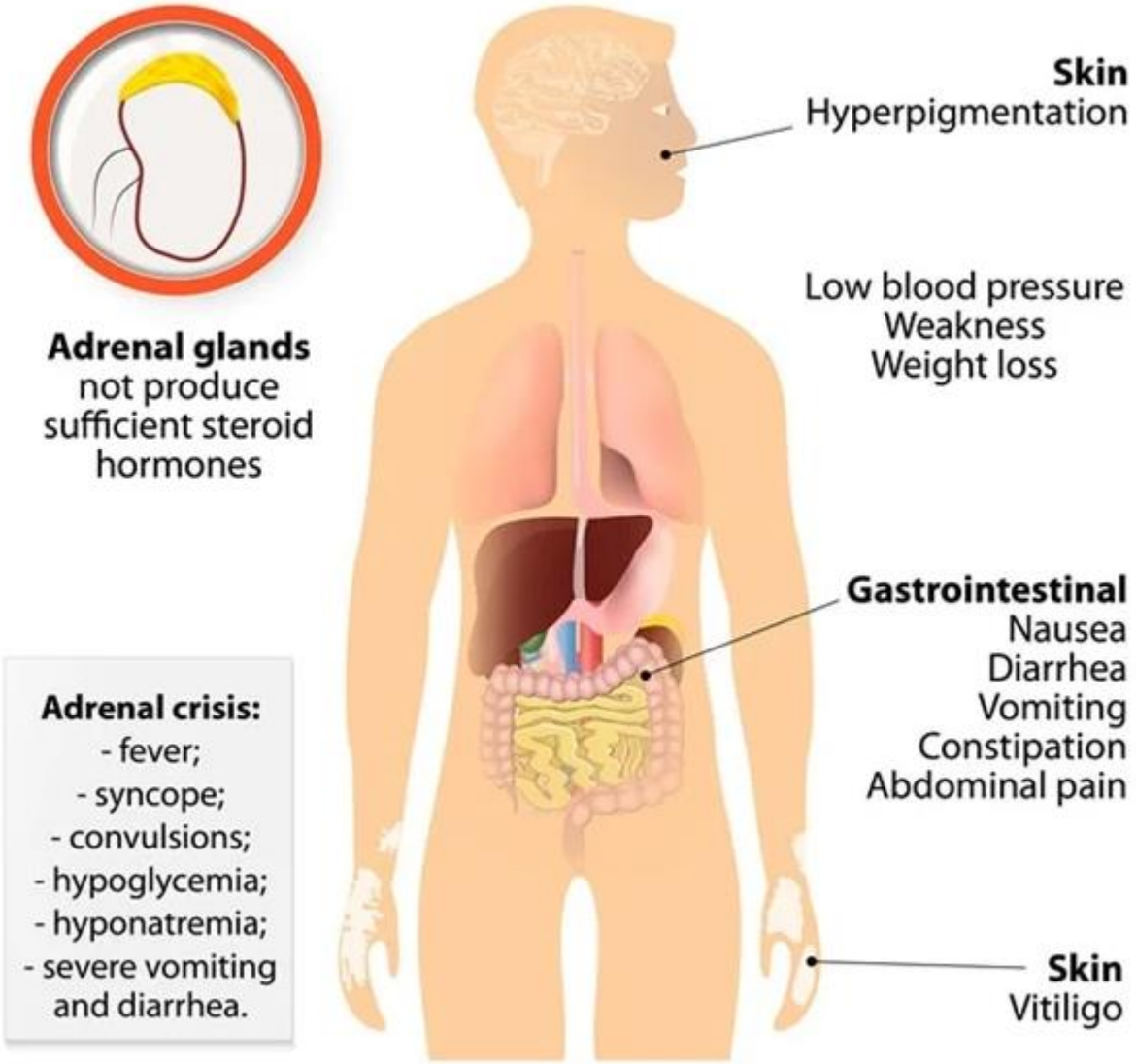
Adrenal Insufficiency

- Addisonian crisis
  - Results from an acute exacerbation of chronic insufficiency
  - Corticosteroids withdrawal is the most common cause.
  - Primary clinical manifestation is shock.
  - May also manifest nonspecific symptoms.

Adrenal Crisis

- Adrenal insufficiency
  - With severe hypotension refractory to vasopressors
- Acute destruction of HPA axis or adrenal gland
- Acute stress leading to primary and secondary adrenal insufficiency resulting in crisis

Presentation



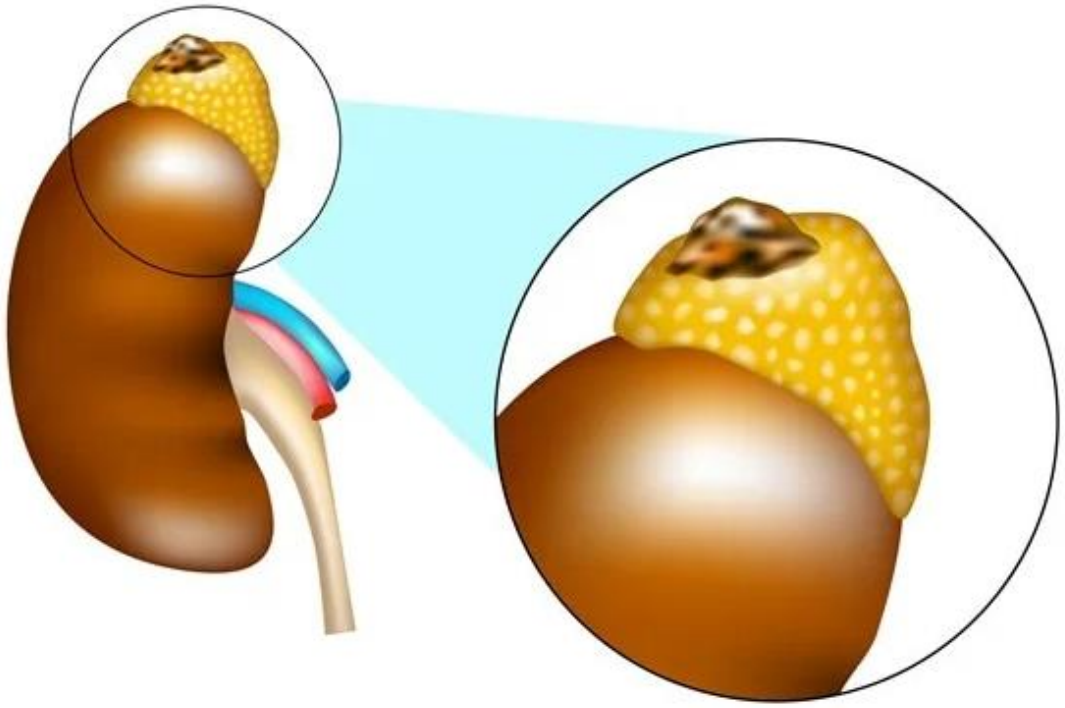
Adrenal Insufficiency

Management

- Attempt to determine underlying cause
  - Stressors i.e. Surgery, bacterial infection, HIV Obtain blood glucose level and treat for hypoglycemia if present.
- Establish IV and provide aggressive fluid resuscitation
- Often 1 L/hour
- Continuous cardiac monitoring
- Vasopressor medications (ACP)
- Treat with corticosteroids medications (Hospital)

Pheochromocytoma

- Causes high circulating levels of catecholamines
- Caused by tumour on adrenal gland
- Can lead to hypermetabolic state mimicking thryotoxicosis
- Glucagon contraindication



Pheochromocytoma

Signs and Symptoms

- 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