- Clinical, laboratory and instrumental examination in thyroid disorders.
- Particularities of clinical and laboratory examination in anemia.
- Particularities of clinical and laboratory examination in diabetes.
- Diabetic coma.

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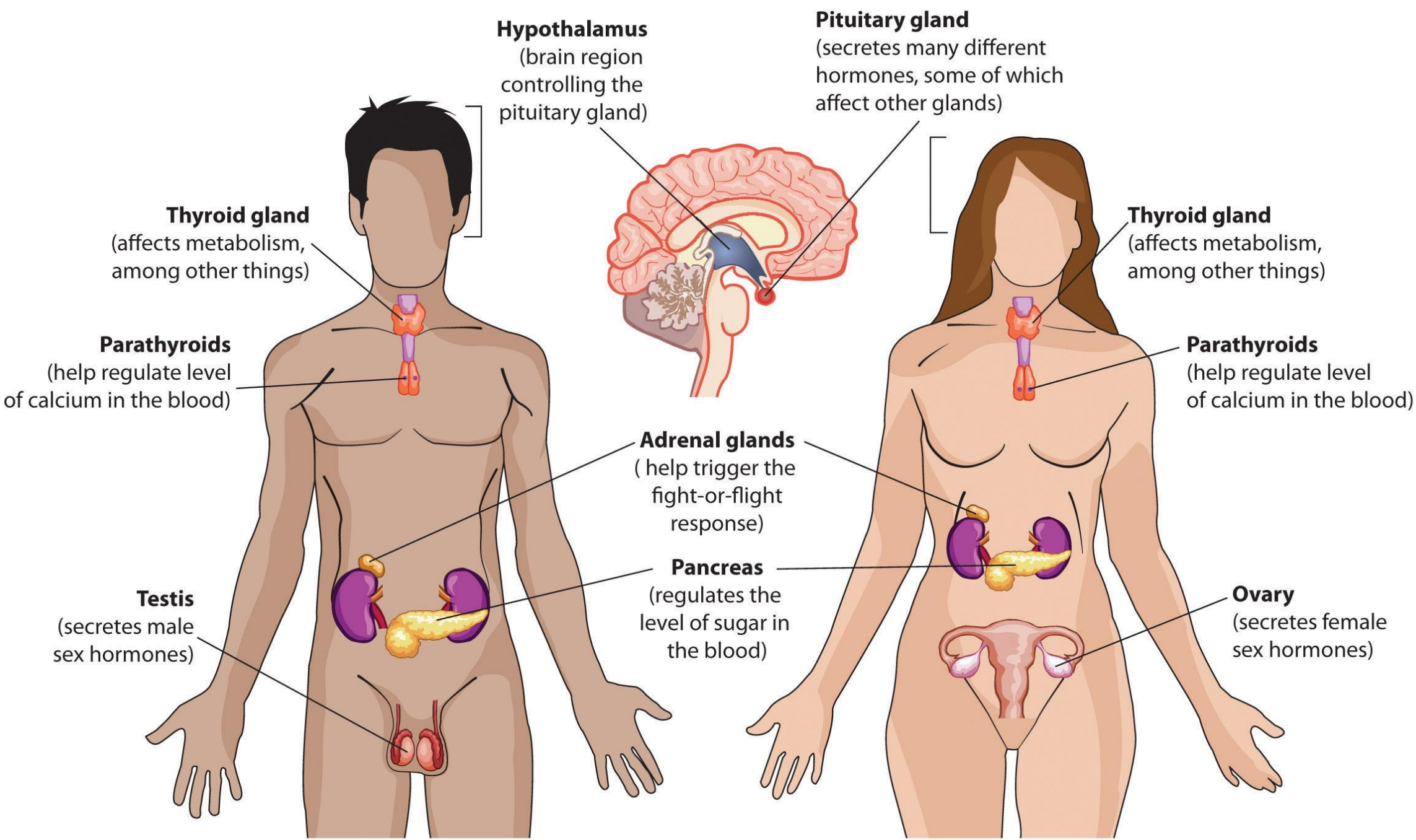
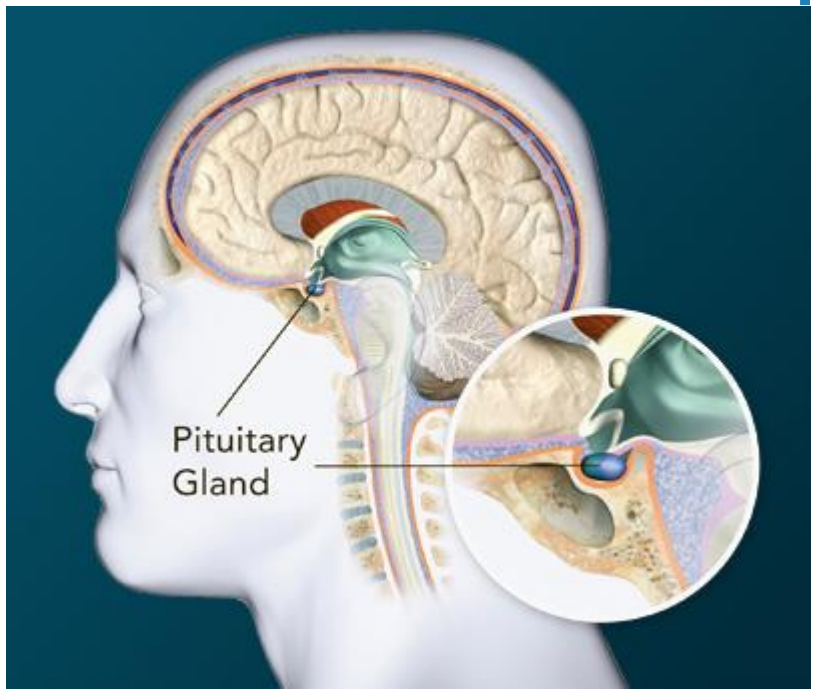

Hypothalamus
(brain region controlling the pituitary gland)
Pituitary gland
(secretes many different hormones, some of which affect other glands)
Thyroid gland
(affects metabolism, among other things)
Parathyroids
(help regulate level of calcium in the blood)
Adrenal glands
( help trigger the fight-or-flight response)
Pancreas
(regulates the level of sugar in the blood)
Testis
(secretes male sex hormones)
Thyroid gland
(affects metabolism, among other things)
Parathyroids
(help regulate level of calcium in the blood)
Ovary
(secretes female sex hormones)

# Pituitary gland

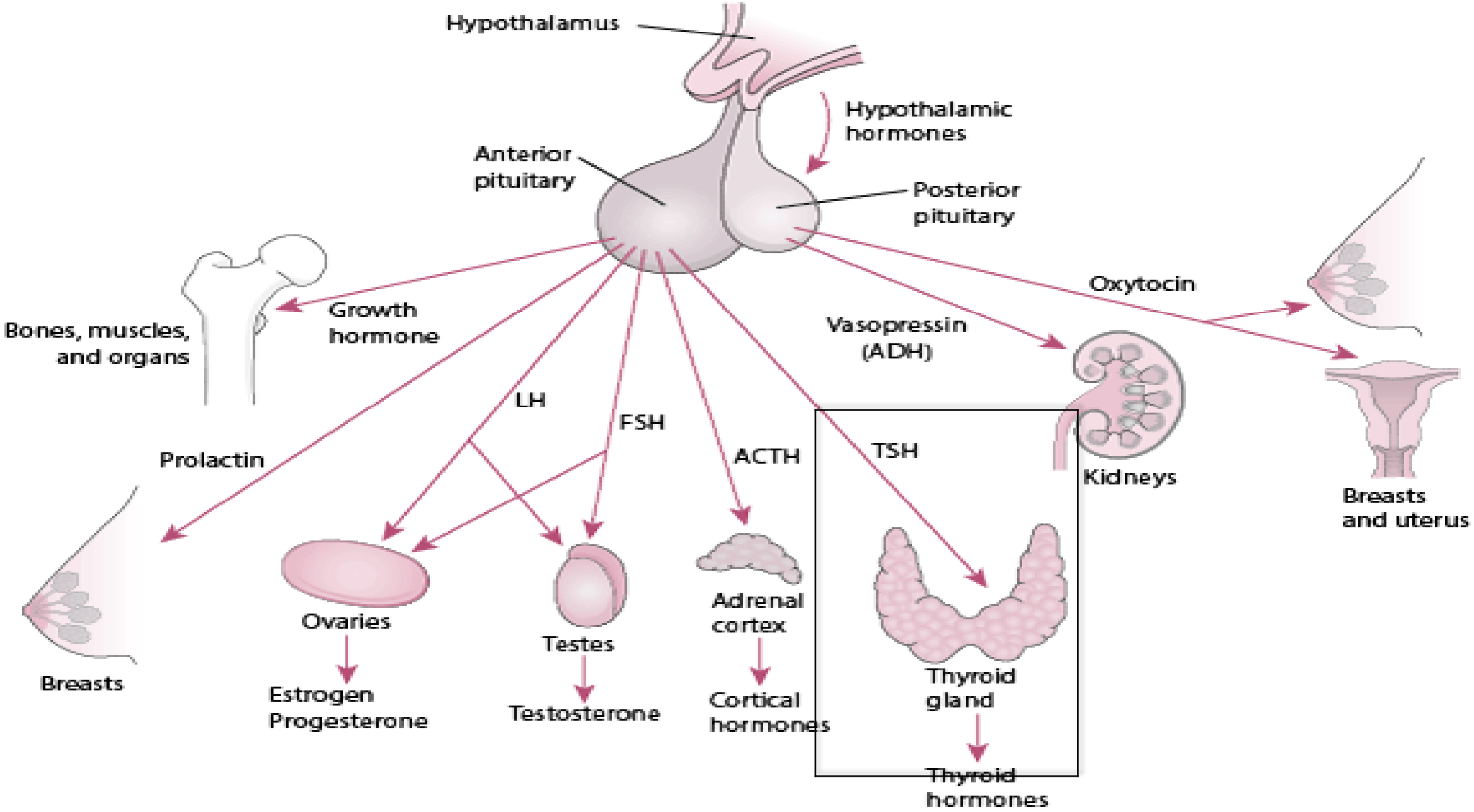



Master gland of body

– Located in the depression of sphenoid bone

Produces many hormones that affect other glands

- **thyroid stimulating hormone**
- Somatotropin- growth hormone
- Lutenizing (LH)- causes ovulation
- ICSH- causes testes to secrete testosterone
- Melanocyte stimulating- distribution of melanin in skin
- ADH- antidiuretic hormone


Hypothalamus
Hypothalamic hormones
Anterior pituitary
Posterior pituitary
Bones, muscles, and organs
Growth hormone
Oxytocin
Vasopressin (ADH)
Kidneys
Breasts and uterus
Prolactin
LH
FSH
ACTH
TSH
Breasts
Ovaries
Estrogen
Progesterone
Testes
Testosterone
Adrenal cortex
Cortical hormones
Thyroid gland
Thyroid hormones

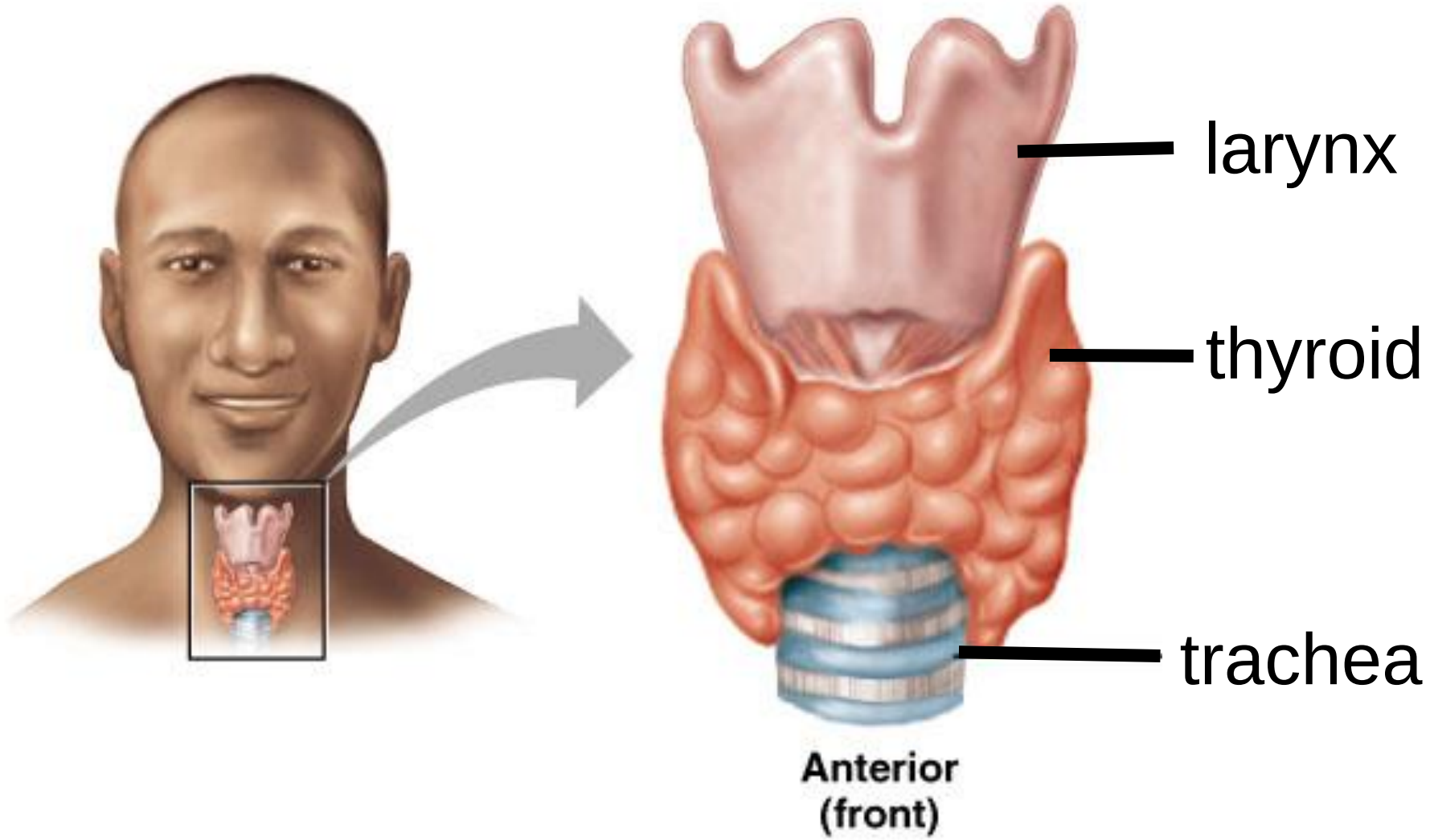
# Thyroid-Stimulating Hormone (TSH)

- Acts on the thyroid gland, stimulating it to release T3 & T4
- These thyroid hormones increase glucose catabolism and body heat production.
- Negative feedback mechanism involved in regulating levels.

# Adrenocorticotropic Hormone (ATCH)

- Acts on the adrenal cortex, stimulating it to secrete glucocorticoids (e.g., cortisol).
- **Glucocorticoids promote the synthesis of glucose from noncarbohydrate sources such as amino acids, and fatty acids**
- Negative feedback mechanism involved in regulating levels.

# Thyroid Gland



# Thyroid Hormones

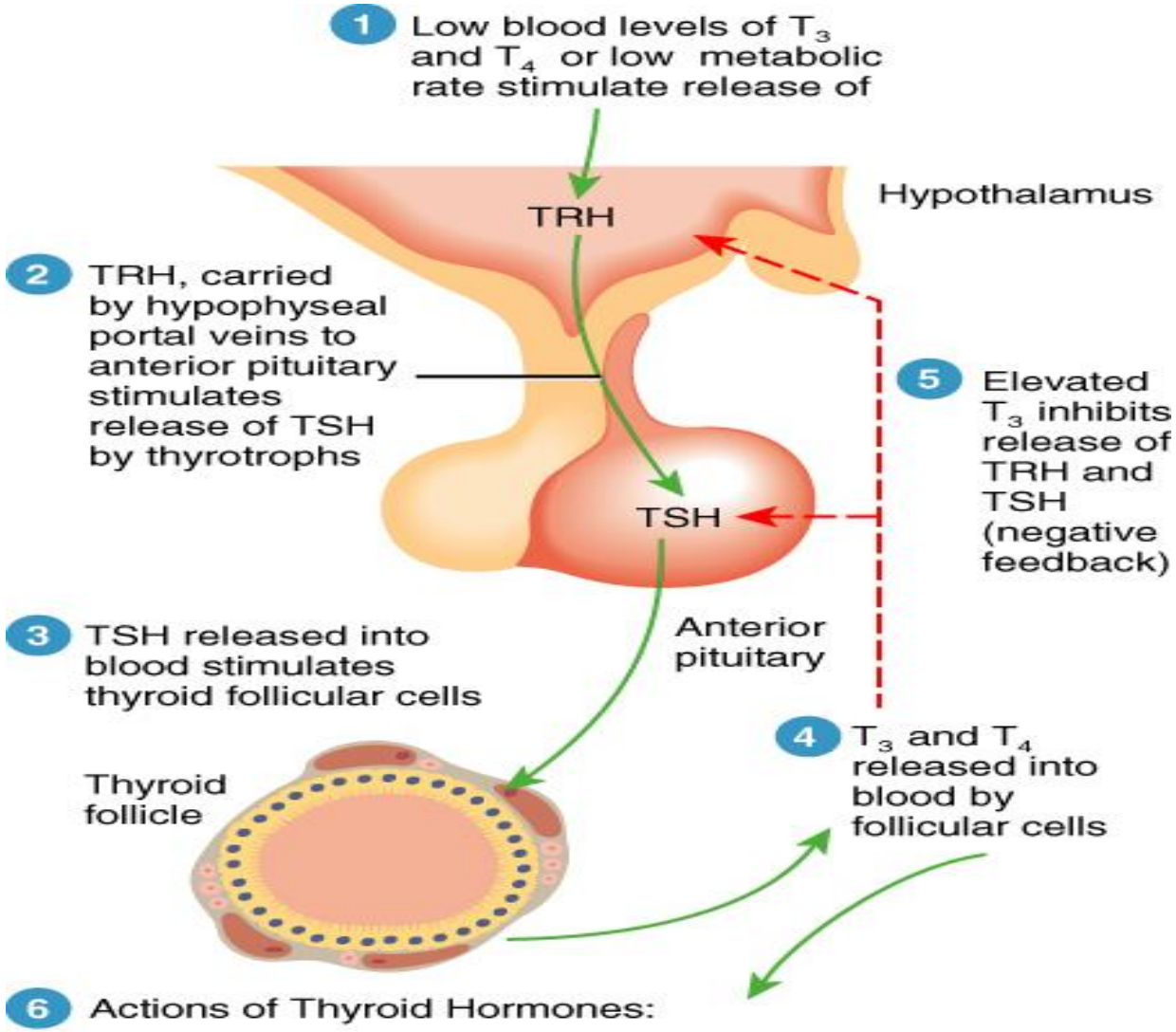
**Thyroid gland selectively uptakes iodine to produce $T_3$ & $T_4$**

- **Thyroxine** ($T_4$)
- **Triiodothyronine** ($T_3$)

Both control metabolic rate and cellular oxidation

- **Calcitonin** (from parafolicular cells)- lowers blood $Ca^{++}$ levels and causes $Ca^{++}$ reabsorption in bone

# Thyroid Hormones




18.12

# Thyroid gland

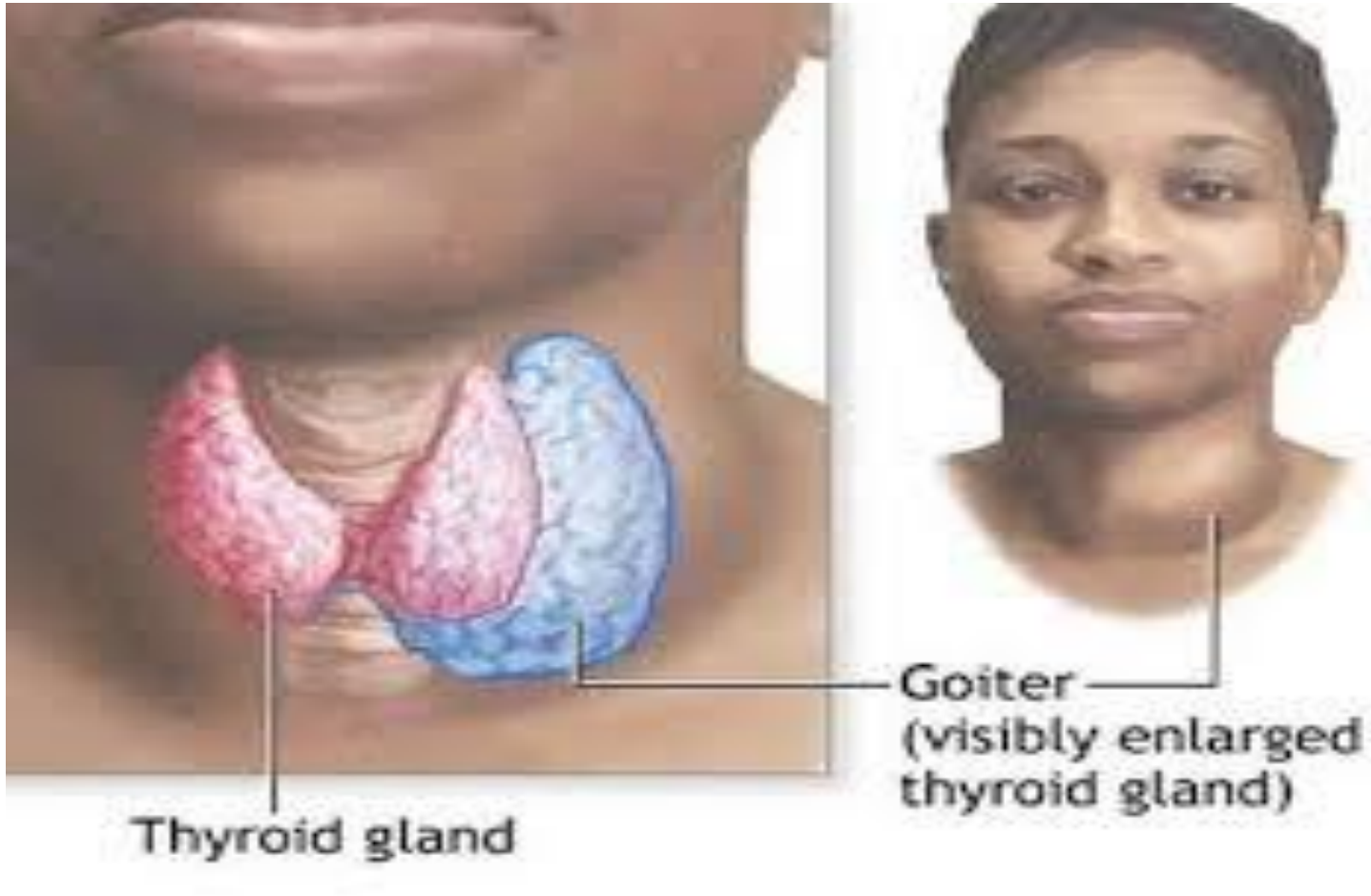
Increased thyroid hormone release causes ***hyperthyroidism,*** commonly called **Graves' disease.**

- Signs and symptoms:
  - insomnia, fatigue
  - tachycardia
  - hypertension
  - heat intolerance
  - weight loss

- Long term hyperthyroidism:
  - ***Exopthalmos***
    - bulging of the eyeballs (picture Barbara Bush)
  - In severe cases - a medical emergency called *thyrotoxicosis* can result.

# Goiter

Lack of iodine in diet
hyposecretion of T3 & T4



# Thyroid gland

**Calcitonin**, when released, lowers the amount of calcium in the blood.

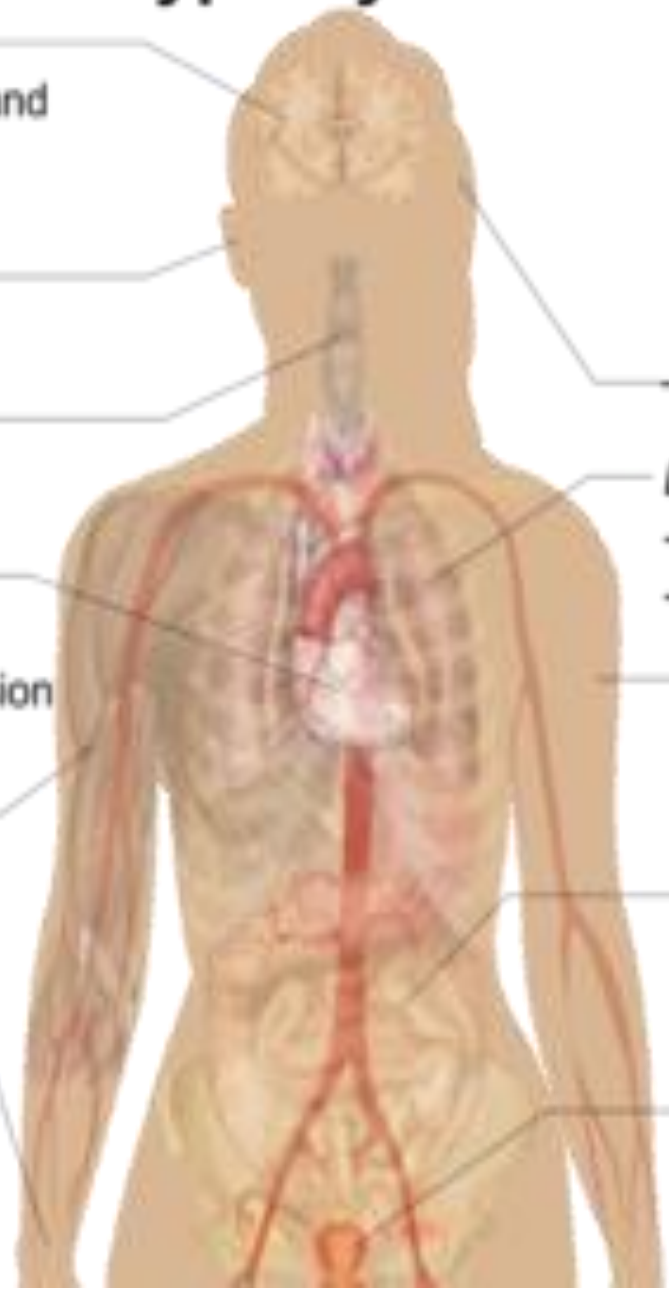
Inadequate levels of thyroid hormones = **hypothyroidism,** or **Myxedema.**


**Myxedema symptoms:**

- **Facial bloating**
- **weakness**
- **cold intolerance**
- **lethargy**
- **altered mental status**
- **oily skin and hair**
- **TX: replacement of thyroid hormone**

Signs and symptoms of

# Hypothyroidism



*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

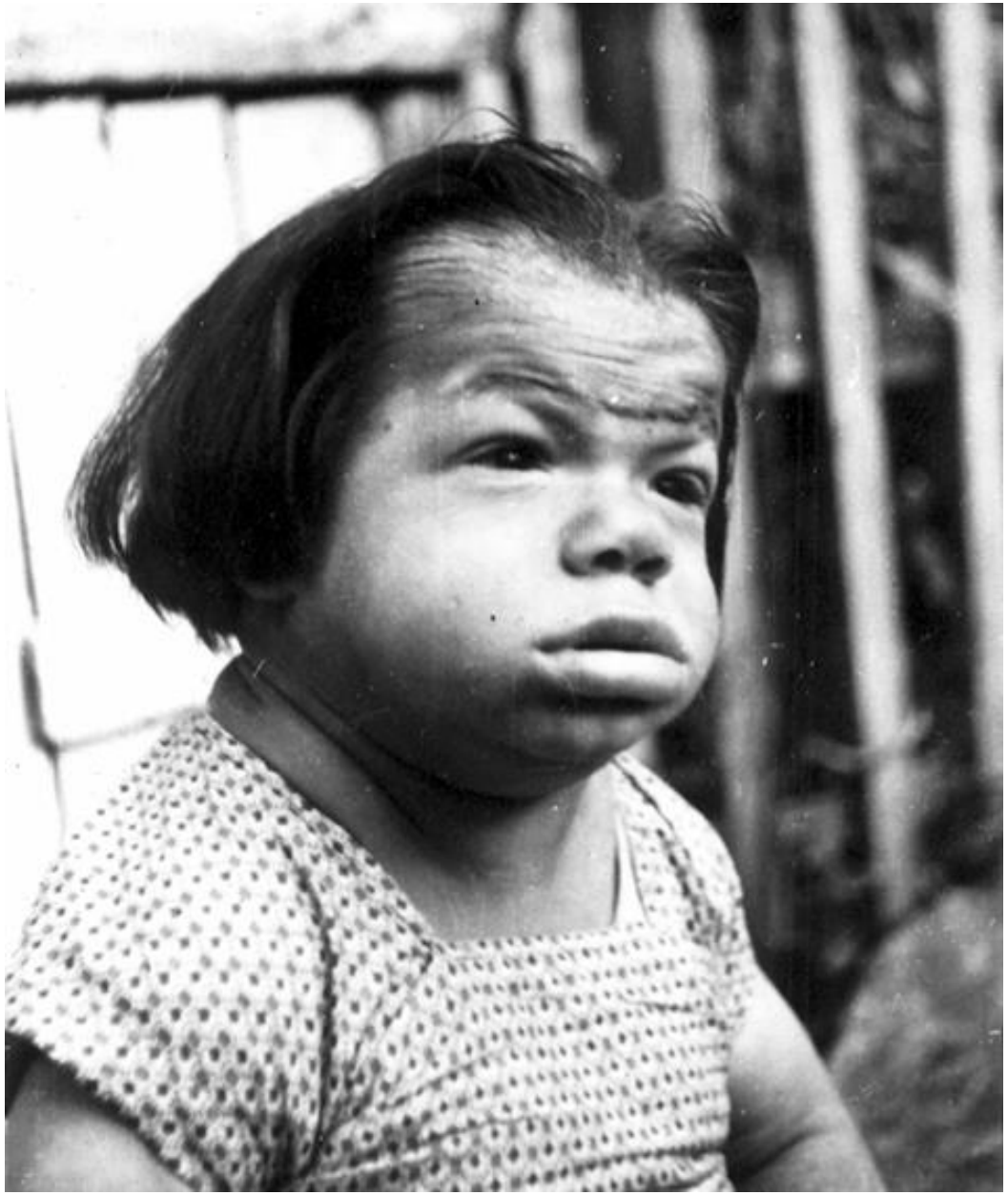
*Skin*
- Paresthesia
- Myxedema

*Intestines*
- Constipation
- Ascites

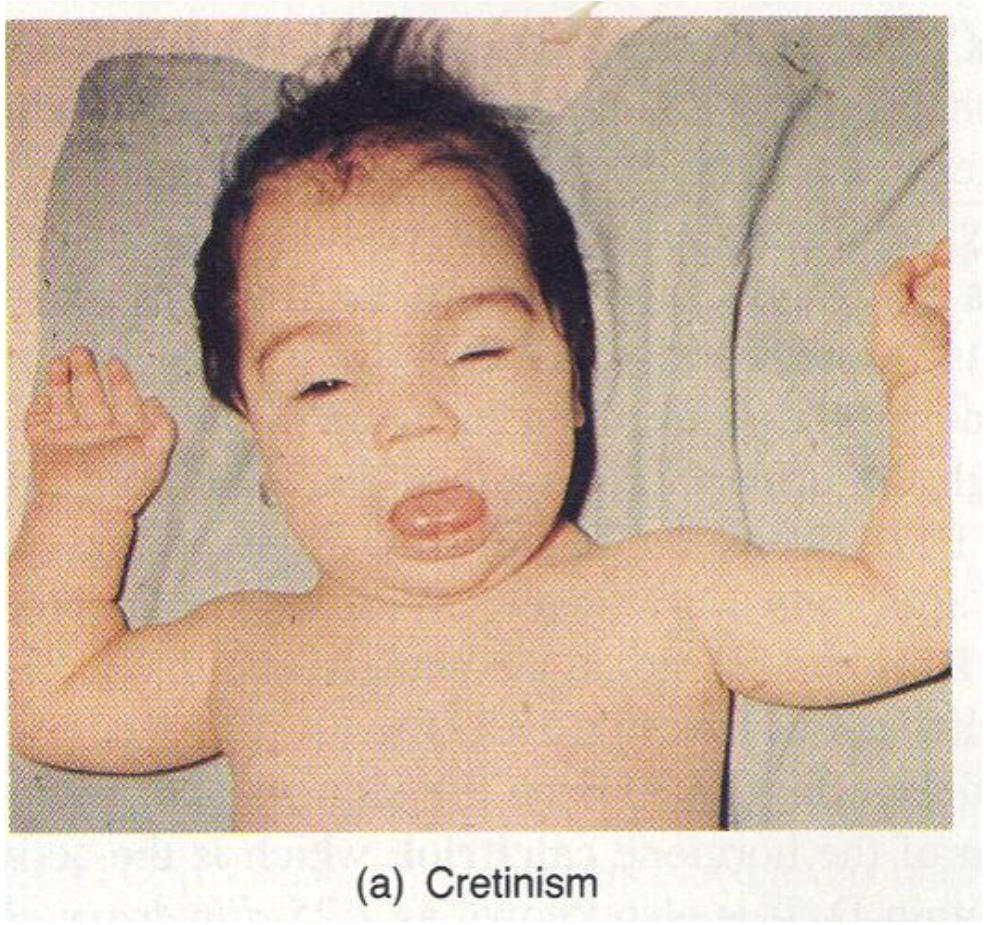
*Reproductive system*
- Menorrhagia

# Cretinism

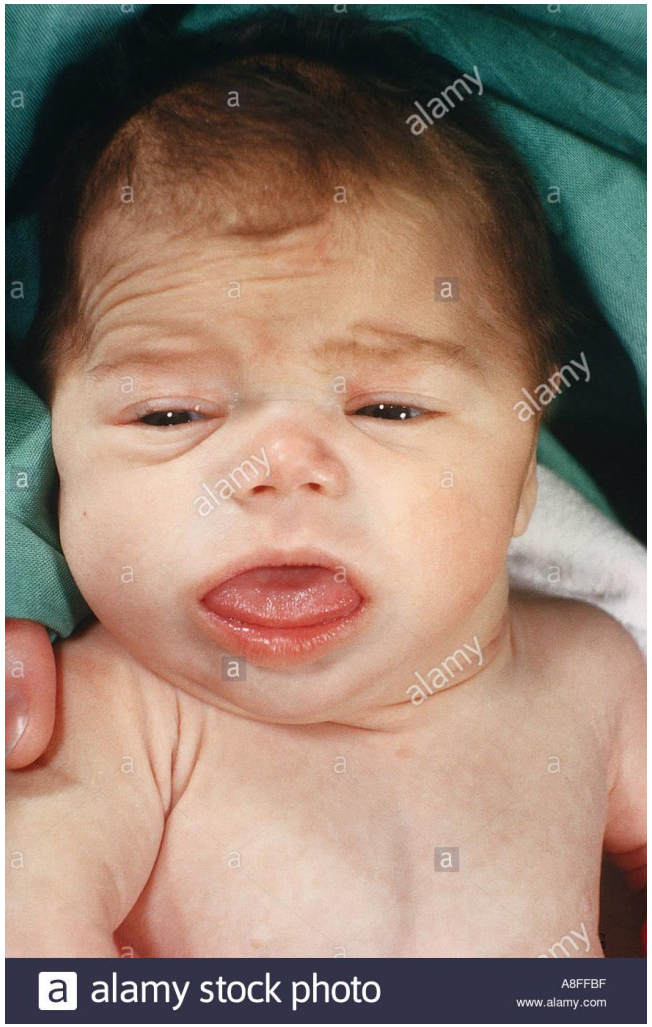
**hyposecretion of T3 & T4**



# Hyposecretion of TH- cretinism



(a) Cretinism



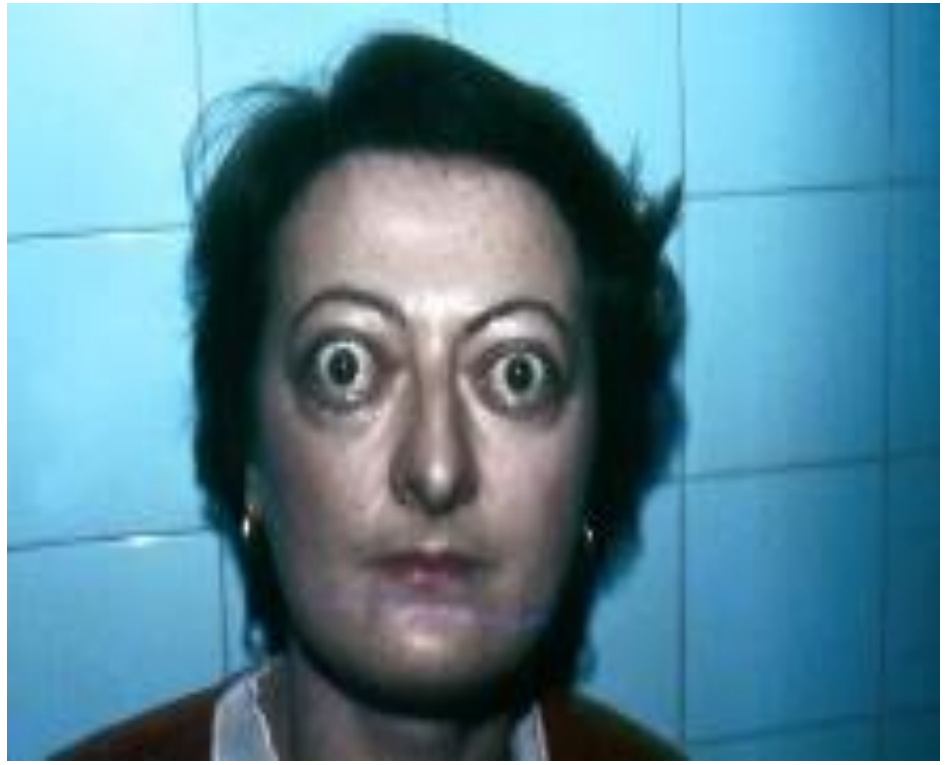
# Myxedema

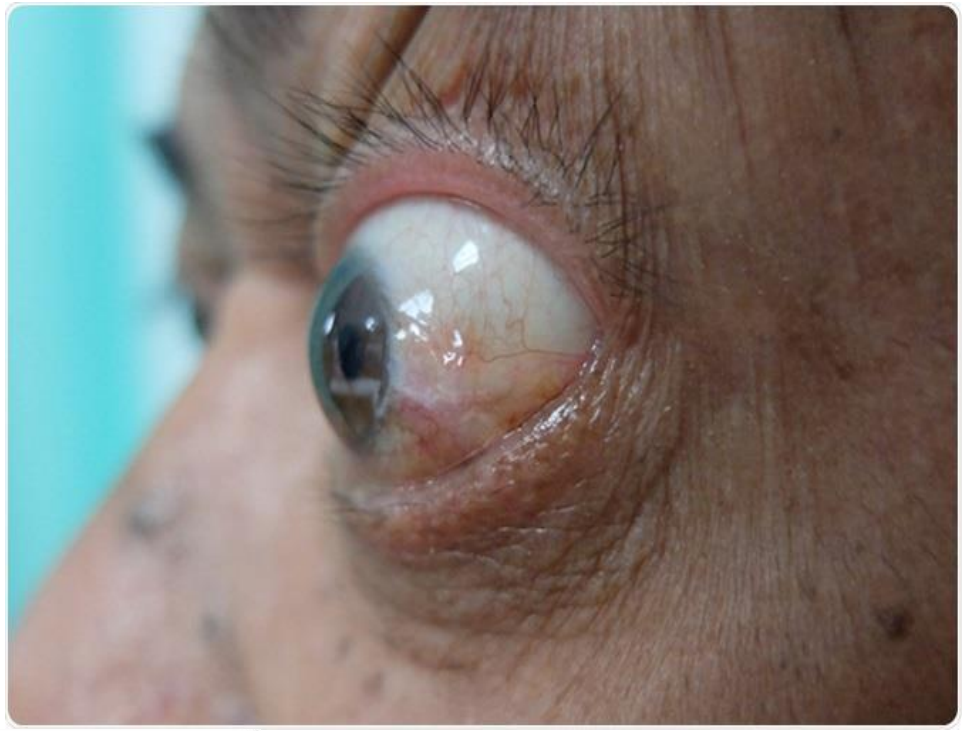
## hyposecretion of T3 & T4

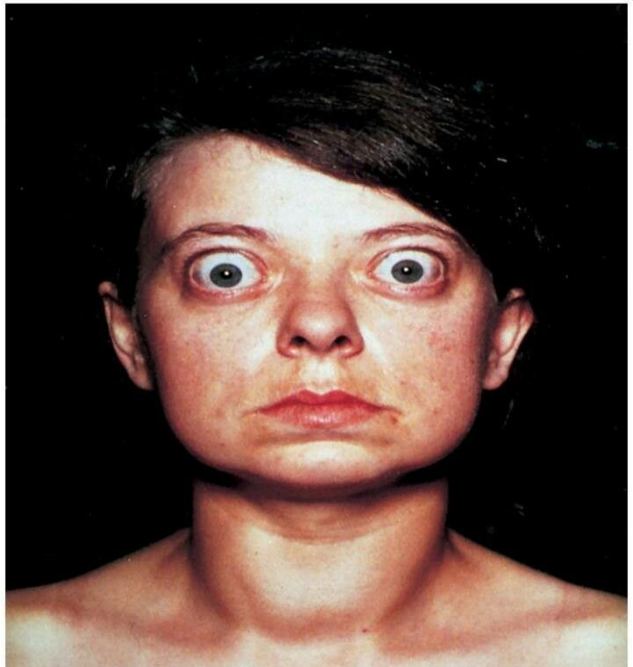
myxedema

After thyroid treatment

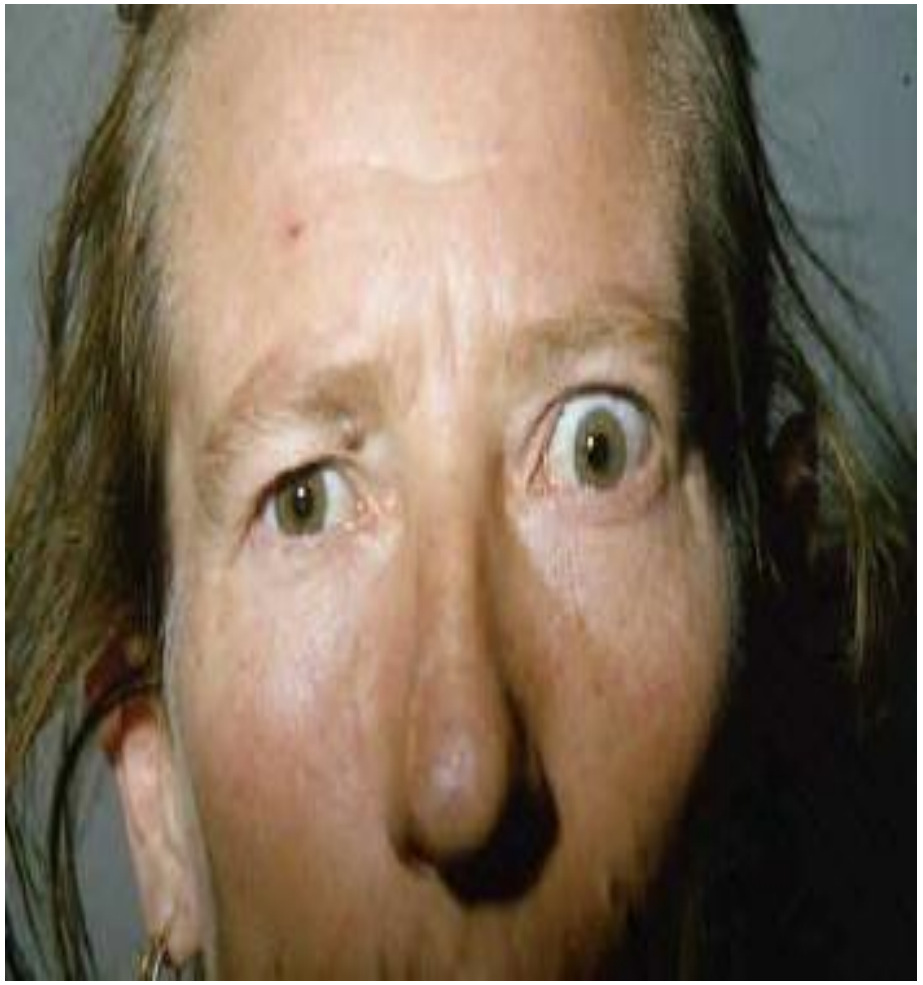
# Exophthalmos-
hyperthyroidism

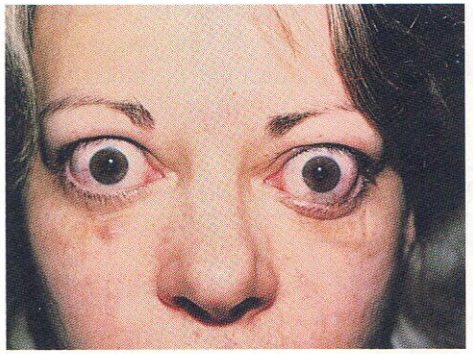




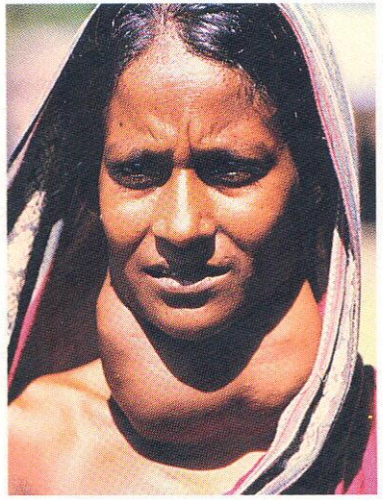


# Hypersecretion of TSH or TH

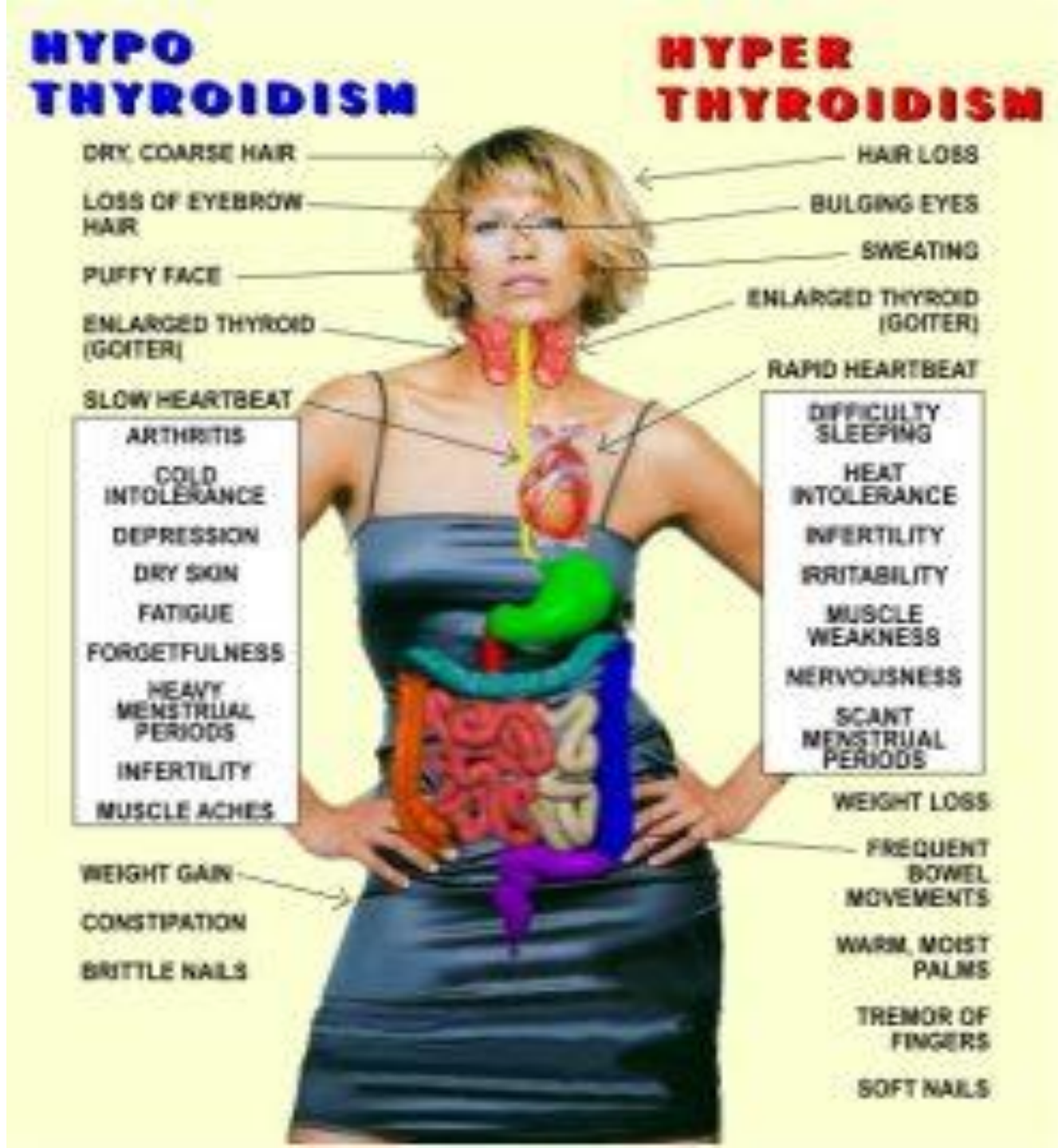




(b) Exophthalmos



(c) Goiter

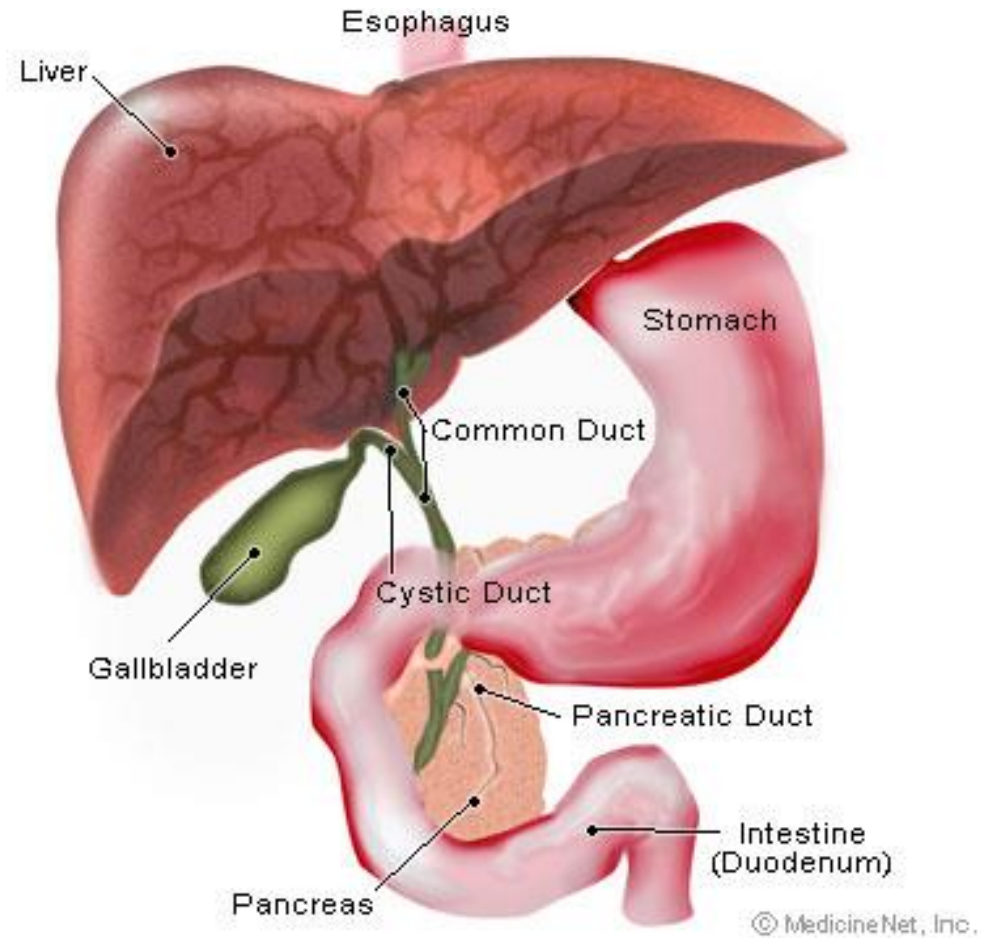

HYPO THYROIDISM
HYPER THYROIDISM
DRY, COARSE HAIR
LOSS OF EYEBROW HAIR
PUFFY FACE
ENLARGED THYROID (GOITER)
SLOW HEARTBEAT
ARTHRITIS
COLD INTOLERANCE
DEPRESSION
DRY SKIN
FATIGUE
FORGETFULNESS
HEAVY MENSTRUAL PERIODS
INFERTILITY
MUSCLE ACHES
WEIGHT GAIN
CONSTIPATION
BRITTLE NAILS
HAIR LOSS
BULGING EYES
SWEATING
ENLARGED THYROID (GOITER)
RAPID HEARTBEAT
DIFFICULTY SLEEPING
HEAT INTOLERANCE
INFERTILITY
IRRITABILITY
MUSCLE WEAKNESS
NERVOUSNESS
SCANT MENSTRUAL PERIODS
WEIGHT LOSS
FREQUENT BOWEL MOVEMENTS
WARM, MOIST PALMS
TREMOR OF FINGERS
SOFT NAILS

# Diabetes Mellitus (DM)

- **Diabetes mellitus (DM)** is a group of diseases characterized by high levels of blood glucose resulting from defects in **insulin production, insulin action, or both.**

- The effects of diabetes mellitus include long–term damage, dysfunction and failure of various organs.

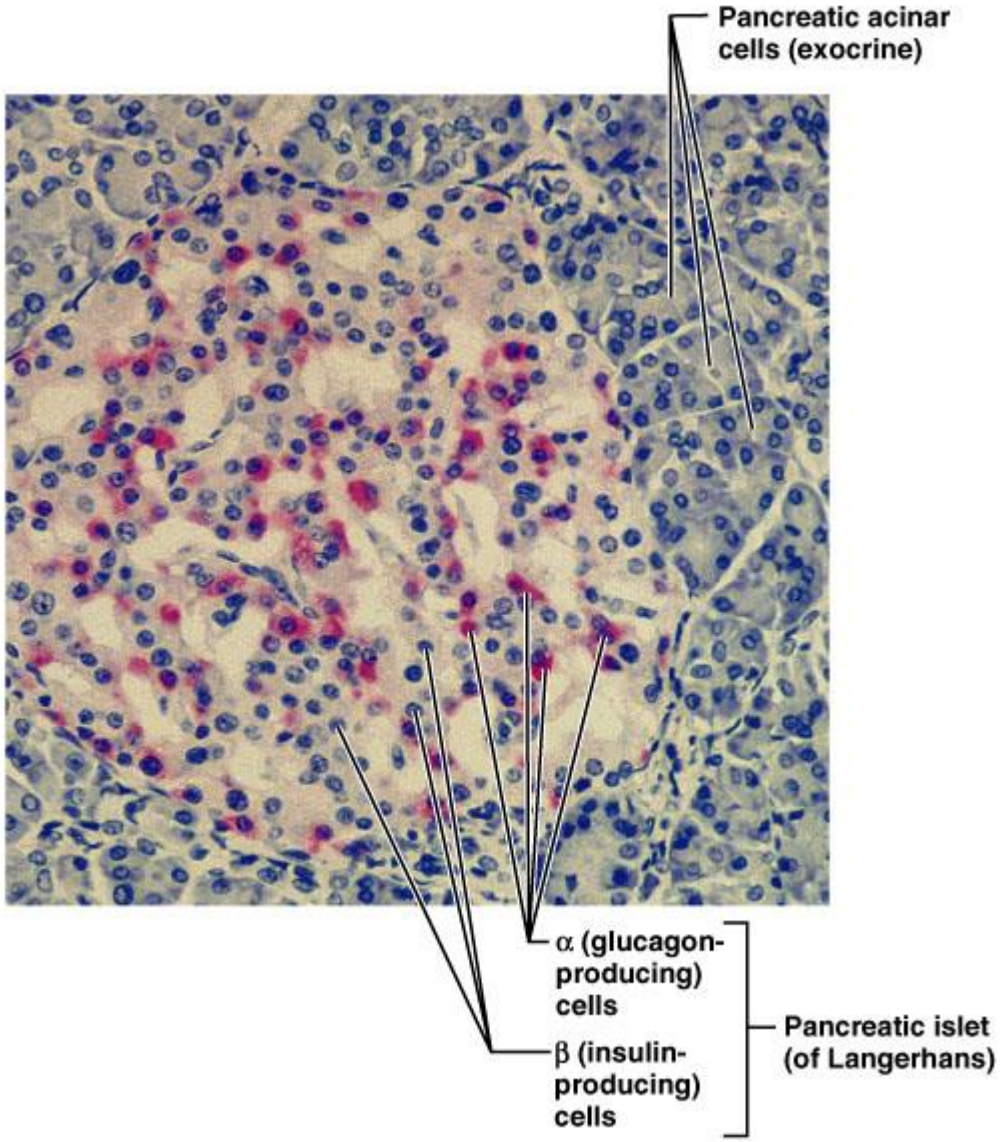
# –Pancreas-




secretes insulin, a hormone that transports glucose into cells.

Also secretes glucagon- increases glucose in bloodstream

# Islets of Langerhan



# Insulin

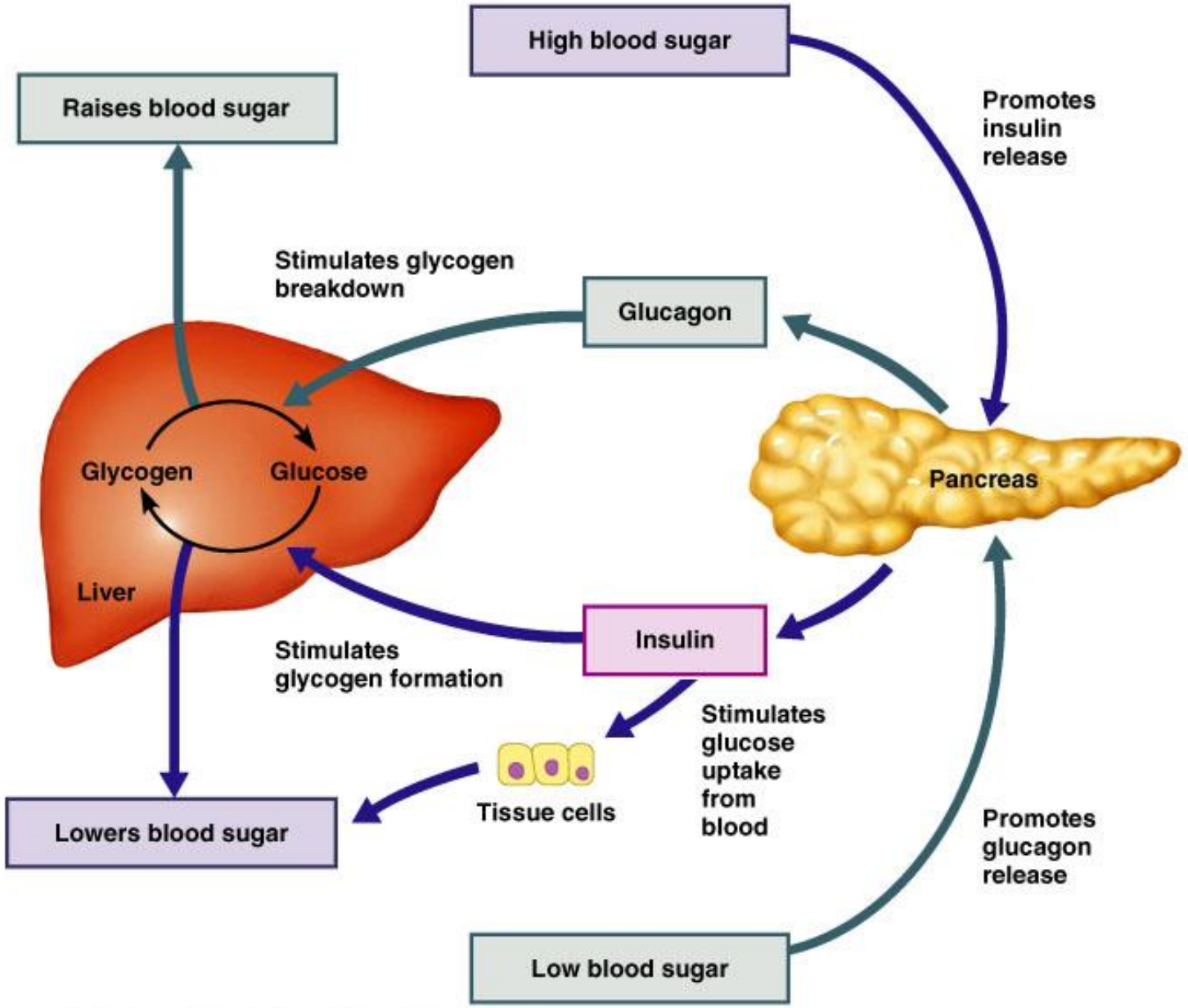
- Produced by the β cells of the Islets of Langerhan
- Catalyze oxidation of glucose for ATP production
- Lowers blood glucose levels by promoting transport of glucose into cells.
- Stimulates glucose uptake by  the liver and muscle cells.
- Stimulates glycogen synthesis in the liver and muscle cells (*glycogenesis*).
- Stimulates amino acid uptake and protein synthesis
- Stimulate lipogenesis
- Decreases glycogenolysis
- Decreases gluconeogenesis

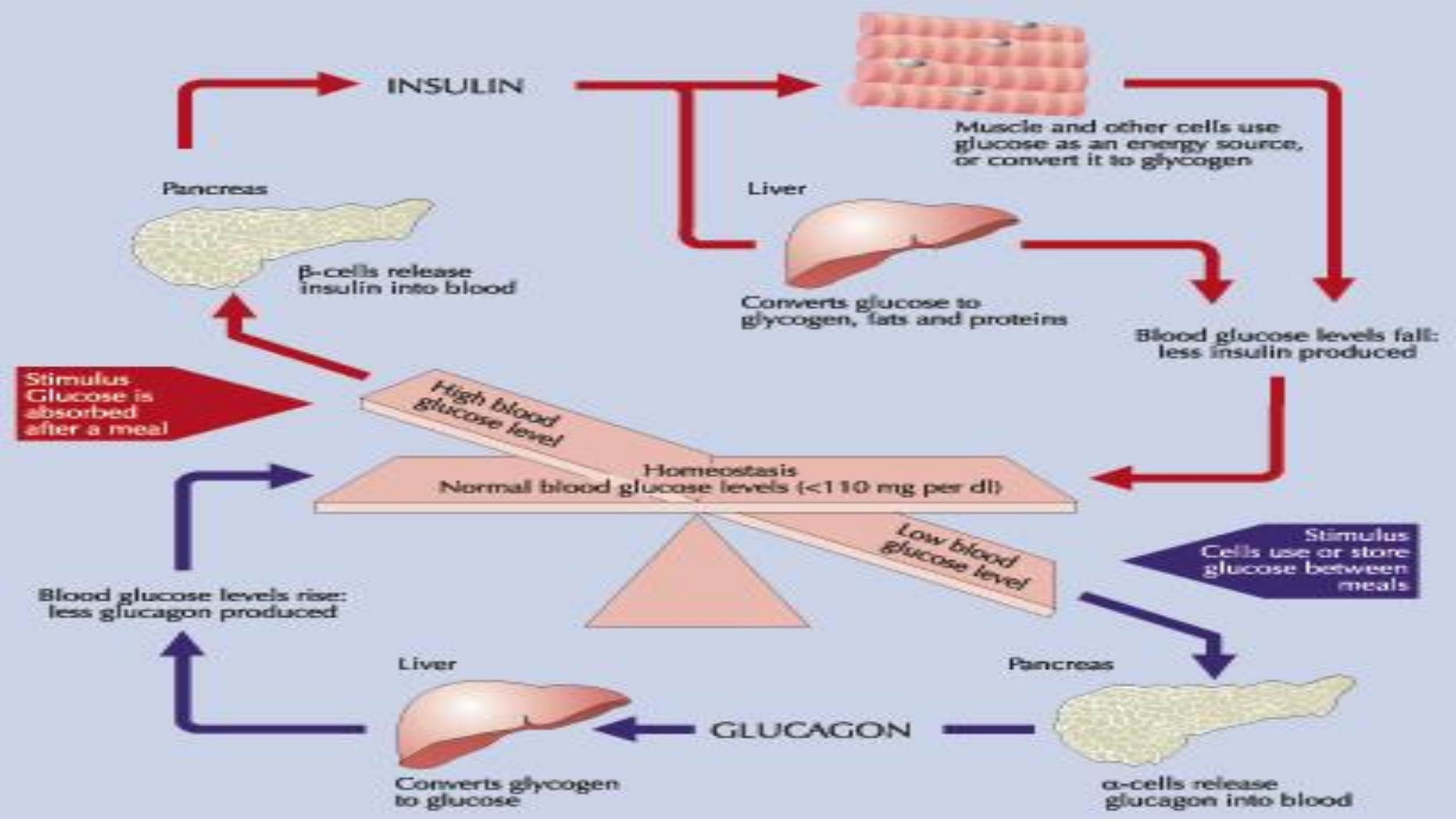
# Glucagon

- Produced by the $\alpha$ cells of the Islets of Langerhans
- Stimulates change of glycogen to glucose in the liver (*glycogenolysis*).
- Synthesis of glucose from lactic acid and non carbohydrate molecules such as fatty acids and amino acids (*gluconeogenesis*)
- Causes ↑ in blood glucose concentration

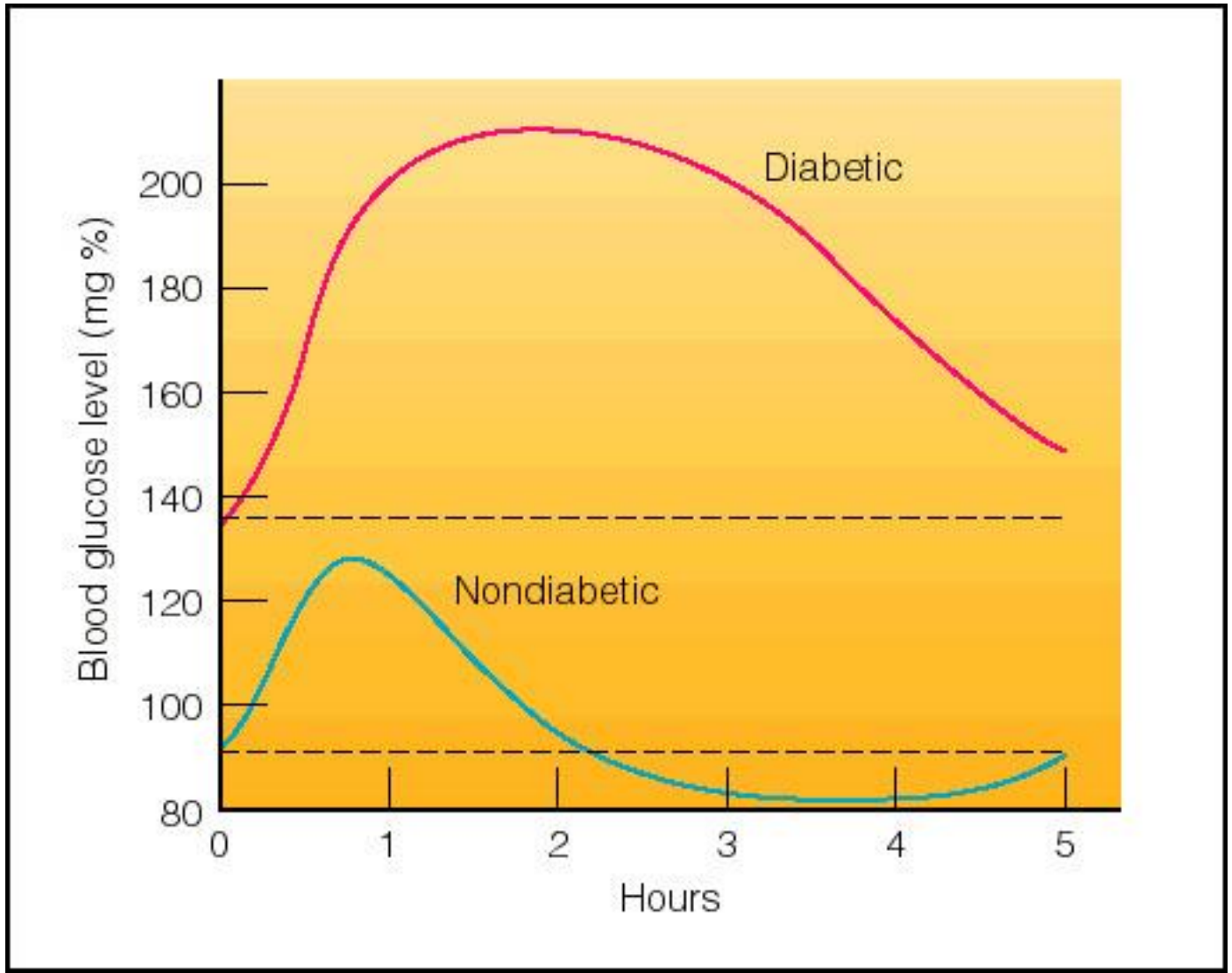
***hypoglycemic***- low blood sugar↑; deficient in glucagon

# Regulation of Blood Sugar Levels




INSULIN
Muscle and other cells use glucose as an energy source, or convert it to glycogen
Pancreas
β-cells release insulin into blood
Liver
Converts glucose to glycogen, fats and proteins
Blood glucose levels fall: less insulin produced
Stimulus
Glucose is absorbed after a meal
High blood glucose level
Homeostasis
Normal blood glucose levels (<110 mg per dl)
Low blood glucose level
Stimulus
Cells use or store glucose between meals
Blood glucose levels rise: less glucagon produced
Liver
GLUCAGON
Pancreas
Converts glycogen to glucose
α-cells release glucagon into blood

# Diabetes Melitus



# DM

- **Symptoms:**

**-***polydipsia (thirst)*
*-polyuria,*
*-polyphagia*
*-blurring of vision*
*-weight loss.*

- In its most severe forms, ketoacidosis or a non–ketotic hyperosmolar state may develop and lead to **stupor, coma** and, in absence of effective treatment**, death.**

- Often symptoms are not severe, or may be absent, and consequently hyperglycaemia sufficient to cause pathological and functional changes may be present for a long time before the diagnosis is made.

# DM- Long-term Effects

- progressive development of the specific complications of:
  - *retinopathy with potential blindness,*
  - *nephropathy that may lead to renal failure,*
  - *-and/or neuropathy with risk of foot ulcers, amputation,*
  - *Charcot joints,*
  - *and features of autonomic dysfunction, including sexual dysfunction.*

- People with diabetes are at increased risk of *cardiovascular, peripheral vascular and cerebrovascular disease*.

# Frecvency of DM

- International Diabetes Federation (IDF) data indicate that by the year 2025, the number of people affected will reach 333 million –90% of these people will have Type 2 diabetes.
- In most Western societies, the overall prevalence has reached 4-6%, and is as high as 10-12% among 60-70-year-old people.
- The annual health costs caused by diabetes and its complications account for around 6-12% of all health-care expenditure.

# Classification of DM

- Type 1 Diabetes Mellitus
- Type 2 Diabetes Mellitus
- Gestational Diabetes
- Other types:
  - -LADA (Latent Autoimmune Diabetes in Adults
  - -MODY (maturity-onset diabetes of youth)
  - -Secondary Diabetes Mellitus

**Chart 1 - Etiologic classification of diabetes mellitus**

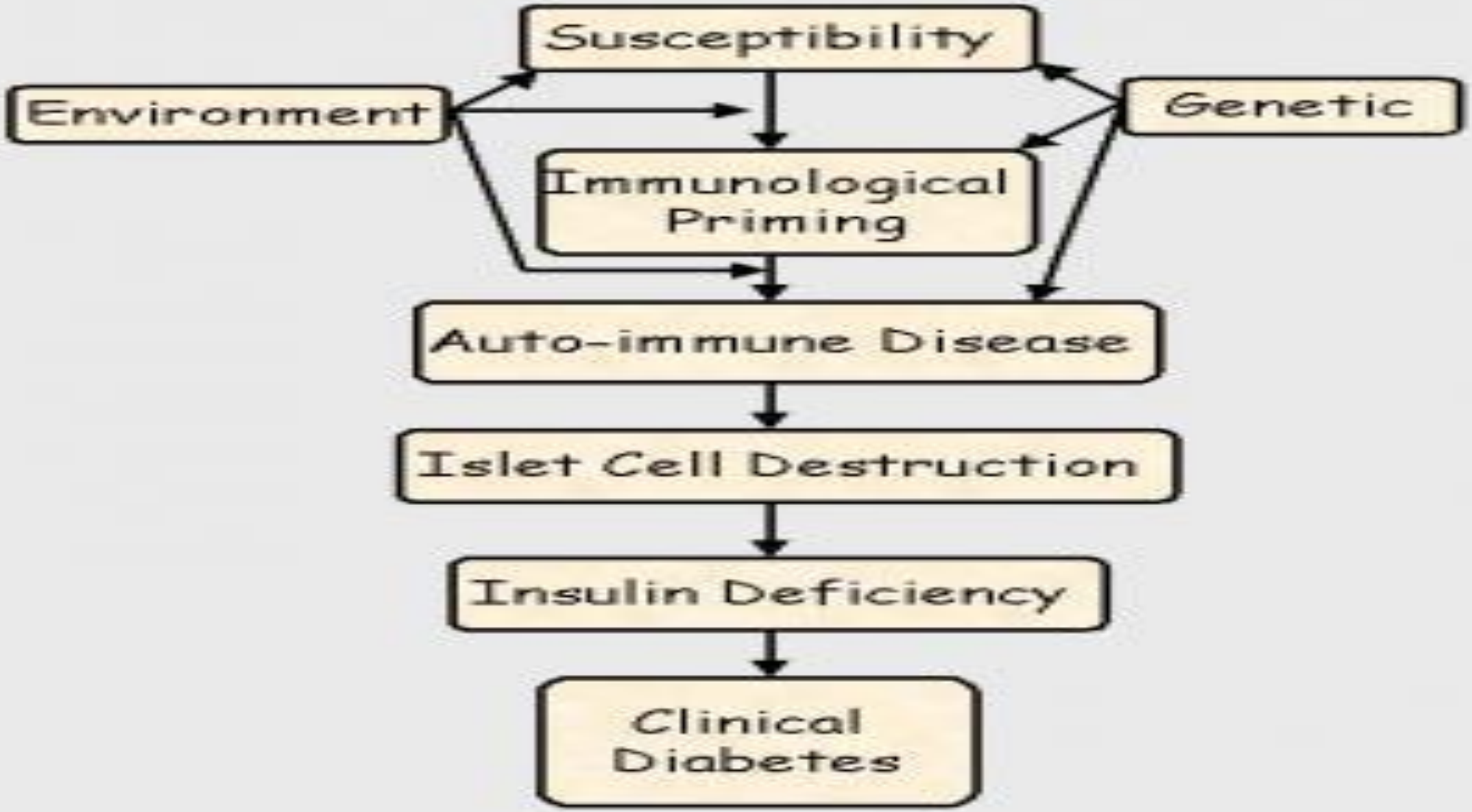
| |
|---|
| I. Type 1 diabetes |
| A. Immunologically mediated |
| B. Idiopathic |
| II. Type 2 diabetes |
| III. Other specific types |
| Genetic disorder of β-cell function (MODY, mitochondrial DNA) |
| Genetic disorders in insulin action (lipoatrophic diabetes) |
| Exocrine pancreas diseases (pancreatitis, hemochromatosis) |
| Endocrinopathies (acromegaly, Cushing's syndrom) |
| Drug-induced (*glucocorticoids*, tiazidics) |
| Infections (cytomegalovirus, congenital rubeola) |
| Uncommon immunological forms (insulin receptor antibodies) |
| Other genetic syndrome (Down, Turner, Prader-Willi syndrom) |
| IV. Gestational diabetes |

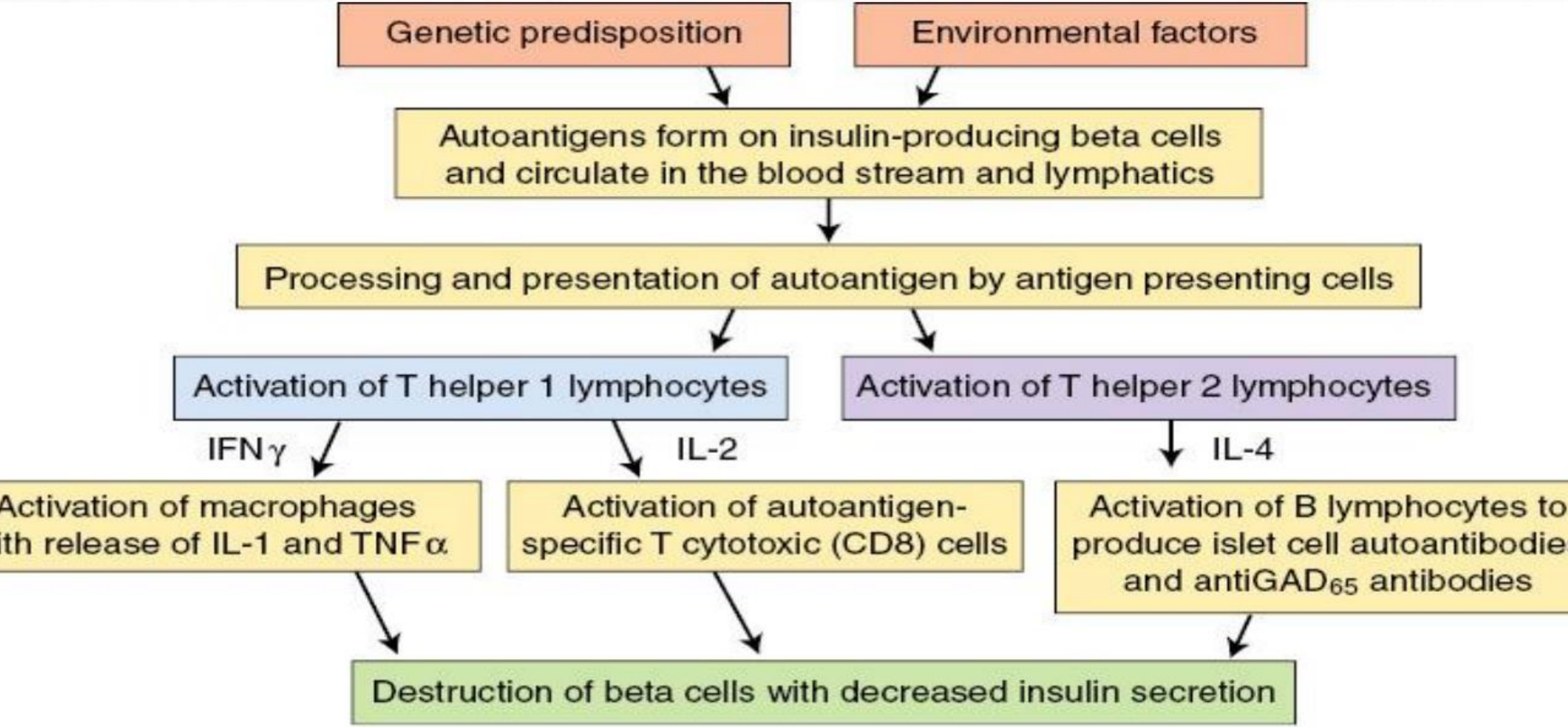
*Source: adapted from American Diabetes Association*[8].

# DM -Type 1

- Type 1 Diabetes Mellitus is a syndrome characterized by hyperglycemia and insulin deficiency resulting from the loss of beta cells in pancreatic islets .
- Type 1 diabetes develops when the body's immune system destroys pancreatic beta cells, the only cells in the body that make the hormone insulin that regulates blood glucose.
- This form of diabetes usually strikes children and young adults, although disease onset can occur at any age.
- Nonimmune (type 1B diabetes), occurs secondary to other diseases and is much less common than autoimmune (type 1A).
- The destruction of beta cells in Type 1A diabetes results from the interaction of both genetic and environmental factors. Although the genetic susceptibility is not well understood, type 1 diabetes is most strongly associated with major histocompatibility complex (MHC), specifically histocompatibility leukocyte antigen (HLA) class II alleles (HLA-DQ and HLA-DR).
- Type 1 diabetes is less hereditary than type 2 but 7-13% of patients also have a first degree relative with type 1 diabetes.
- Environmental factors include viral infections (especially enteroviruses), exposure to infectious microorganisms (such as Helicobacter pylori), exposure to cow's milk proteins and a lack of vitamin D

# Diagram of Possible mechanism for development of Type I diabetes

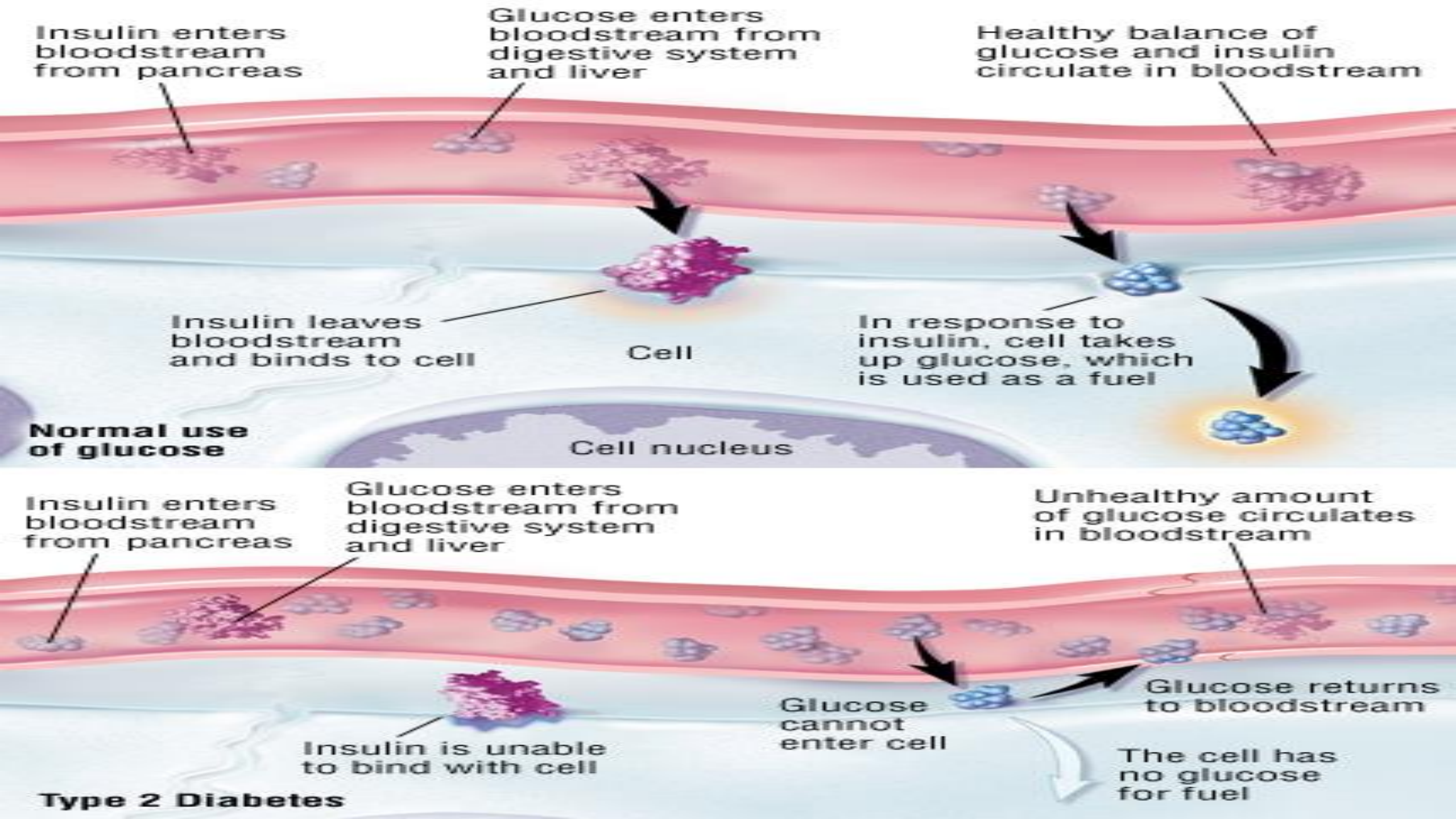


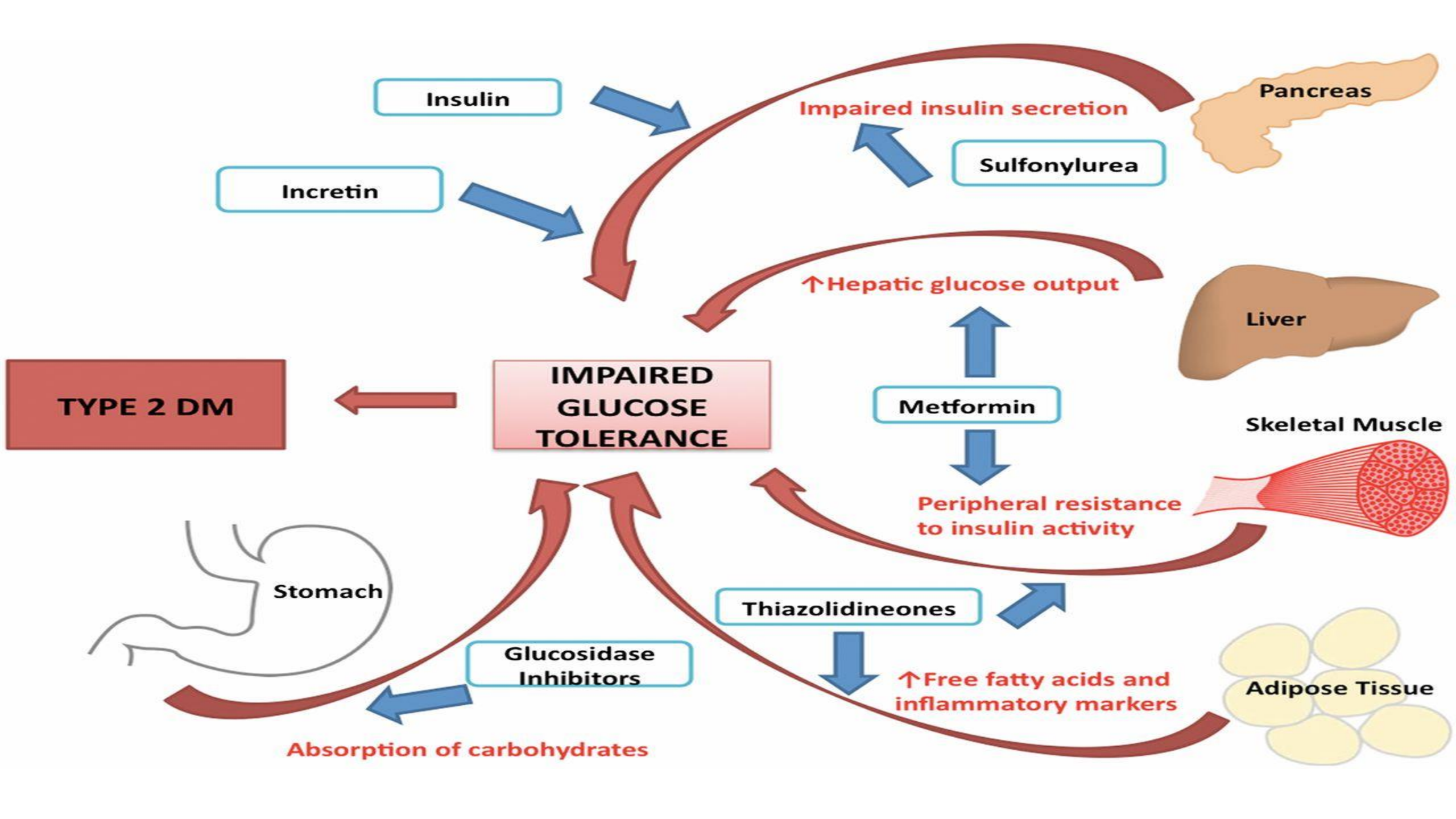


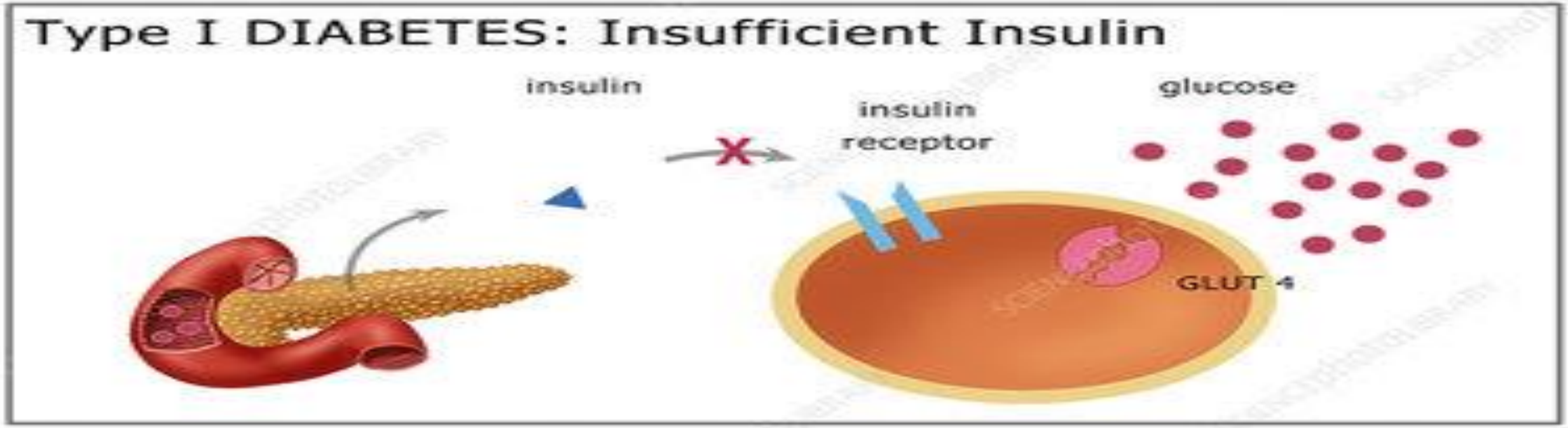

**ure 21-13 Pathophysiology of type 1 diabetes mellitus.** *$GAD_{65}$,* glutamic acid
arboxylase; *INF*-γ Interferon-gamma; *IL,* interleukin; *TNF*-α, tumor necrosis factor-alpha

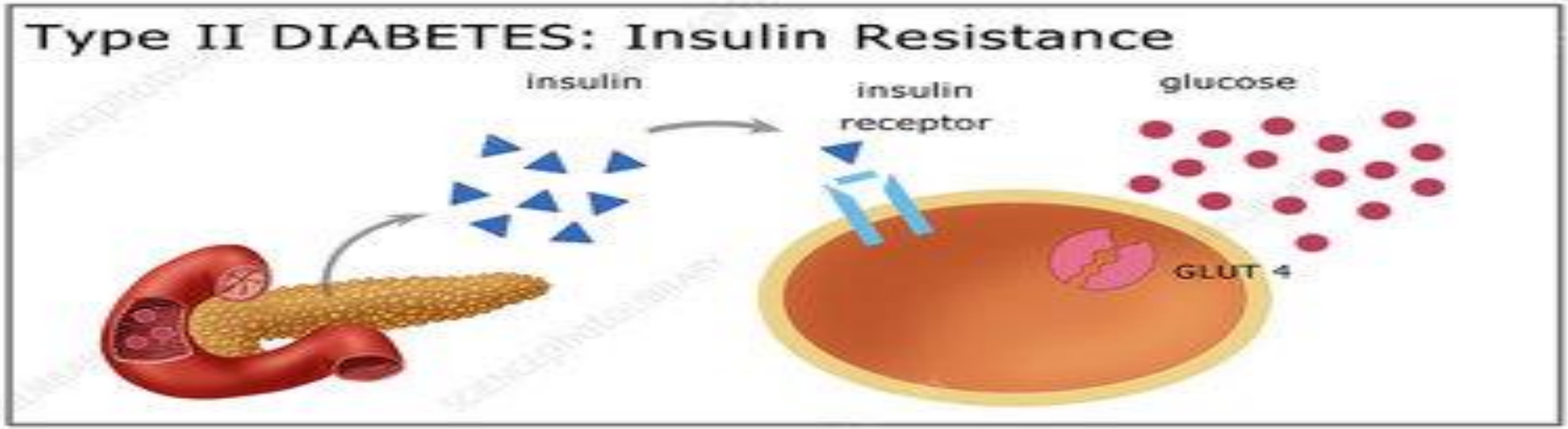
# DM-Type 2

- Was previously called non-insulin-dependent diabetes mellitus (NIDDM) or adult-onset diabetes.
- Type 2 diabetes may account for about 90% to 95% of all diagnosed cases of diabetes.
- It usually begins as insulin resistance, a disorder in which the cells do not use insulin properly. As the need for insulin rises, the pancreas gradually loses its ability to produce insulin.
- Type 2 diabetes is associated with older age, obesity, family history of diabetes, history of gestational diabetes, impaired glucose metabolism, physical inactivity, and race/ethnicity.
- African Americans, Hispanic/Latino Americans, American Indians, and some Asian Americans and Native Hawaiians or Other Pacific Islanders are at particularly high risk for type 2 diabetes.
- Type 2 diabetes is increasingly being diagnosed in children and adolescents.


Insulin enters bloodstream from pancreas
Glucose enters bloodstream from digestive system and liver
Healthy balance of glucose and insulin circulate in bloodstream
Insulin leaves bloodstream and binds to cell
Cell
In response to insulin, cell takes up glucose, which is used as a fuel
Cell nucleus
Normal use of glucose
Insulin enters bloodstream from pancreas
Glucose enters bloodstream from digestive system and liver
Unhealthy amount of glucose circulates in bloodstream
Insulin is unable to bind with cell
Glucose cannot enter cell
Glucose returns to bloodstream
The cell has no glucose for fuel
Type 2 Diabetes


Insulin
Incretin
Impaired insulin secretion
Pancreas
Sulfonylurea
↑Hepatic glucose output
Liver
TYPE 2 DM
IMPAIRED GLUCOSE TOLERANCE
Metformin
Skeletal Muscle
Peripheral resistance to insulin activity
Stomach
Thiazolidineones
Glucosidase Inhibitors
↑Free fatty acids and inflammatory markers
Adipose Tissue
Absorption of carbohydrates


Type I DIABETES: Insufficient Insulin
insulin
insulin receptor
glucose
GLUT 4


Type II DIABETES: Insulin Resistance
insulin
insulin receptor
glucose
GLUT 4

## Difference between insulin-dependent diabetes mellitus (IDDM)/type 1 DM & non-insulin-dependent diabetes mellitus (NIDDM)/type 2 DM

| Properties | Insulin-dependent diabetes mellitus (IDDM) | Non-insulin-dependent diabetes mellitus (NIDDM) |
|---|---|---|
| **Type of diabetes mellitus** | **Type 1**; also known as juvenile-onset diabetes mellitus | **Type 2**; also known as maturity-onset diabetes mellitus |
| **Age of onset** | It usually occurs in children (or adolescents) | It usually occurs in adults of middle age (>35-40 years) |
| **Prevalence** | It accounts for about 10% of all the cases of diabetes | Most common form of diabetes mellitus and accounts for >80% of all the cases of diabetes |
| **Cause of diabetes** | Absolute deficiency of insulin, caused by autoimmune destruction of β-cells | Relative deficiency of insulin, caused by dysfunction of β-cells or due to insulin resistance |

# Gestational diabetes

- A form of glucose intolerance that is diagnosed in some women during pregnancy.
- Gestational diabetes occurs more frequently among African Americans, Hispanic/Latino Americans, and American Indians. It is also more common among obese women and women with a family history of diabetes.
- During pregnancy, gestational diabetes requires treatment to normalize maternal blood glucose levels to avoid complications in the infant.
- After pregnancy, 5% to 10% of women with gestational diabetes are found to have type 2 diabetes.
- Women who have had gestational diabetes have a 20% to 50% chance of developing diabetes in the next 5-10 years.

# Other types of DM

- Can result from specific genetic conditions (such as maturity-onset diabetes of youth), surgery, drugs, malnutrition, infections, and other illnesses.

- Such types of diabetes may account for 1% to 5% of all diagnosed cases of diabetes.

# LADA

- Latent Autoimmune Diabetes in Adults (LADA) is a form of ***autoimmune*** (***type 1 diabetes***) which is diagnosed in individuals who are older than the usual age of onset of type 1 diabetes.
- Alternate terms that have been used for "LADA" include Late-onset Autoimmune Diabetes of Adulthood, "Slow Onset Type 1" diabetes, and sometimes also "Type 1.5
- Often, patients with LADA are mistakenly thought to have ***type 2 diabetes***, based on their age at the time of diagnosis.

# LADA

| Features of LADA |
| --- |
| Patients usually aged ≥ 25 years |
| Clinical presentation "masquerading" as non-obese type 2 diabetes |
| Initial control achieved with diet alone or diet and oral hypoglycaemic agents |
| Insulin dependency occurs within months but can take 10 years or more |
| Other features of type 1 diabetes |
| Low fasting and post-glucagon stimulated C-peptide |
| HLA susceptibility alleles |
| ICA+ |
| GADA+ |

# LADA

- About 80% of adults apparently with recently diagnosed Type 2 diabetes but with GAD auto-antibodies (i.e. LADA) progress to insulin requirement within 6 years.

- The potential value of identifying this group at high risk of progression to insulin dependence includes:
  - the avoidance of using metformin treatment
  - the early introduction of insulin therapy

# MODY

- MODY – Maturity Onset Diabetes of the Young
- MODY is a monogenic form of diabetes with an autosomal dominant mode of inheritance:
  - Mutations in any one of several transcription factors or in the enzyme glucokinase lead to insufficient insulin release from pancreatic ß-cells, causing MODY.
  - Different subtypes of MODY are identified based on the mutated gene.
- Originally, diagnosis of MODY was based on presence of non-ketotic hyperglycemia in adolescents or young adults in conjunction with a family history of diabetes.
- However, genetic testing has shown that MODY can occur at any age and that a family history of diabetes is not always obvious.

# MODY

- Within MODY, the different subtypes can essentially be divided into 2 distinct groups: glucokinase MODY and transcription factor MODY, distinguished by characteristic phenotypic features and pattern on oral glucose tolerance testing.

- Glucokinase MODY requires no treatment, while transcription factor MODY (i.e. Hepatocyte nuclear factor -1alpha) requires low-dose sulfonylurea therapy and PNDM (caused by Kir6.2 mutation) requires high-dose sulfonylurea therapy.

# Secondary DM

Secondary causes of Diabetes mellitus include:

- Acromegaly,
- Cushing syndrome,
- Thyrotoxicosis,
- Pheochromocytoma
- Chronic pancreatitis,
- Cancer
- Drug induced hyperglycemia:
  - Atypical Antipsychotics - Alter receptor binding characteristics, leading to increased insulin resistance.
  - Beta-blockers - Inhibit insulin secretion.
  - Calcium Channel Blockers - Inhibits secretion of insulin by interfering with cytosolic calcium release.
  - Corticosteroids - Cause peripheral insulin resistance and gluconeogensis.
  - Fluoroquinolones - Inhibits insulin secretion by blocking ATP sensitive potassium channels.
  - Naicin - They cause increased insulin resistance due to increased free fatty acid mobilization.
  - Phenothiazines - Inhibit insulin secretion.
  - Protease Inhibitors - Inhibit the conversion of proinsulin to insulin.
  - Thiazide Diuretics - Inhibit insulin secretion due to hypokalemia. They also cause increased insulin resistance due to increased free fatty acid mobilization.

# Prediabetes: Impaired glucose tolerance and impaired fasting glucose

- Progression to diabetes among those with prediabetes is not inevitable. Studies suggest that weight loss and increased physical activity among people with prediabetes prevent or delay diabetes and may return blood glucose levels to normal.

- People with prediabetes are already at increased risk for other adverse health outcomes such as heart disease and stroke.

# Values of Diagnosis of Diabetes Mellitus

**Values for diagnosis of diabetes mellitus and other categories of hyperglycaemia**

| | Glucose concentration, mmol $l^{-1}$ (mg $dl^{-1}$) | | |
|---|---|---|---|
| | Whole blood | | Plasma[a] |
| | Venous | Capillary | Venous |
| **Diabetes Mellitus:** | | | |
| Fasting | ≥ 6.1 (≥ 110) | ≥ 6.1 (≥ 110) | ≥ 7.0 (≥ 126) |
| ***or*** | | | |
| 2-h post glucose load | ≥ 10.0 (≥ 180) | ≥ 11.1 (≥ 200) | ≥ 11.1 (≥ 200) |
| ***or both*** | | | |
| **Impaired Glucose Tolerance (IGT):** | | | |
| Fasting (if measured) | < 6.1 (< 110) | < 6.1 (< 110) | < 7.0 (< 126) |
| ***and*** | | | |
| 2-h post glucose load | ≥ 6.7 (≥ 120) and < 10.0 (< 180) | ≥ 7.8 (≥ 140) and < 11.1 (< 200) | ≥ 7.8 (≥ 140) and < 11.1 (< 200) |
| **Impaired Fasting Glycaemia (IFG):** | | | |
| Fasting | ≥ 5.6 (≥ 100) and < 6.1 (< 110) | ≥ 5.6 (≥ 100) and < 6.1 (< 110) | ≥ 6.1 (≥ 110) and < 7.0 (< 126) |
| ***and*** (if measured) | | | |
| 2-h post glucose load | < 6.7 (< 120) | < 7.8 (< 140) | < 7.8 (< 140) |

# Complications of diabetes mellitus

## A- ACUTE COMPLICATIONS

1. Hypoglycemia
2. Diabetic Ketoacidosis
3. Non-ketotic Hyperosmolar Diabetic Syndrome or Coma
4. Lactic Acidosis

## B- CHRONIC COMPLICATIONS

### 1- Microvascular;

- Nephropathy
- Retinopathy
- Neuropathy

### 2- Macrovascular;

- Coronary artery disease
- Cerebrovascular disease
- Peripheral arterial disease

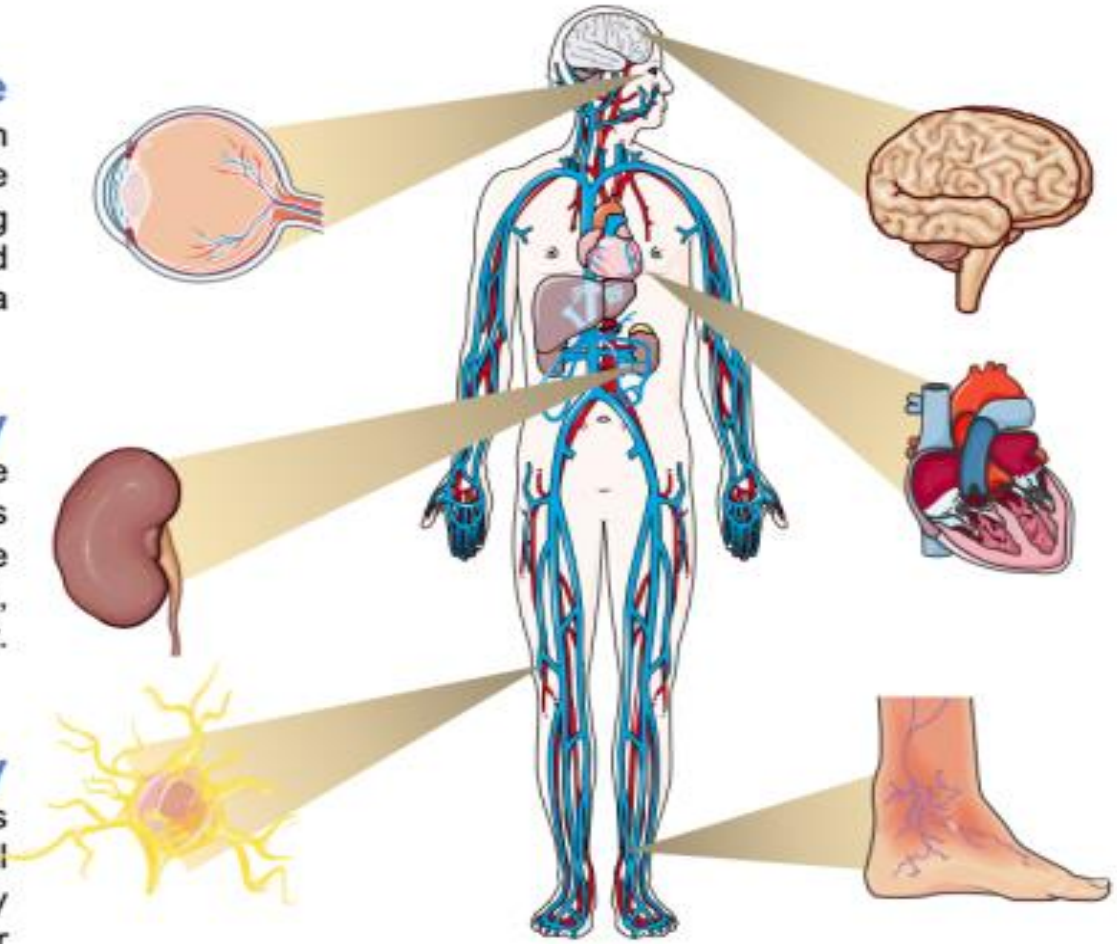
# Major Complications of Diabetes

## Microvascular

### Eye
High blood glucose and high blood pressure can damage eye blood vessels, causing retinopathy, cataracts and glaucoma

### Kidney
High blood pressure damages small blood vessels and excess blood glucose overworks the kidneys, resulting in nephropathy.

### Neuropathy
Hyperglycemia damages nerves in the peripheral nervous system. This may result in pain and/or numbness. Feet wounds may go undetected, get infected and lead to gangrene.

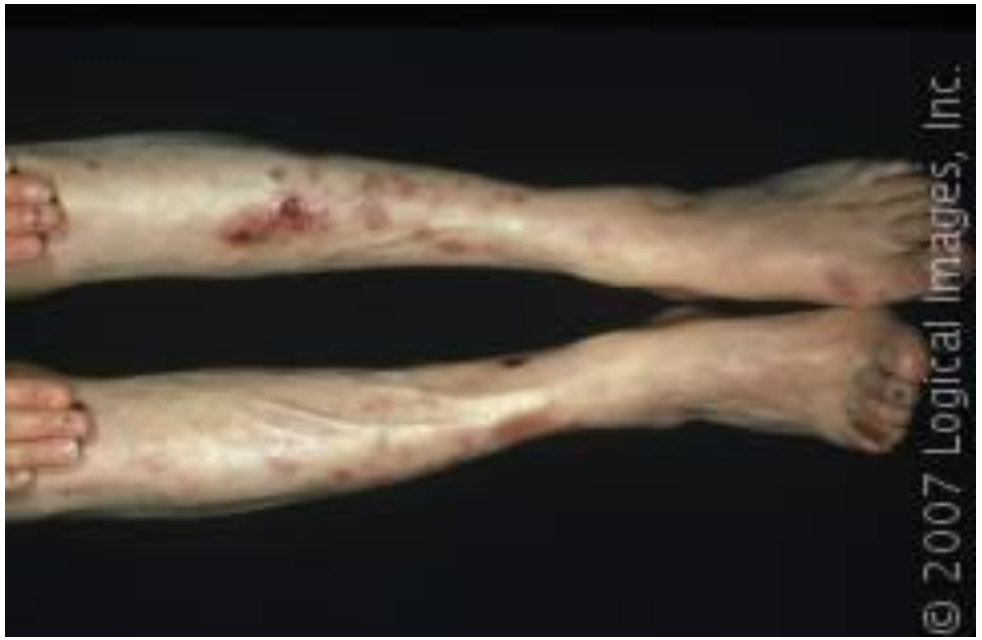


## Macrovascular

### Brain
Increased risk of stroke and cerebrovascular disease, including transient ischemic attack, cognitive impairment, etc.

### Heart
High blood pressure and insulin resistance increase risk of coronary heart disease

### Extremities
Peripheral vascular disease results from narrowing of blood vessels increasing the risk for reduced or lack of blood flow in legs. Feet wounds are likely to heal slowly contributing to gangrene and other complications.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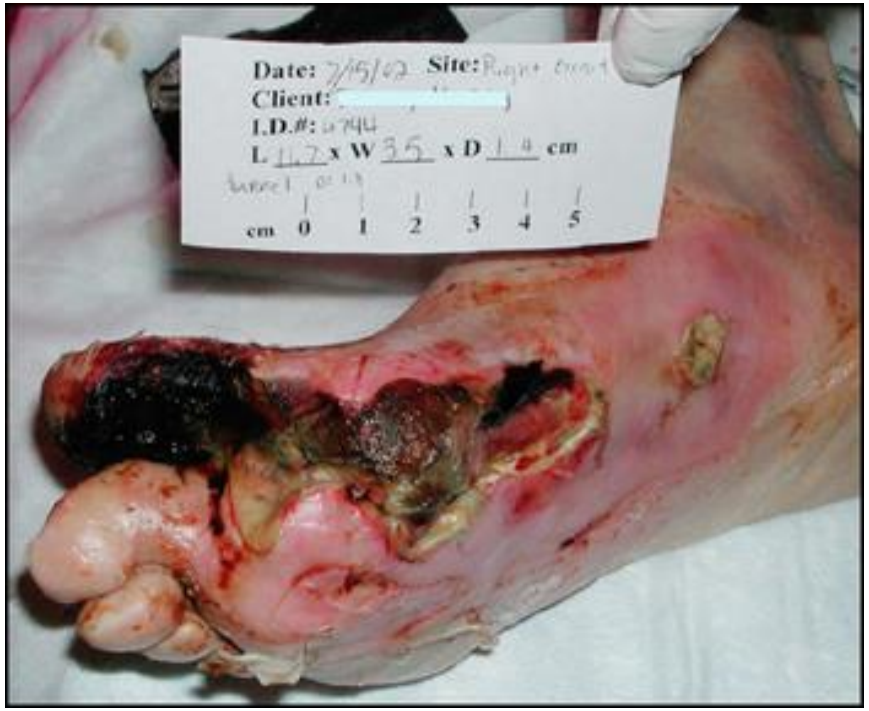
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# Prevention or delay of diabetes:

## Life style modification

- lifestyle changes can prevent or delay the onset of type 2 diabetes among high-risk adults.

- Lifestyle interventions included diet and moderate-intensity physical activity (such as walking for 2 1/2 hours each week).

# Management of DM

- The major components of the treatment of diabetes are:

| | |
|---|---|
| A | • **Diet and Exercise** |
| B | • **Oral hypoglycaemic therapy** |
| C | • **Insulin Therapy** |

# Diabetic coma

# Diabetic coma

- A diabetic coma is a life-threatening diabetes complication that causes unconsciousness.

- Diabetes, dangerously high blood sugar (hyperglycemia) or dangerously low blood sugar (hypoglycemia) can lead to a diabetic coma.

# Symptoms of hyperglycemia

- Before developing a diabetic coma, patients usually experience signs and symptoms of high blood sugar or low blood sugar.:

-Increased thirst

-Frequent urination

-Fatigue

-Nausea and vomiting

-Shortness of breath

-Stomach pain

-Fruity breath odor

-A very dry mouth

-A rapid heartbeat

# Symptoms of hypoglycemia

- Signs and symptoms of a low blood sugar level may include:

-Shakiness or nervousness
-Anxiety
-Fatigue
-Weakness
-Sweating
-Hunger
-Nausea
-Dizziness or lightheadedness
-Difficulty speaking
-Confusion

# Risk factors for diabetic coma

Anyone who has diabetes is at risk of a diabetic coma, but the following factors can increase the risk:

- **Insulin delivery problems.** If you're on an insulin pump, you have to check your blood sugar frequently. Insulin delivery can stop if the pump fails or the tubing (catheter) is twisted or falls out of place. A lack of insulin can lead to diabetic ketoacidosis.
- **An illness, trauma or surgery.** When you're sick or injured, blood sugar levels tend to rise, sometimes dramatically. This may cause diabetic ketoacidosis if you have type 1 diabetes and don't increase your insulin dosage to compensate.
- Medical conditions, such as congestive heart failure or kidney disease, also may increase your risk of diabetic hyperosmolar syndrome.
- **Poorly managed diabetes.** If you don't monitor your blood sugar properly or take your medications as directed, you'll have a higher risk of developing long-term complications and a diabetic coma.

# Risk factors for diabetic coma

- **Deliberately skipping meals or insulin**. Sometimes, people with diabetes who also have an eating disorder choose not to use their insulin as directed with the hope of losing weight. This is a dangerous, life-threatening practice that increases the risk of a diabetic coma.
- **Drinking alcohol.** Alcohol can have unpredictable effects on your blood sugar. Alcohol's sedating effects may make it harder for you to know when you're having low blood sugar symptoms. This can increase your risk of a diabetic coma caused by hypoglycemia.
- **Illegal drug use.** Illegal drugs, such as cocaine and ecstasy, can increase your risk of severe high blood sugar levels and conditions linked to diabetic coma.

# Complications of diabetic coma

- Left untreated, a diabetic coma can lead to:

-**Permanent brain damage**

**-Death**

# Treatment of diabetic coma

- Diabetic coma requires emergency medical treatment. The type of treatment depends on whether  blood sugar level is too high or too low.
- test the unconscious person's blood sugar and follow these steps:

- **High blood sugar:**

-Insulin to help your tissues absorb the glucose in your blood

-Intravenous fluids to restore water to your tissues

-Potassium, sodium or phosphate supplements to help your cells function correctly

-Treatment for any underlying infections

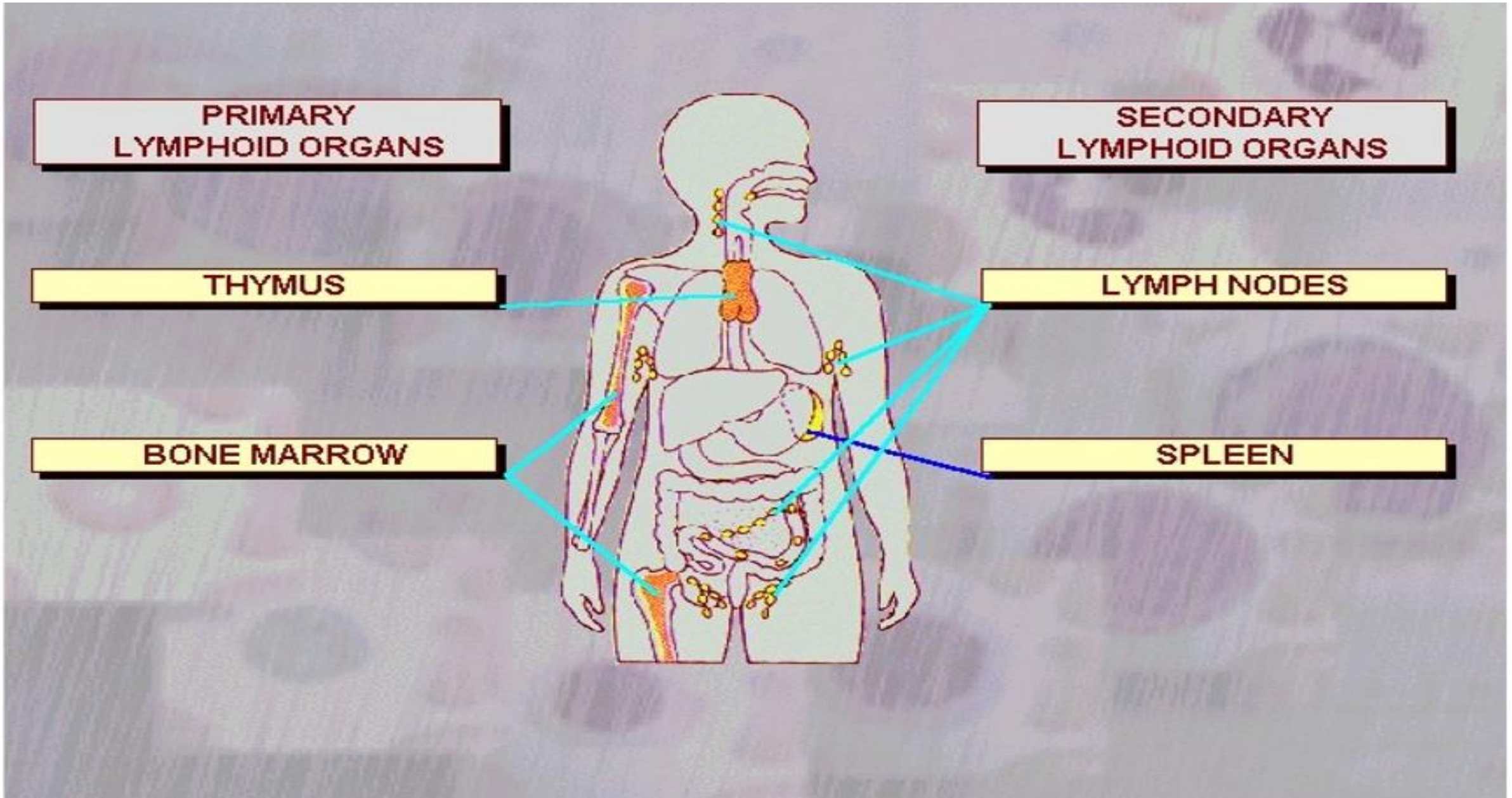
- **Low blood sugar: (It’s more dangerous)**

-Can be given a glucagon injection, which will cause  blood sugar level to quickly rise.

-Intravenous dextrose also may be given to raise blood glucose levels.

# Particularities of clinical and laboratory examination in anemia.

# hematopoietic system



# Anemia

- It is a manifestaton of a certain disease associated with:

-Decreased in Red Blood Cells

-Decreased in Hematocrit

- Decreased in Hemoglobin

# GENERAL CAUSES OF ANEMIA

- 1. Decreased Red Blood Cell Producton

-Associated with Bone Marrow

-and Kidney Problems

- 2. Increased Red Blood Cell Destructon

-Primary cause is Hemolysis (Hemolytc Anemia)

- 3. Blood Loss

-Accident

-Giving Birth

# Anemia Due to Decrease in RBC Producton

- Iron Defciency Anemia (IDA)
- Anemia of Chronic Infammaton (ACI)
- Sideroblastc Anemia (SA)
- Megaloblastc Anemia (MA)
- Aplastc Anemia (AA)
- Thalassemia
- Anemia due to Chronic Renal Failure
- Anemia due to Chronic Endocrine
- Disorder (Cushing Syndrome, Addison’s Disease)
- Anemia due to Marrow Infltra□on

# Anemia Due to Increase Destructon of RBC

A. Intracorpuscular Abnormality

- 1. Membrane Defect (Spectrin, Ankidin and Protein 4.1)

-Hereditary Spherocytosis

-Hereditary Elliptocytosis

-Hereditary Pyropoikilocytosis – Abnormal sensitvity to heat (severe Elliptocytosis).

-Hereditary Stomatocytosis

-Hereditary Acanthocytosis

-Hereditary Rh Null Disease

# Anemia Due to Increase Destruction of RBC

2. Enzyme Defciency
-G6PD Defciency – decrease in Hgb, eatng of beans, soya, Fava beans.
-Pyruvate Kinase Defciency –
-survival of the RBC
-Porphyria – Heme synthesis

- 3. Paroxysmal Nocturnal Hemoglobinuria (PNH)

- 4. Globin Abnormality
- Hemoglobinopathies(Hb SS, CC, SC)

# Anemia Due to Increase Destruction of RBC

B. Extracorpuscular Abnormality

- 1. Mechanical

-Microangiopathic Hemolytic

-Anemia (MAHA)

-Thrombotc

-Thrombocytopenic

-Purpura (TTP) – decreased in platelets

-Hemolytc Uremic

-Syndrome (HUS) – caused by 0157H:7 serotype of E. coli

-Traumatc Cardiac Hemolytc anemia

# Anemia Due to Increase Destruction of RBC

2. Infecton
-Hemolytic Anemia
-Malaria
-Babesia
-Bartonella
-Ehrlichia

- 3. Chemical and Physical Agents
- Caused by drugs, toxins, burns

- 4. Antbody – Mediated Anemia

-Acquired Hemolitic Anemia

# Anemia due to Blood Loss

- 1. Acute Post Hemorrhagic Anemia
- 2. Chronic Post Hemorrhagic Anemia

# CLINICAL FINDINGS OF ANEMIA

- 1. History
- 2. Physical Examinaton
- 3. Signs and Symptoms
- 4. Laboratory Procedures

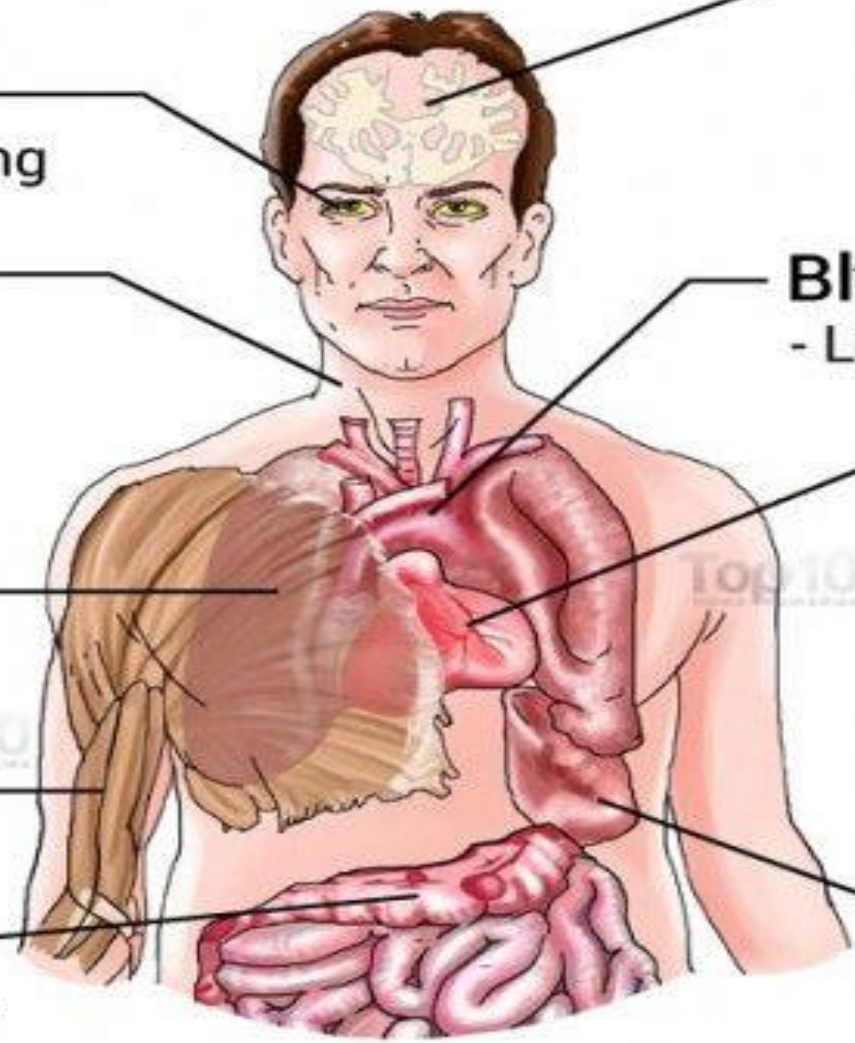
-CBC

-Iron Studies

-Hemoglobin Electrophoresis


# Symptoms of Anemia

Red = In severe anemia



**Central**
- Fatigue
- Dizziness
- Fainting

**Eyes**
- Yellowing

**Skin**
- Paleness
- Coldness
- Yellowing

**Blood vessels**
- Low blood pressure

**Heart**
- Palpitations
- Rapid heart rate
- Chest pain
- Angina
- Heart attack

**Respiratory**
- Shortness of breath

**Muscular**
- Weakness

**Spleen**
- Enlargement

**Intestinal**
- Changed stool color

# Common Symptoms of Anemia

- Shortness of Breath
- Fatigue

# History of Patent

- Diet
- Bleeding History
- Drug Ingeston
- Occupaton
- Exposure to chemicals
- Travels
- Previous Medicatons
- Ethnic Groups
- Family History of Disease
- Hobbies
- Neurologic Symptoms

# Physical Examinaton

- **Skin**

-Pallor (hgb) , Jaundice

-(hemolysis) and Petechiae (breakage of the capillaries

- Eyes (Hemorrhage)
- Mouth (Mucosal Bleeding)
- Sternal Tenderness
- Lymphadenopathy
- Cardiac murmurs
- Splenomegaly
- Hepatomegaly
- Vital Signs
- Temperature, Blood Pressure
- and Heart Rate

# Evaluating Anemia: Physical Examination:

- Mouth: bruises, gum swelling, glossitis (vitB12, iron), angular stomatitis (iron def), pharyngitis (infection), ulceration/mucositis (white cell disorders).
- Neck: lymph nodes (infection, infiltration), thyroid exam.
- Skin: petechiae, purpura, pallor, jaundice, café au lait macules.
- Hands: dactylitis (SCD), bone deformities (marrow failure syndromes).

# Clinical Features of Iron Deficiency:

- Smooth Tongue – Iron defciency can result in a painless, smooth, shiny and reddened tongue

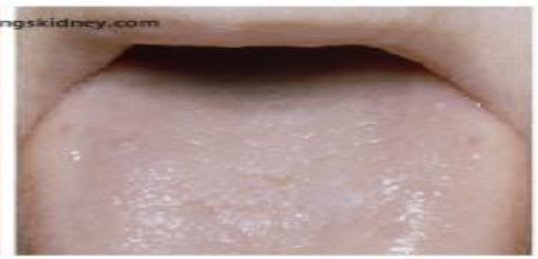
- Koilonychia – a conditon also referred to as “spoon – shaped nails” is associated with iron deficiency in which the fingernails are thin, briAle and concave with raise edge.
- Pica – conditon where in there’s craving for uncertain food

# PHYSICAL EXAMINATION TO DIAGNOSE ANEMIA

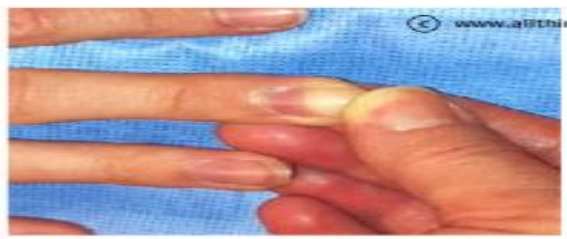

NO PALLOR


PALLOR IN ANEMIA

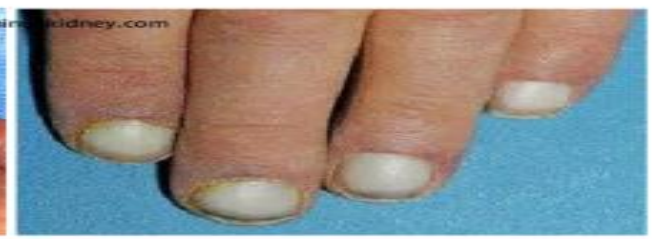

NO PALLOR




PALLOR IN ANEMIA

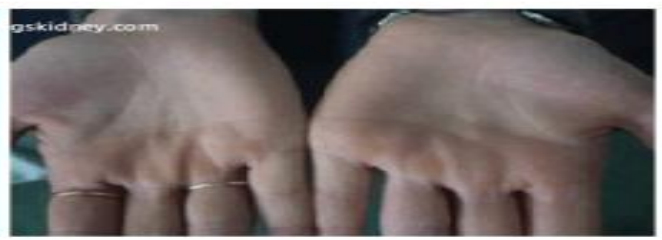



HEALTHY, PINK NAIL BED
PALES ON PRESSURE

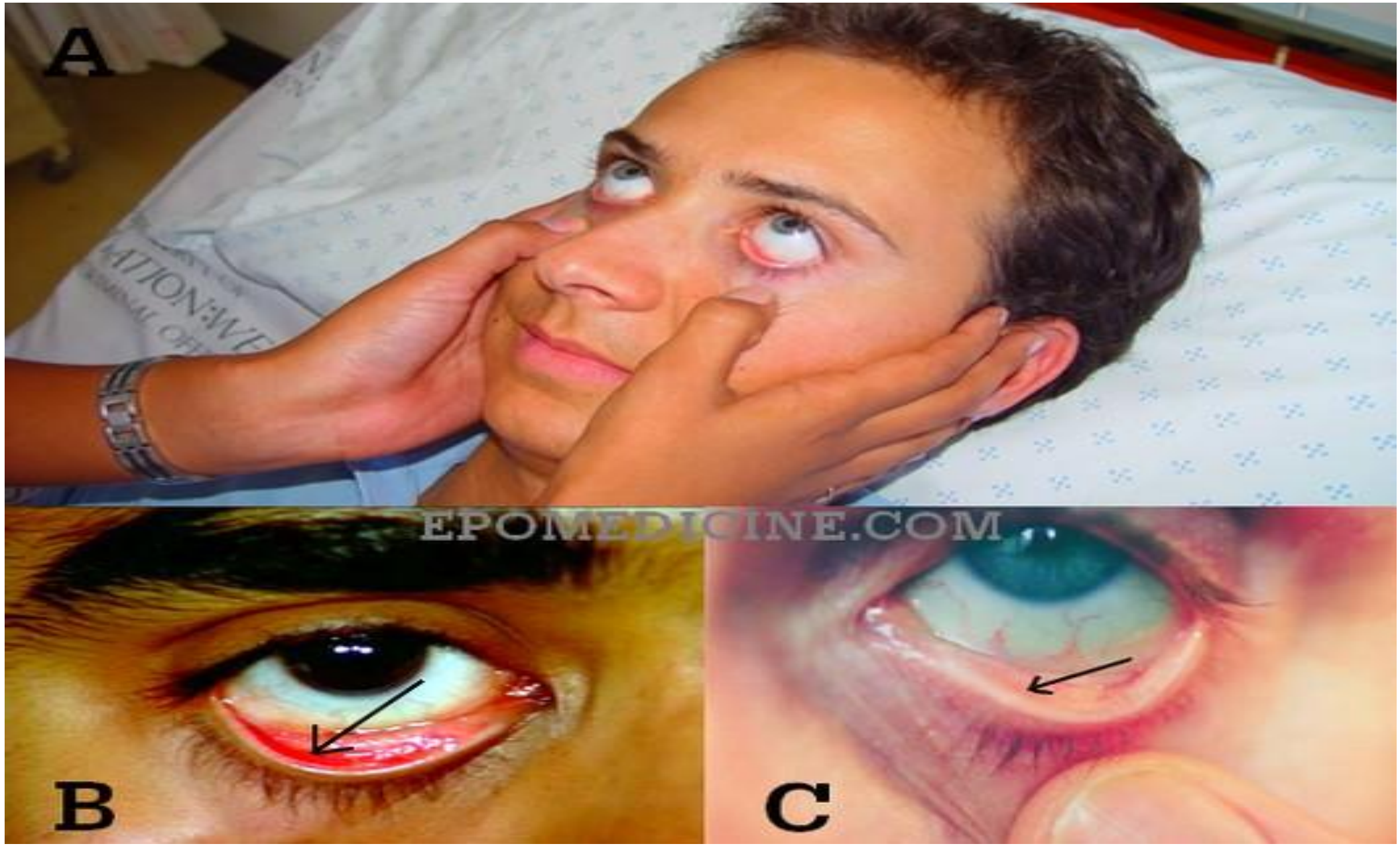



PALE NAIL-BEDS IN ANEMIA

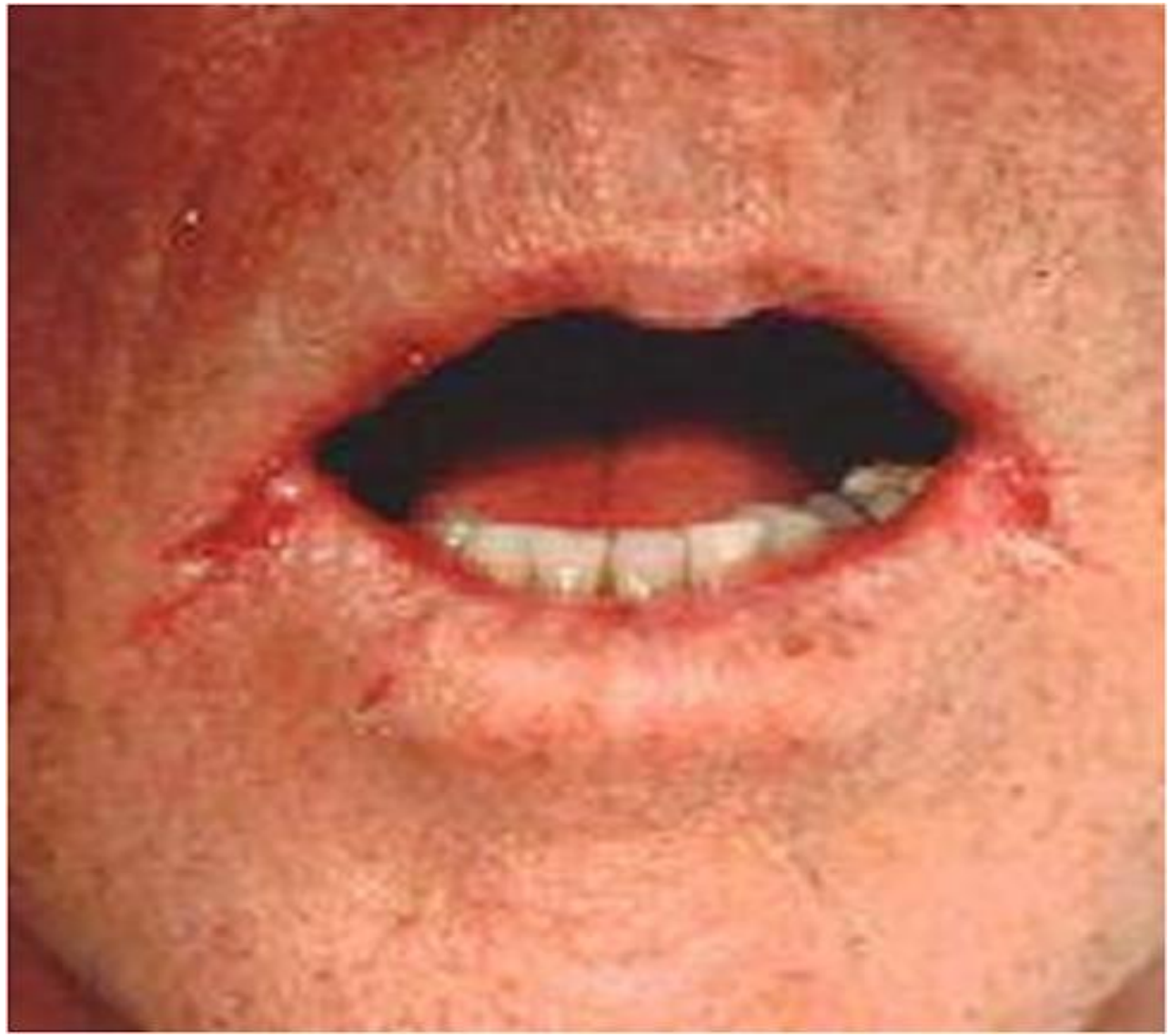

PROMINENT CREASES ON THE
PALM IN GOOD HEALTH




PALE PALMAR CREASES IN ANEMIA


A
EPOMEDICINE.COM
B
C

# Severe iron deficiency



# Signs of iron deficiency anemia

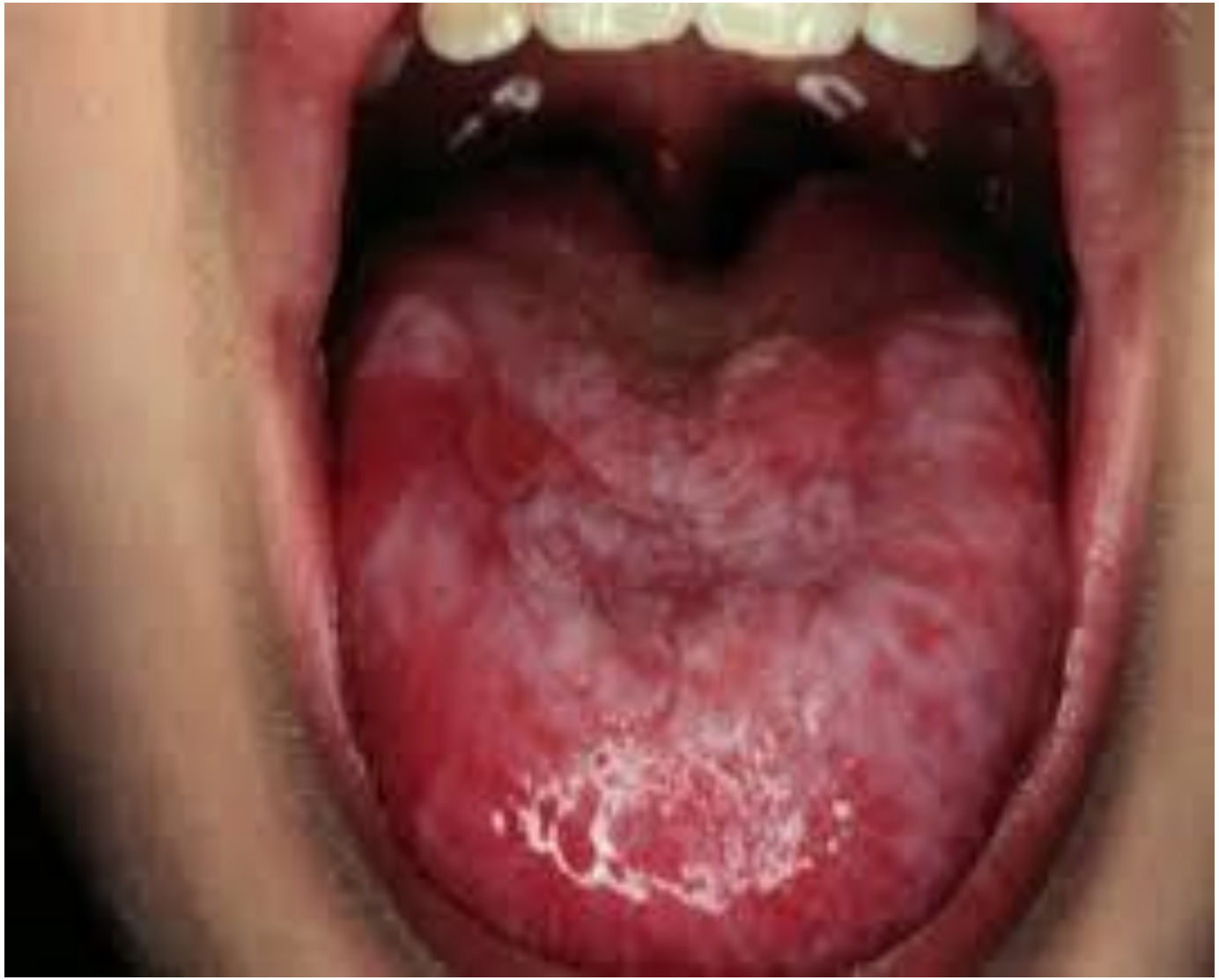
- Pallor

- Glossitis

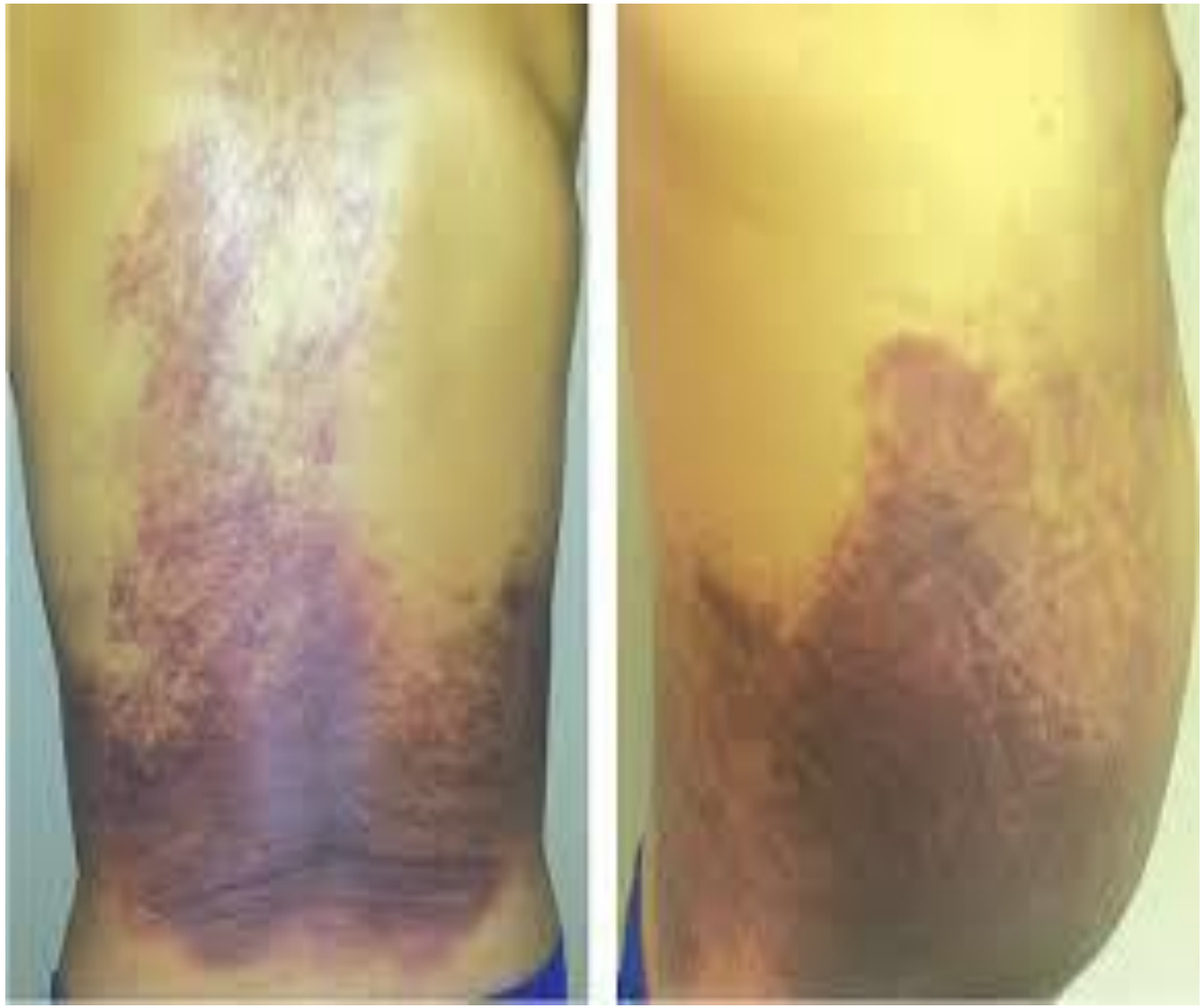

Anemia


Normal blood

# Aplastic anemia



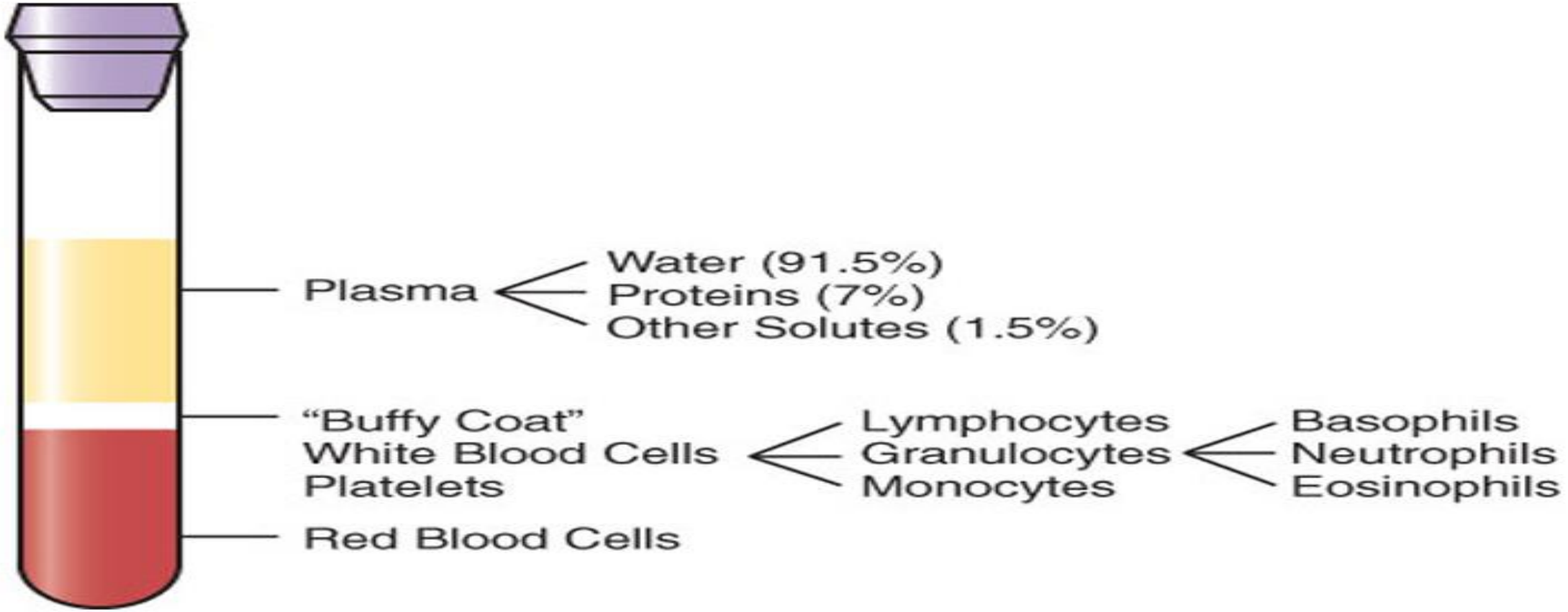
# Hemolitic anemia

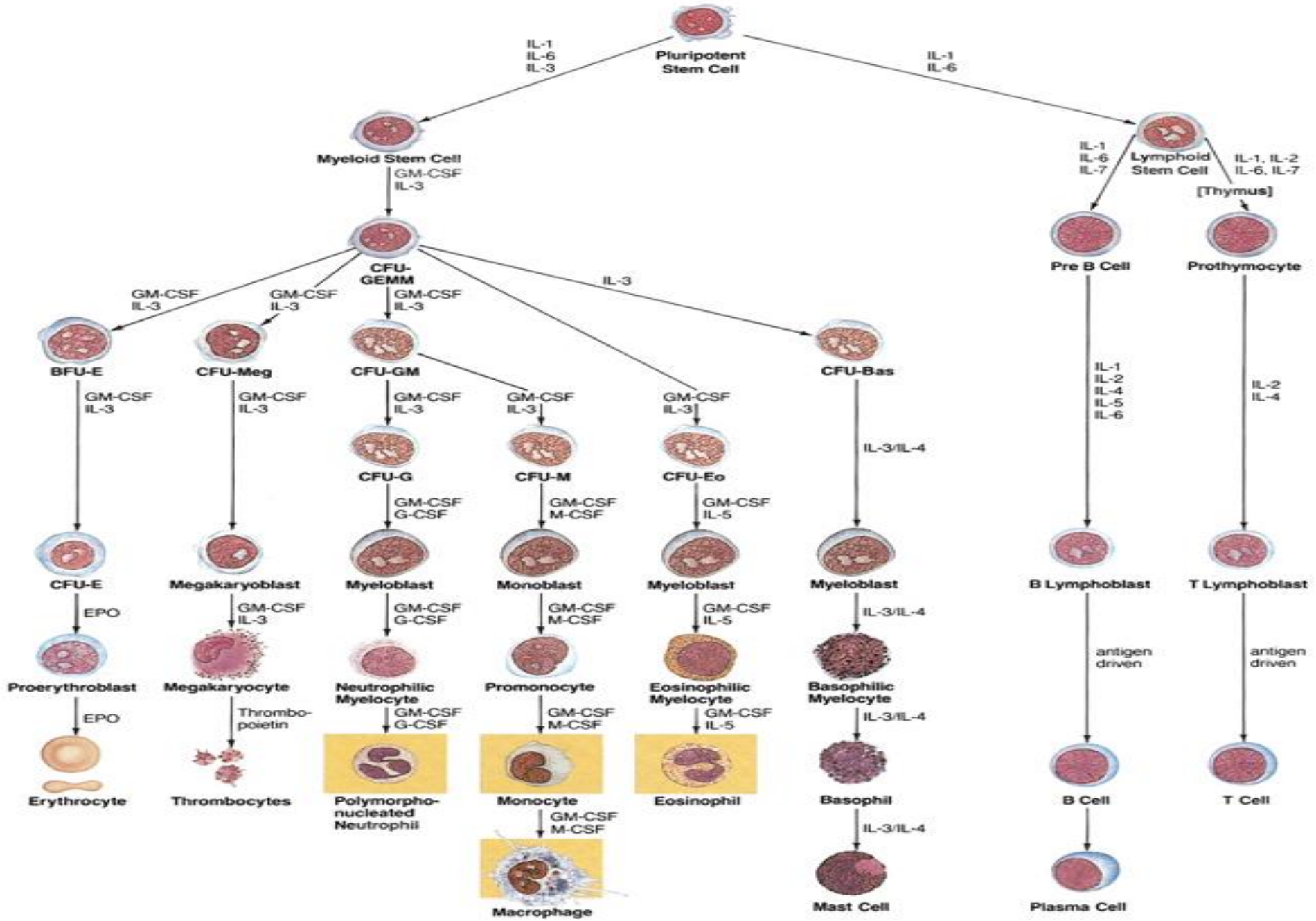


# Laboratory Test for Anemia Assessment

- Complete Blood Count
- Retculocyte Count
- Peripheral Smear
- Bone Marrow Examinaton – used when the primary tests are doub:ul.
- Iron Studies – Backbone test for Assessing Anemia; Serum Iron, Serum Ferritn, Total Iron – Binding Capacity
- Blood Chemistry (Kidney Function Test,
- Liver Functon Test)
- Urinalysis
- Fecalysis – for occult blood
- Hematological Special Test Procedures

# COMPOSITION OF BLOOD






Thank you