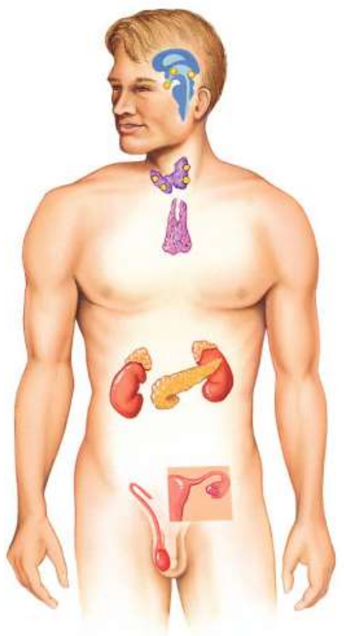
GENERAL ANATOMY OF THE ENDOCRINE SYSTEM

THERE ARE TWO MAIN COMMUNICATION SYSTEMS IN THE BODY:



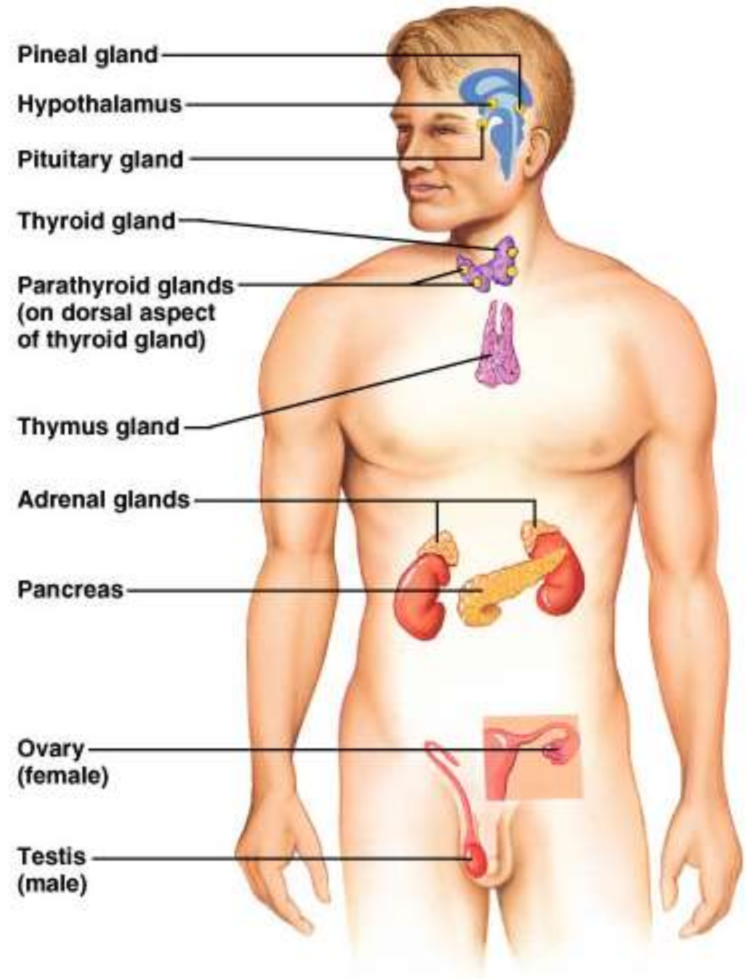
- **NERVOUS SYSTEM**

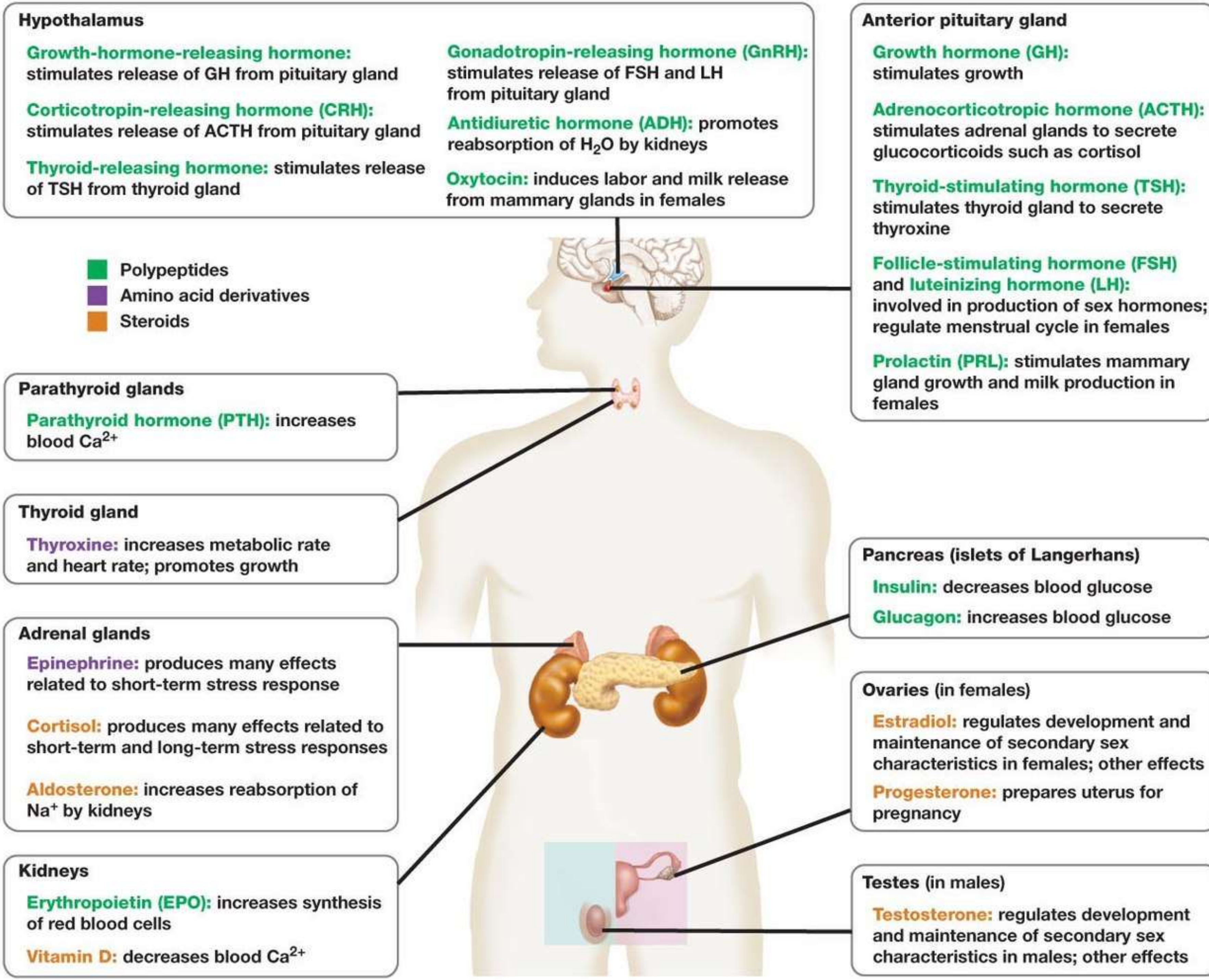
- **ENDOCRINE SYSTEM**

ENDOCRINOLOGY – STUDY OF HORMONES AND ENDOCRINE GLANDS.

ENDOCRINE ORGANS

- Endocrine organs:
  - Pituitary gland
  - Pineal gland
  - Thyroid gland
  - Parathyroid glands
  - Adrenal: 2 glands
    - Cortex
    - Medulla
- Endocrine cells in other organs:
  - Pancreas
  - Thymus
  - Gonads
  - Hypothalamus

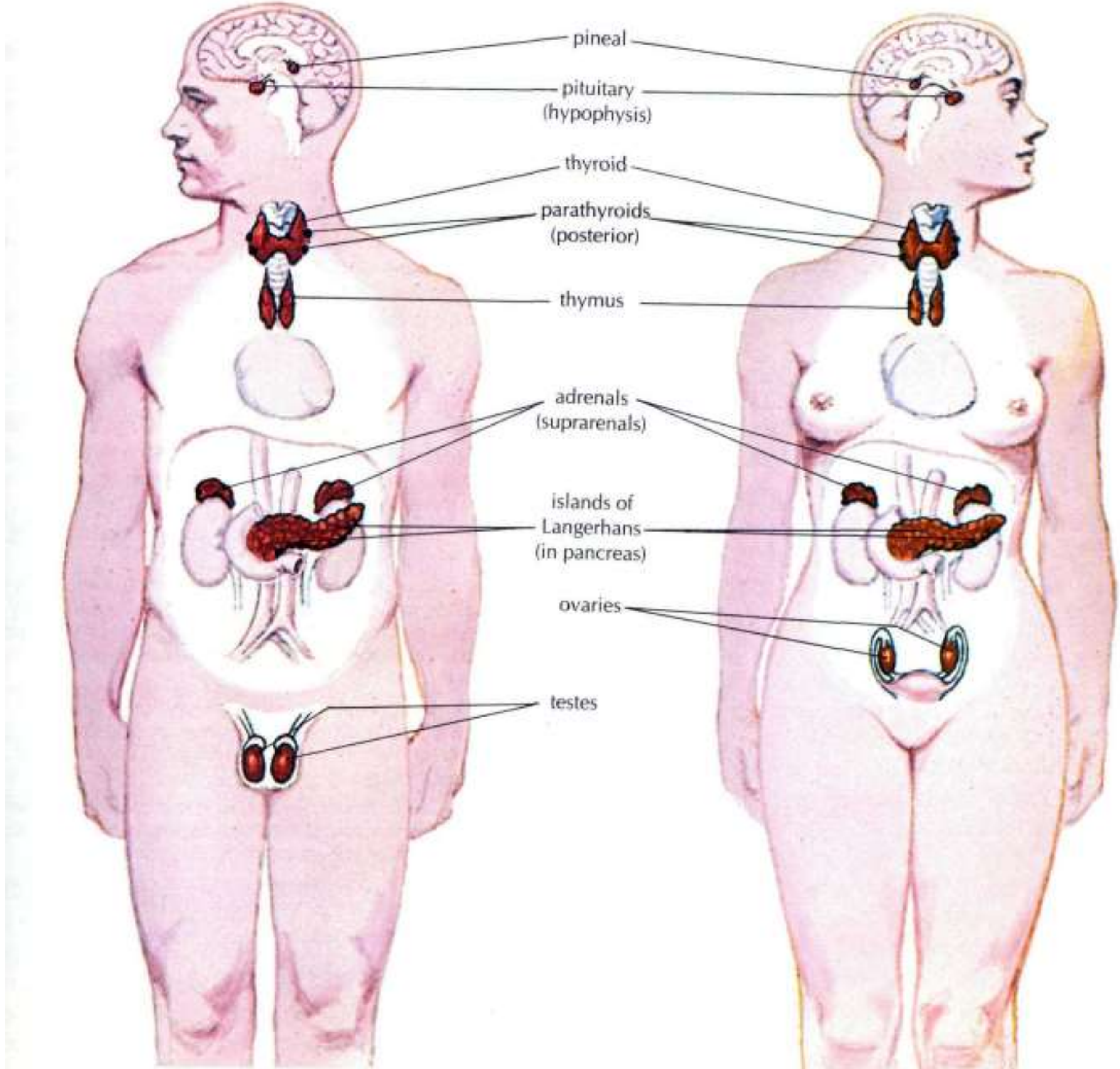


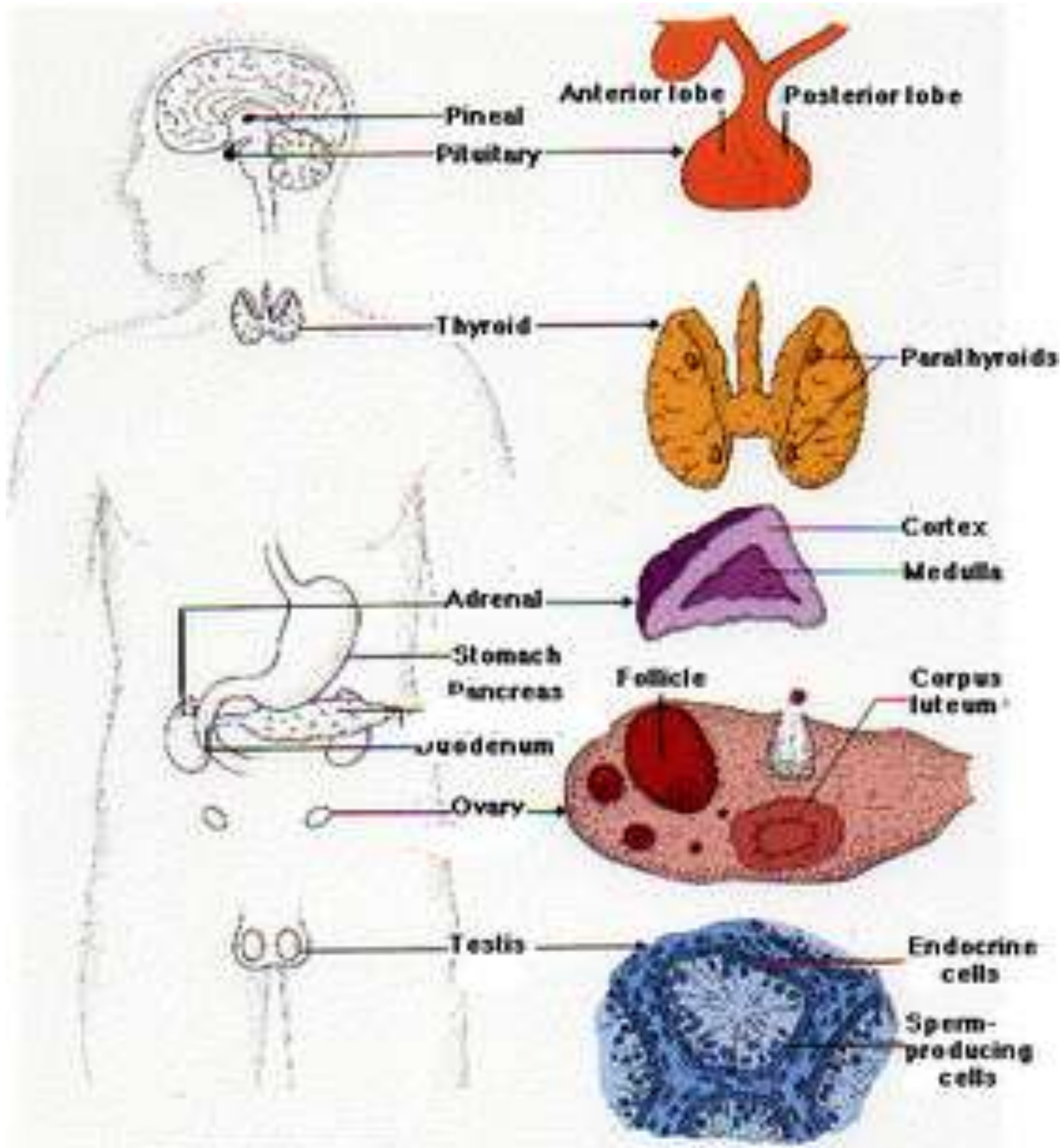

Hypothalamus
Growth-hormone-releasing hormone: stimulates release of GH from pituitary gland
Corticotropin-releasing hormone (CRH): stimulates release of ACTH from pituitary gland
Thyroid-releasing hormone: stimulates release of TSH from thyroid gland
Gonadotropin-releasing hormone (GnRH): stimulates release of FSH and LH from pituitary gland
Antidiuretic hormone (ADH): promotes reabsorption of H_2O by kidneys
Oxytocin: induces labor and milk release from mammary glands in females
Polypeptides
Amino acid derivatives
Steroids
Anterior pituitary gland
Growth hormone (GH): stimulates growth
Adrenocorticotropic hormone (ACTH): stimulates adrenal glands to secrete glucocorticoids such as cortisol
Thyroid-stimulating hormone (TSH): stimulates thyroid gland to secrete thyroxine
Follicle-stimulating hormone (FSH) and luteinizing hormone (LH): involved in production of sex hormones; regulate menstrual cycle in females
Prolactin (PRL): stimulates mammary gland growth and milk production in females
Parathyroid glands
Parathyroid hormone (PTH): increases blood Ca^{2+}
Thyroid gland
Thyroxine: increases metabolic rate and heart rate; promotes growth
Adrenal glands
Epinephrine: produces many effects related to short-term stress response
Cortisol: produces many effects related to short-term and long-term stress responses
Aldosterone: increases reabsorption of Na^+ by kidneys
Kidneys
Erythropoietin (EPO): increases synthesis of red blood cells
Vitamin D: decreases blood Ca^{2+}
Pancreas (islets of Langerhans)
Insulin: decreases blood glucose
Glucagon: increases blood glucose
Ovaries (in females)
Estradiol: regulates development and maintenance of secondary sex characteristics in females; other effects
Progesterone: prepares uterus for pregnancy
Testes (in males)
Testosterone: regulates development and maintenance of secondary sex characteristics in males; other effects

THE ENDOCRINE SYSTEM IS THE CENTER OF HUMORAL REGULATION OF THE BODY. THIS REGULATION OF THE BODY IS MADE BY BIOLOGICALLY ACTIVE SUBSTANCES THAT ARE TRANSPORTED BY THE BLOOD OR LYMPHATIC SYSTEM.

ENDOCRINE GLANDS (GLANDULAE ENDOCRINE) ARE SPECIALIZED ORGANS THAT PRODUCE AND SECRETE INTO THE INTERNAL ENVIRONMENT BIOLOGICALLY ACTIVE SUBSTANCES THAT ARE CONTROL FUNCTIONS OF VARIOUS ORGANS AND SYSTEMS.

Glands of the endocrine system.




Anterior lobe
Posterior lobe
Pineal
Pituitary
Thyroid
Parathyroids
Cortex
Medulla
Adrenal
Stomach
Pancreas
Duodenum
Follicle
Corpus luteum
Ovary
Testis
Endocrine cells
Sperm-producing cells

THE MAIN ANATOMICAL FEATURES OF ENDOCRINE GLANDS ARE THE LACK OF EXCRETORY DUCTS. THIS IS THE MAIN DIFFERENCE BETWEEN THEM AND EXOCRINE GLANDS.

THE SECOND FEATURE OF ENDOCRINE GLANDS IS THAT THEY HAVE EXTREMELY DENSE NETWORK OF BLOOD VESSELS.

ENDOCRINE GLANDS DIFFER IN THEIR DEVELOPMENT – THEY DEVELOP FROM DIFFERENT GERM LAYERS.

CLASSIFICATION ENDOCRINE GLAND ORIGIN:

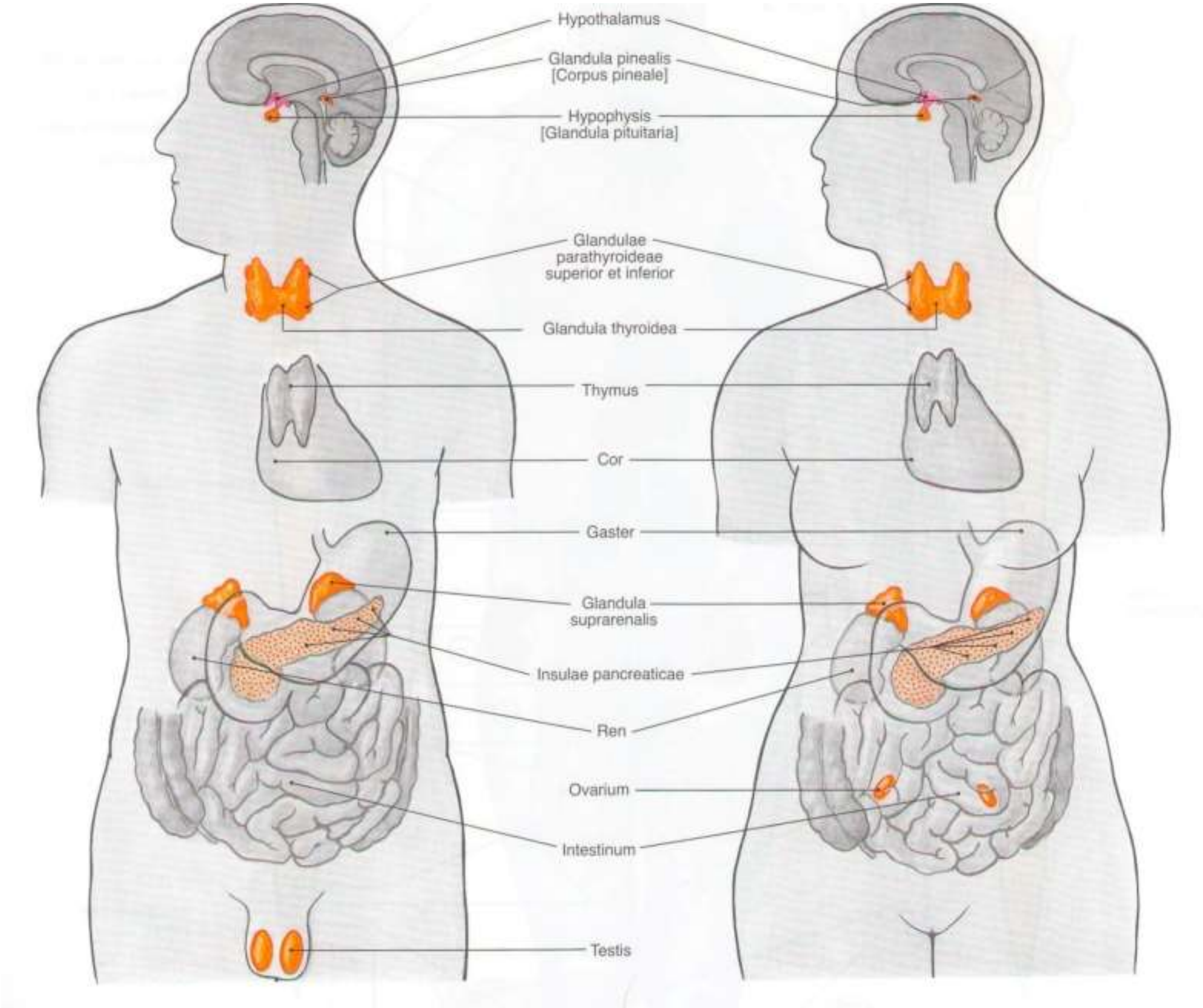
1. **GLANDS ECTODERMAL ORIGIN.**
2. **GLANDS MESODERMAL ORIGIN.**
3. **GLANDS ECTODERMAL ORIGIN.**

ACCORDING TO THE INTERNATIONAL ANATOMIC NOMENCLATURE ENDOCRINE GLANDS (GLANDULAE ENDOCRINE), OR ENDOCRINE GLANDS INCLUDE:

- **THYROID**
- **PARATHYROID GLAND**
- **ADRENAL GLAND**
- **THE ISLETS OF THE PANCREAS**
- **PITUITARY**
- **EPIPHYSIS**

GLANDULAE ENDOCRINAE:

- HYPOPHYSIS
- GLANDULA PINAELIS
- GLANDULA THYROIDEA
- GLANDULAE PARATHYROIDEAE
- GLANDULA SUPRARENALIS
- PARAGANGLIA
- INSULAE PANCREATICAE
- PARS ENDOCRINAE GLANDULAE GENITALES


Hypothalamus
Glandula pinealis
[Corpus pineale]
Hypophysis
[Glandula pituitaria]
Glandulae
parathyroideae
superior et inferior
Glandula thyroidea
Thymus
Cor
Gaster
Glandula
suprarenalis
Insulae pancreaticae
Ren
Ovarium
Intestinum
Testis

- **Endocrine system** jointly with the nervous system mades **body´s communication network**

  - it is composed of **various endocrine glands and endocrine cells**

  - the glands are capable of synthetizing and releasing special chemical messengers - **hormones**

An Overview of the Endocrine System

KEY TO PITUITARY HORMONES

ACTH	Adrenocorticotropic hormone
TSH	Thyroid-stimulating hormone
GH	Growth hormone
PRL	Prolactin
FSH	Follicle-stimulating hormone
LH	Luteinizing hormone
MSH	Melanocyte-stimulating hormone
ADH	Antidiuretic hormone

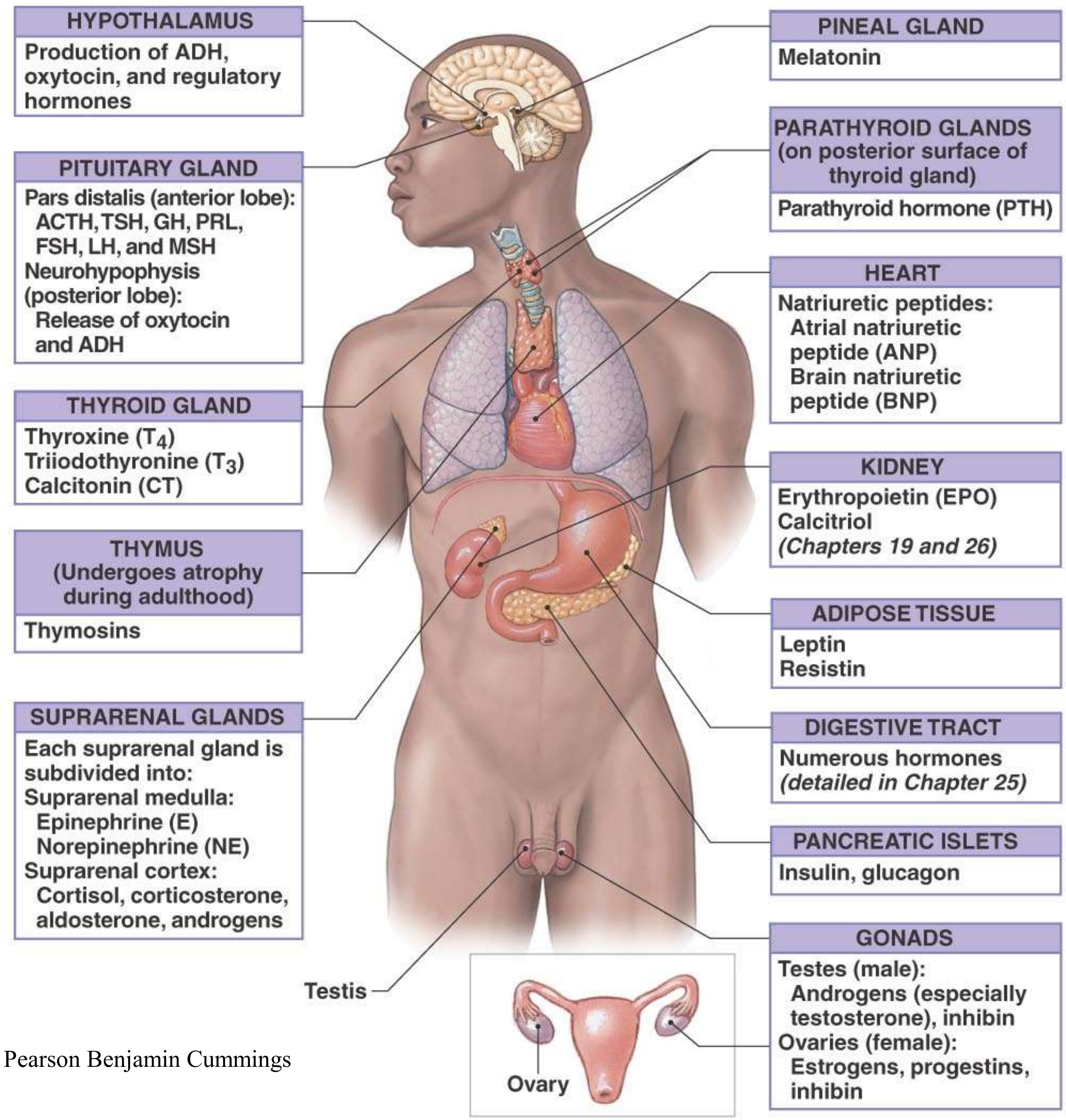


Fig 19.1

HORMONES AFFECT CERTAIN TARGET TISSUES OR ORGANS AND REGULATE THEIR ACTIVITIES.

- **Hormones** - substances which are secreted by **specialised cells** in very **low concentrations** and they are able to influence **secreted cell itself** (autocrine influence), **adjacent cells** (paracrine influence) or **remote cells** (hormonal influence)

THE BODY'S CHEMICAL MESSENGERS (HORMONES) ARE MADE BY ENDOCRINE GLANDS.

These glands have no ducts but secrete their hormones directly into the blood, reaching every cell in the body.

Chemical Classification of Hormones

- Steroid Hormones:
  - –Lipid soluble
  - –Diffuse through cell membranes
  - –Endocrine organs
    - Adrenal cortex
    - Ovaries
    - Testes
    - placenta

Chemical Classification of Hormones

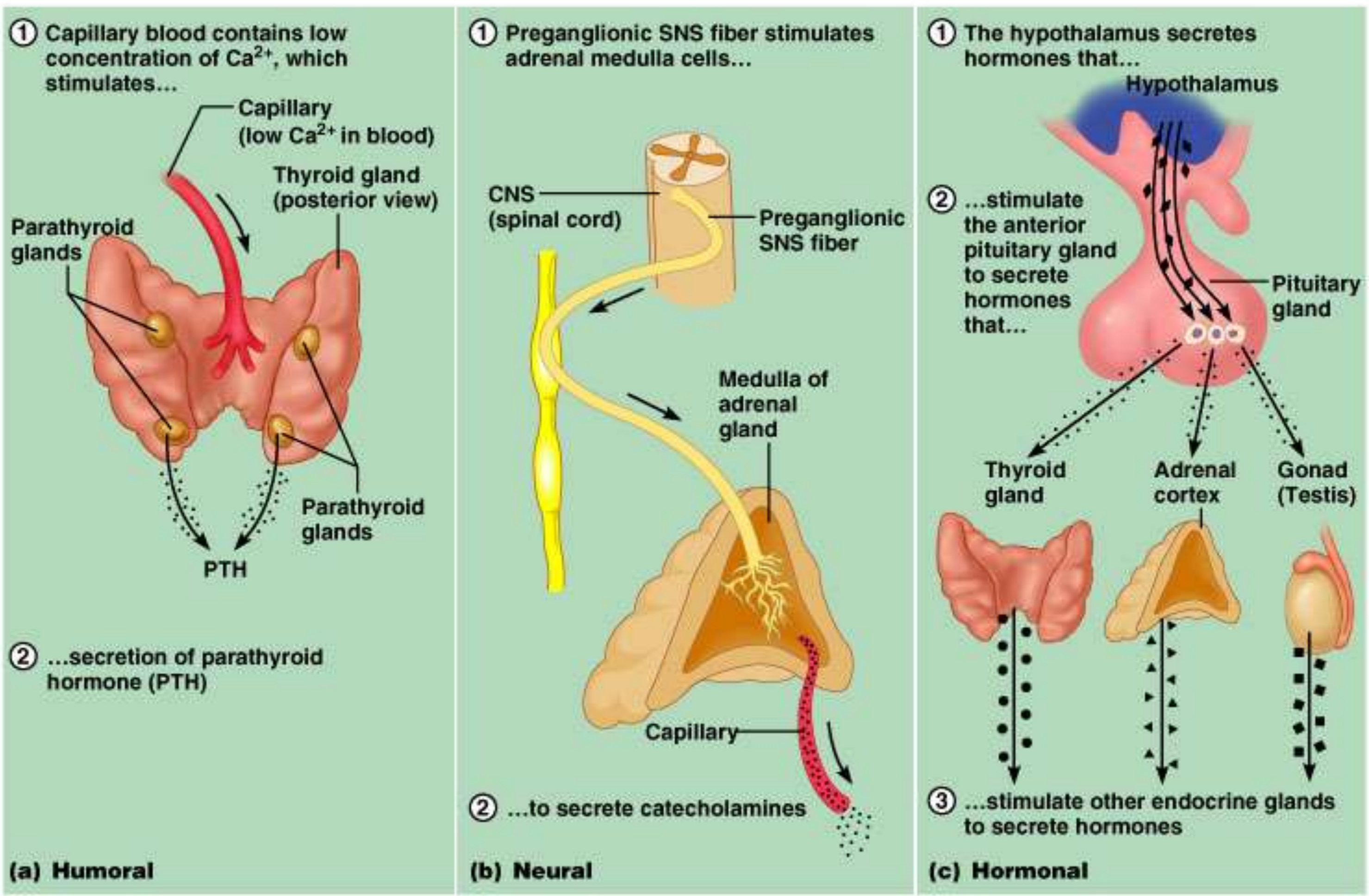
- Nonsteroid Hormones:
  - Not lipid soluble
  - Received by receptors external to the cell membrane
  - Endocrine organs
    - Thyroid gland
    - Parathyroid gland
    - Adrenal medulla
    - Pituitary gland
    - Pancreas

Mechanisms of hormone release

(a) Humoral: in response to changing levels of ions or nutrients in the blood

(b) Neural: stimulation by nerves

(c) Hormonal: stimulation received from other hormones



ORGANS THAT ARE AFFECTED BY HORMONES ARE CALLED TARGET - ORGANS.

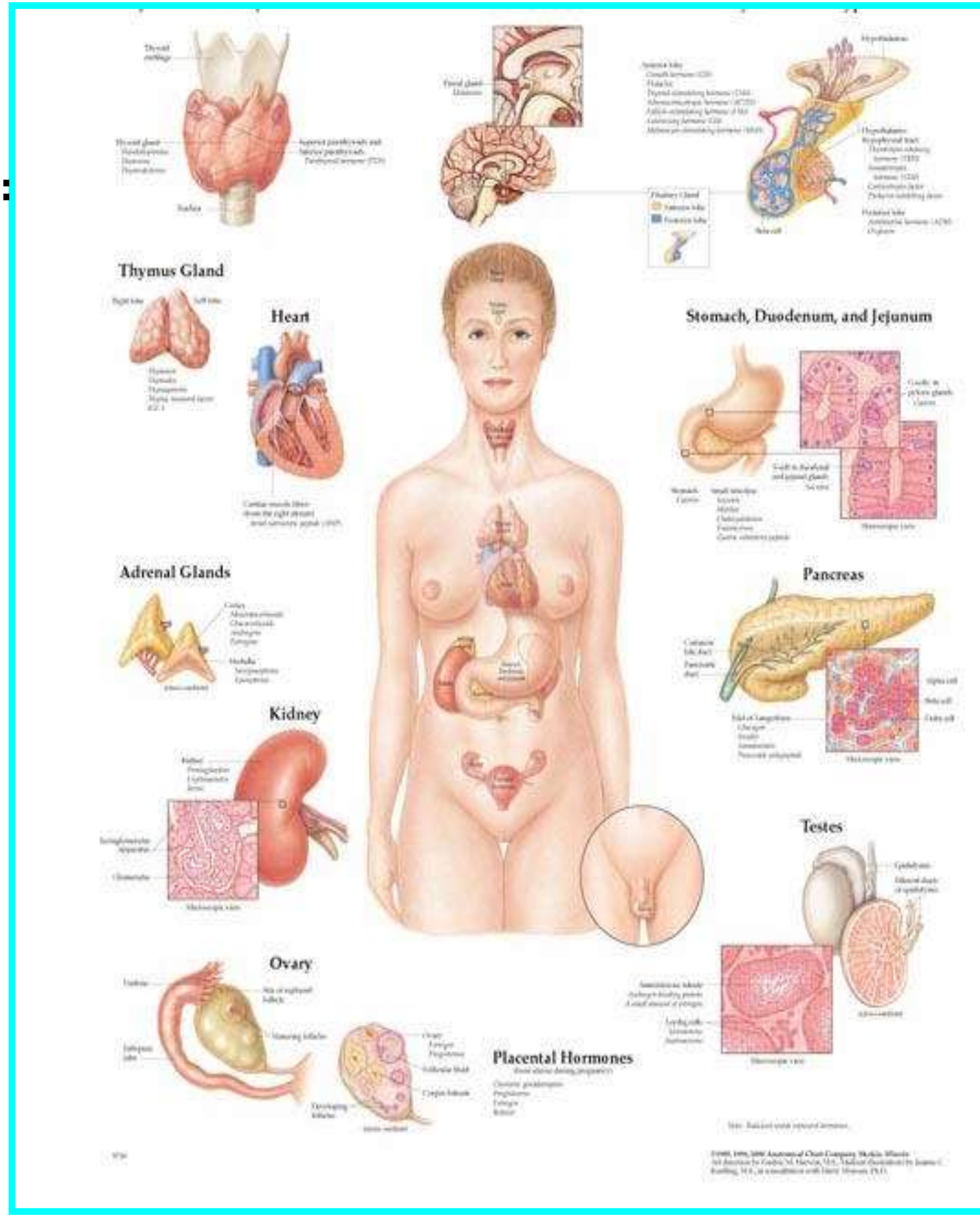
IN TARGET ORGANS, CELLS, WHICH DIRECTLY AFFECTED BY HORMONES ARE CALLED TARGET CELLS.

THERE ARE TWO MAIN MECHANISMS OF ACTION OF HORMONES ON TARGET CELLS:

- **DISTANT;**
- **CONTACT.**

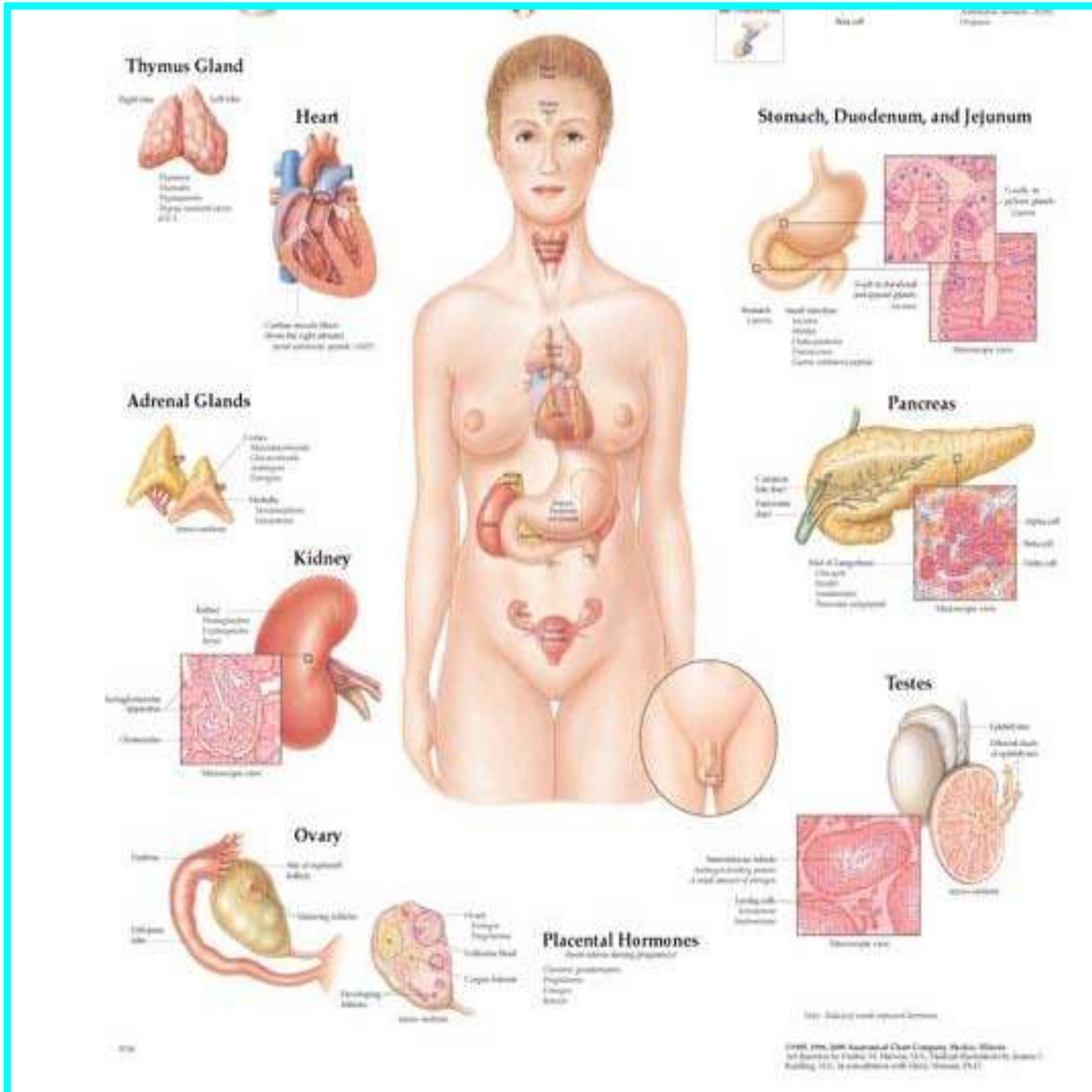
MAJOR ENDOCRINE GLANDS:

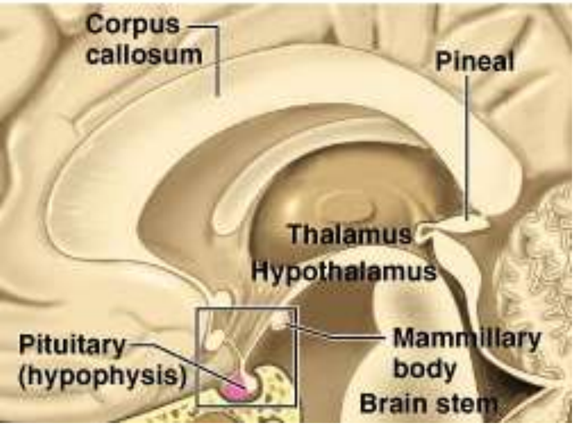
- **HYPOTHALAMUS**
- **PITUITARY GLAND**
- **THYROID GLAND**
- **PARATHYROID GLAND**
- **THYMUS**
- **ADRENAL GLAND**



OTHER ORGANS CONTAINING ENDOCRINE TISSUE:

- **PANCREAS**
- **KIDNEYS**
- **HEART**
- **DIGESTIVE TRACT**
- **PLACENTA**
- **TESTES**
- **OVARIES**
- **PINEAL GLAND**



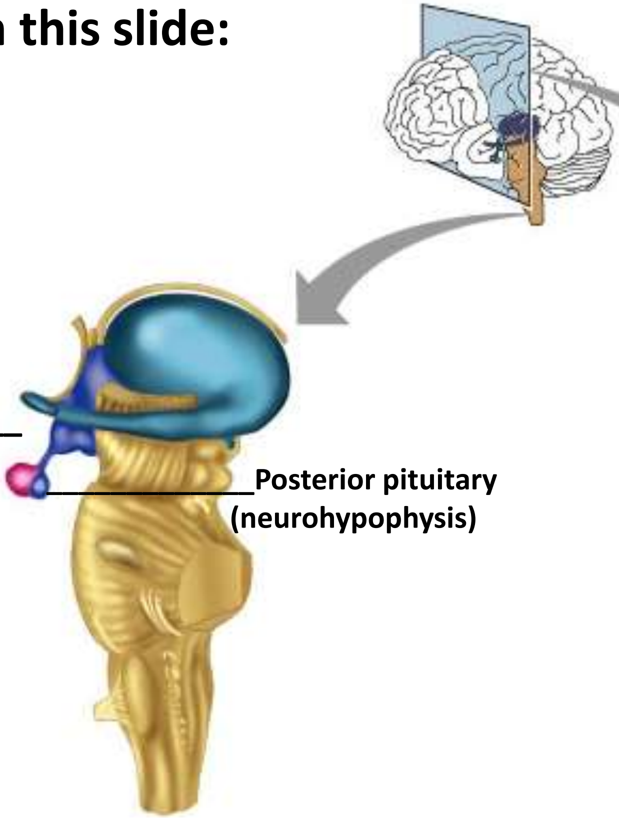


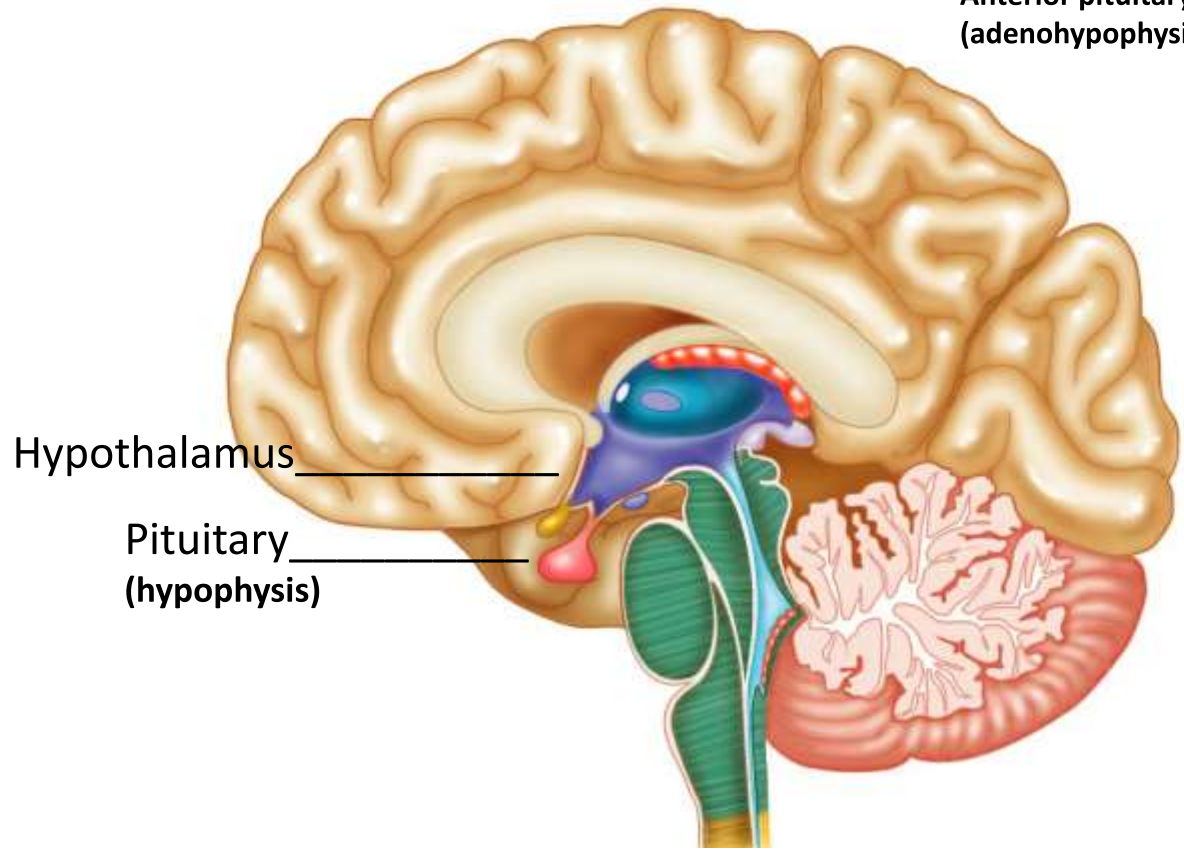
Learn the 3 endocrine organs on this slide:

Hypothalamus

Pituitary (hyophysis)

Pineal





The Pituitary

Sits in hypophyseal fossa: depression in sella turcica of sphenoid bone

Pituitary secretes 9 hormones

Two divisions:

- Anterior pituitary (adenohypophysis)

1. TSH
2. ACTH
3. FSH
4. LH

__The first four are "tropic" hormones, they regulate the function of other hormones__

5. GH
6. PRL
7. MSH

- Posterior pituitary (neurohypophysis)

8. ADH (antidiuretic hormone), or vasopressin
9. Oxytocin

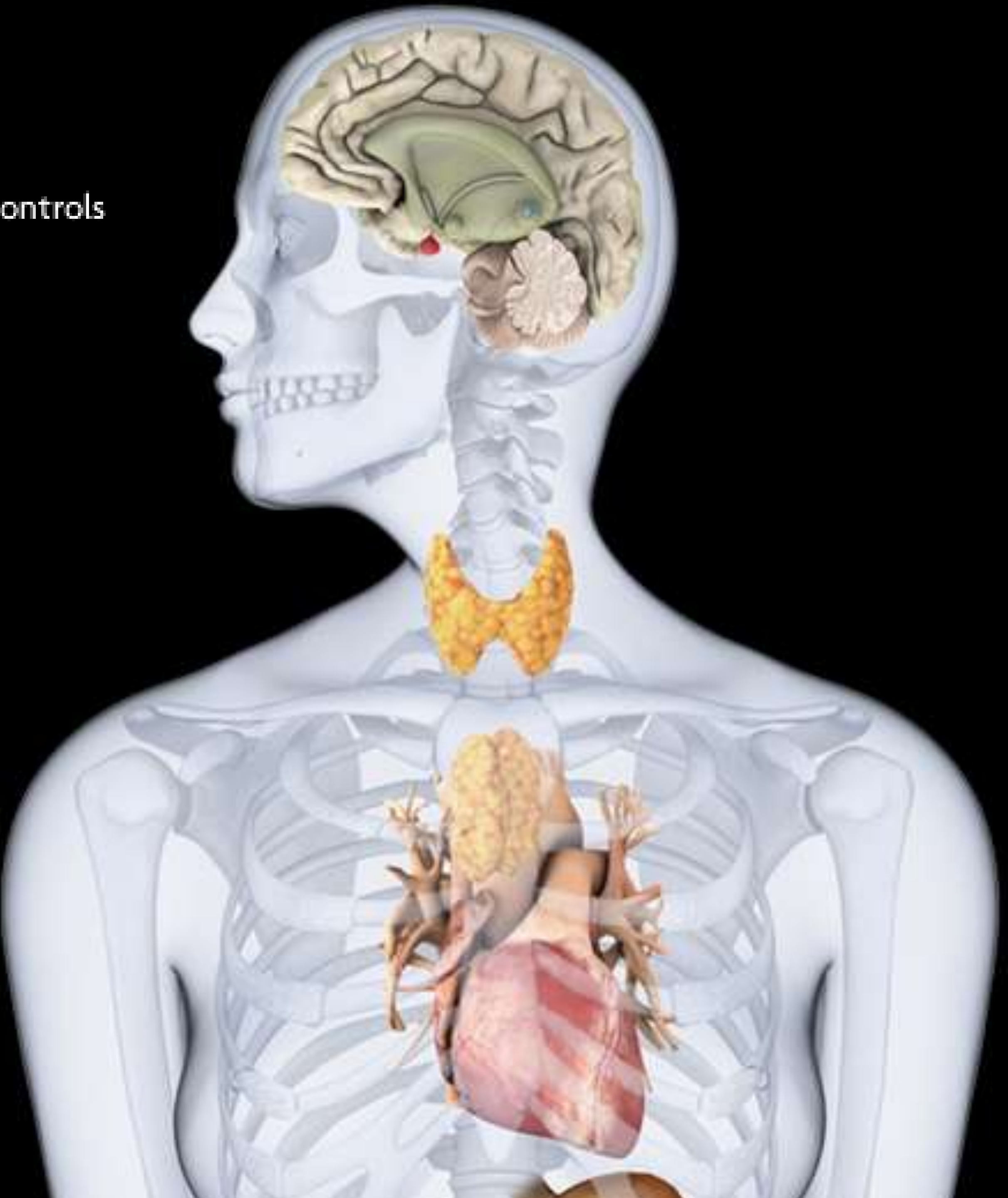
What the letters stand for…

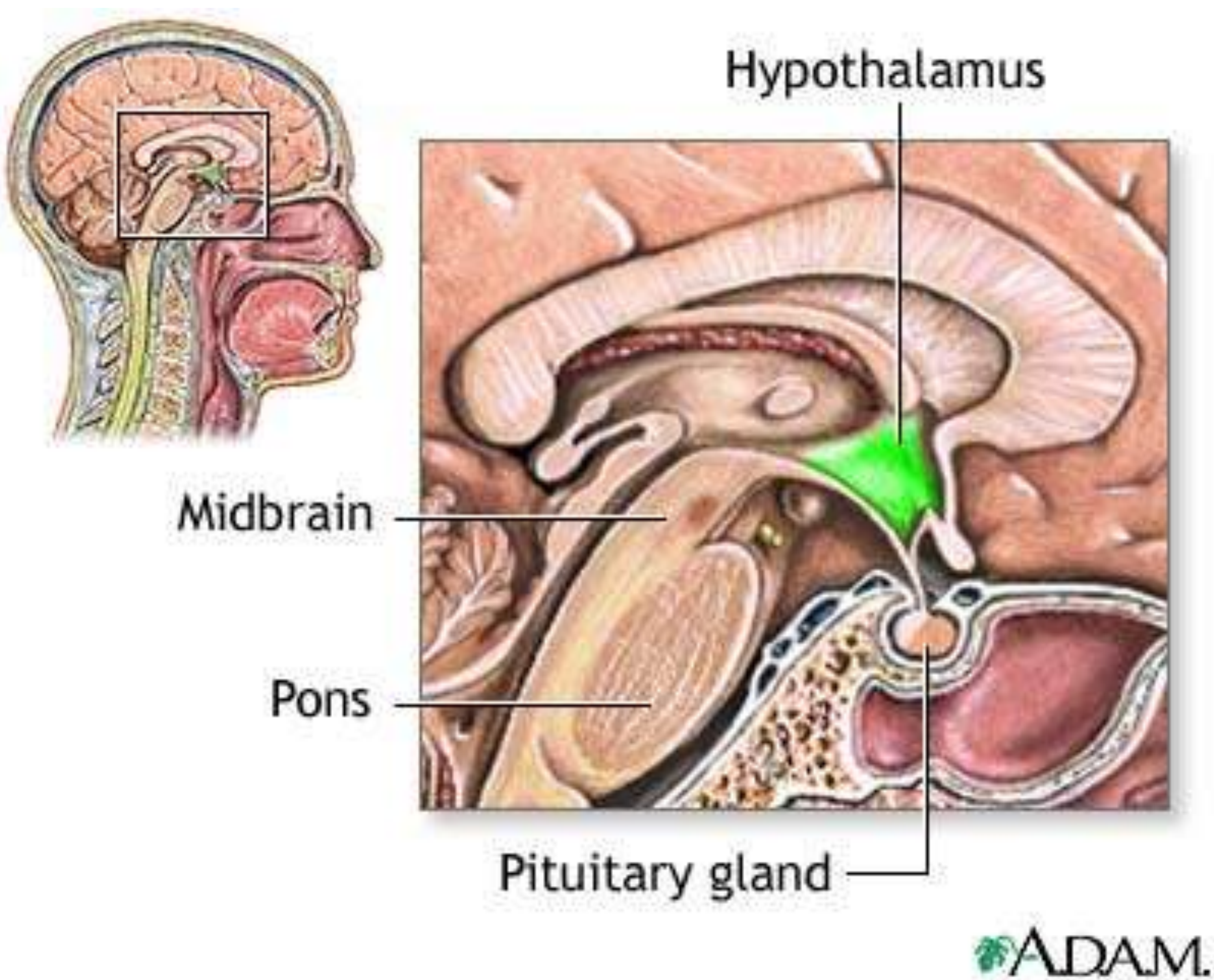
- TSH: thyroid-stimulating hormone
- ACTH: adrenocorticotropic hormone
- FSH: follicle-stimulating hormone
- LH: luteinizing hormone
- GH: growth hormone
- PRL: prolactin
- MSH: melanocyte-stimulating hormone

- ADH: antidiuretic hormone
- Oxytocin

Pituitary gland

Called the "master gland", this organ controls many other endocrine glands




Hypothalamus
Midbrain
Pons
Pituitary gland
A.D.A.M.

PITUITARY GLAND

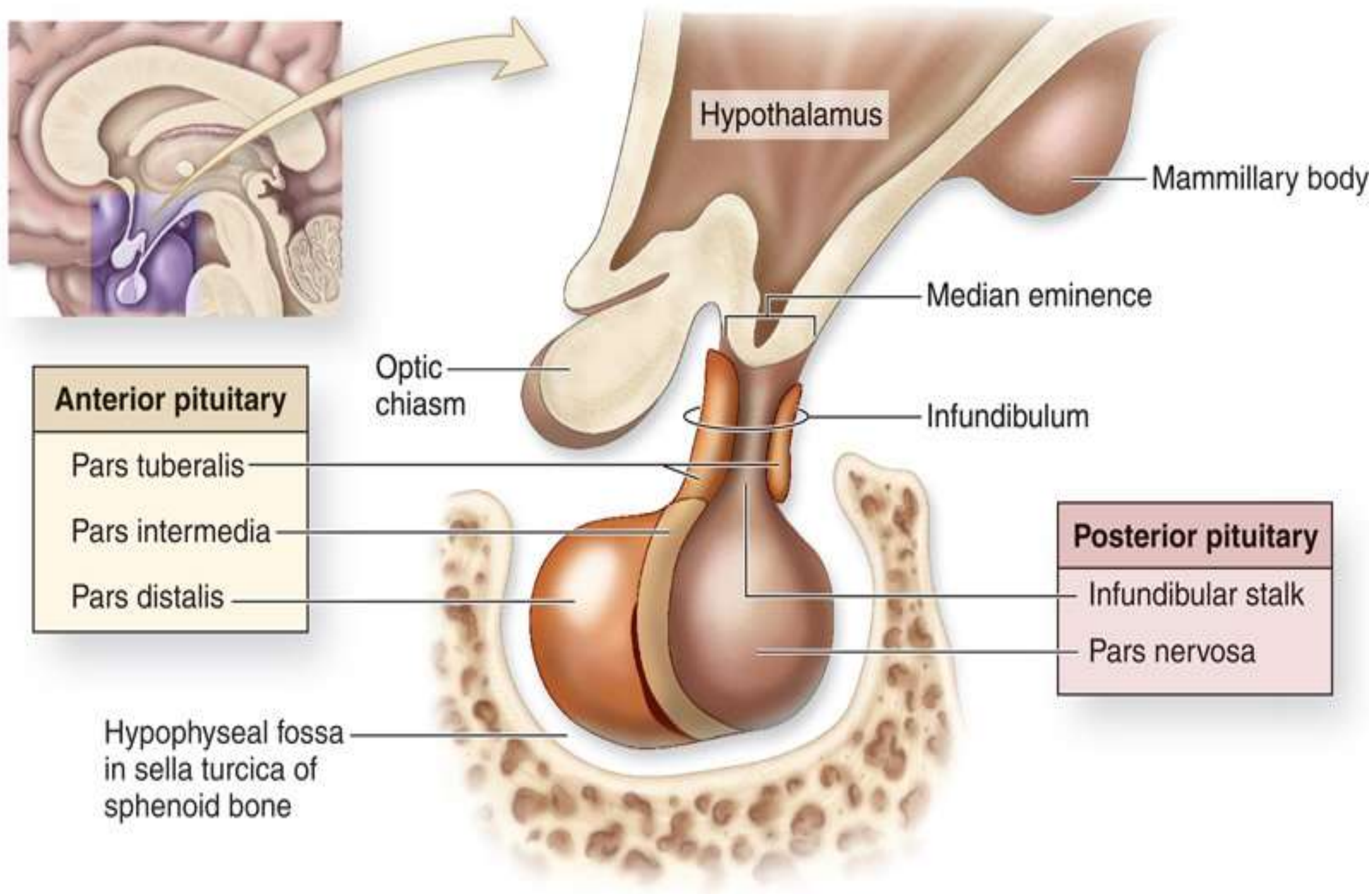
- **Location:** Posterior to the hypothalamus
- **Primary Hormones:** 9 primary hormones including GH, ACTH, TSH, FSH, LH, ADH and Oxytocin
- **Functions:** Secretion of essential endocrine hormones; “the master gland”;
- **Major Disorders:** dwarfism, gigantism, Diabetes encephalis

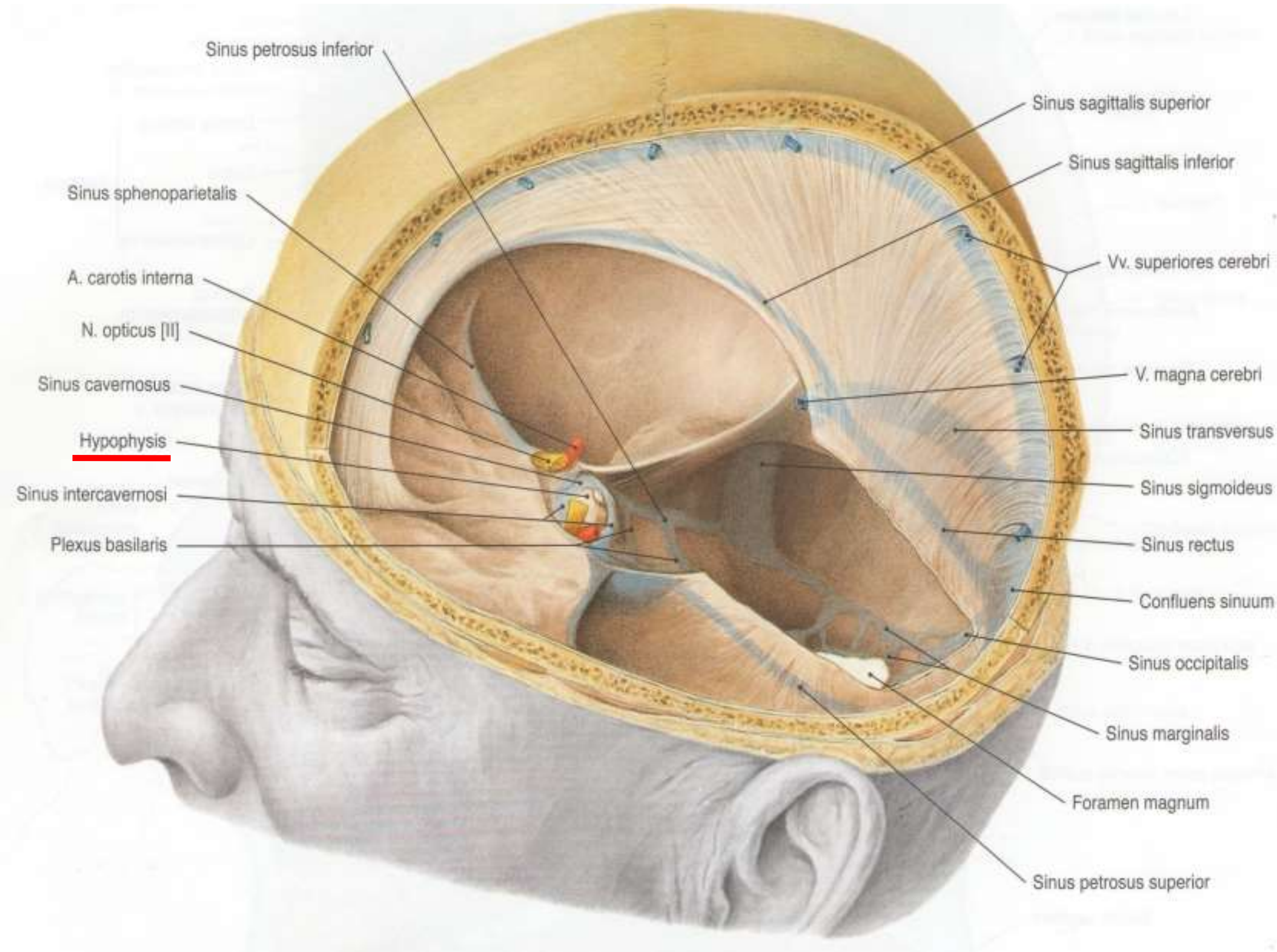
The pituitary gland (hypophysis) - ductless gland that regulates the activity of a number of endocrine glands (thyroid, gonads, adrenal cortex).

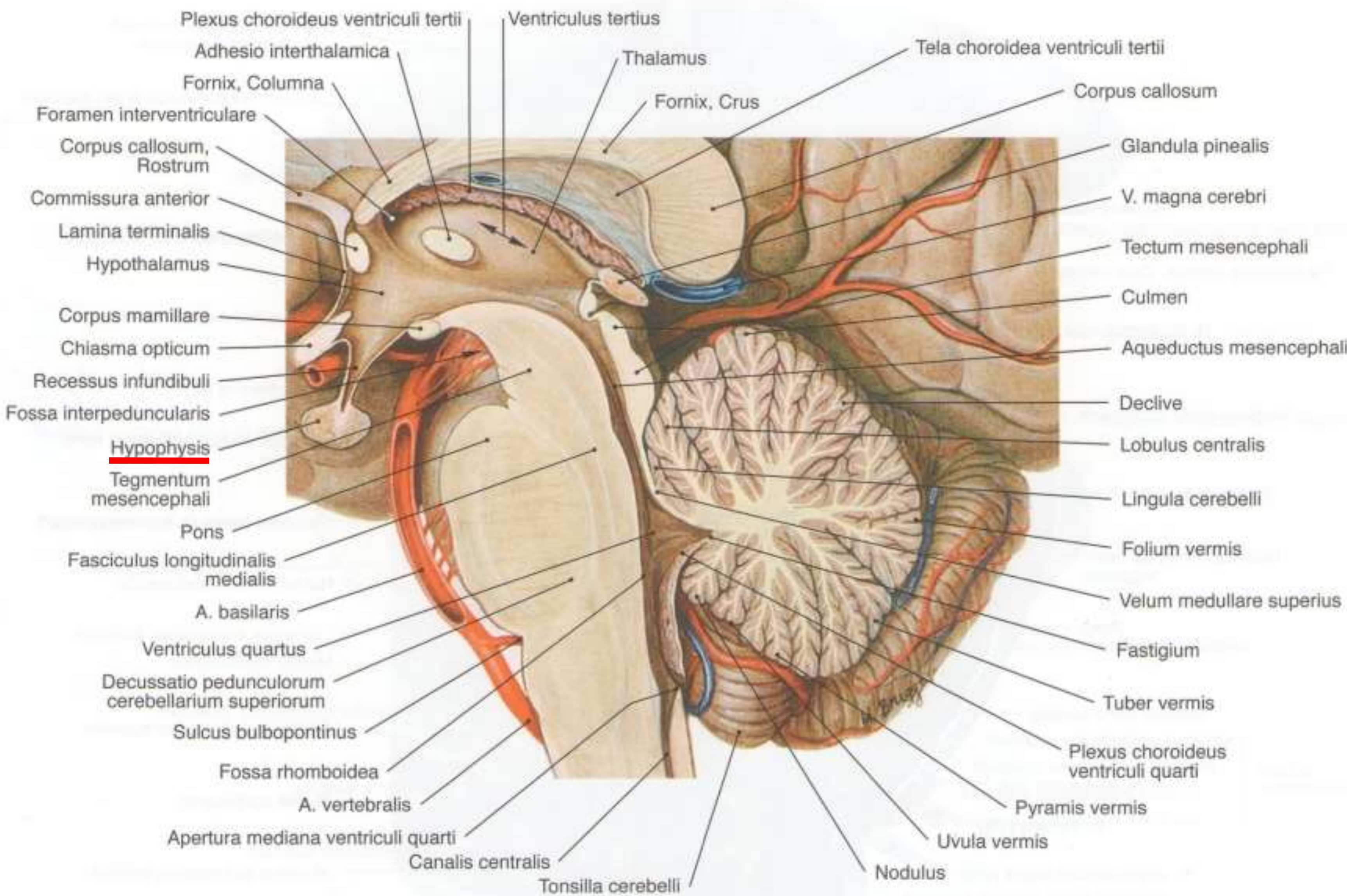
- Anterior lobe (adenohypophysis)

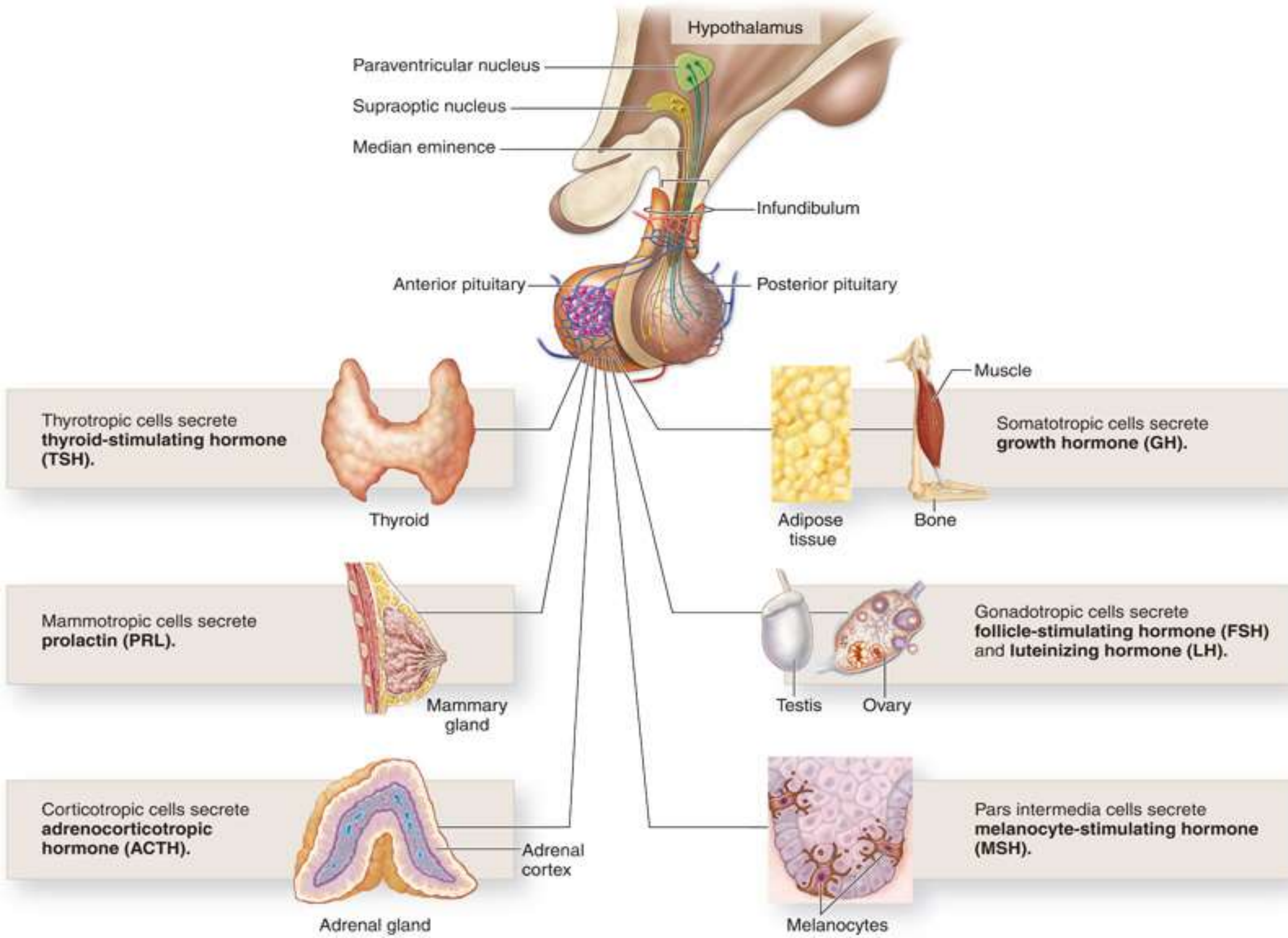
- Intermediate part

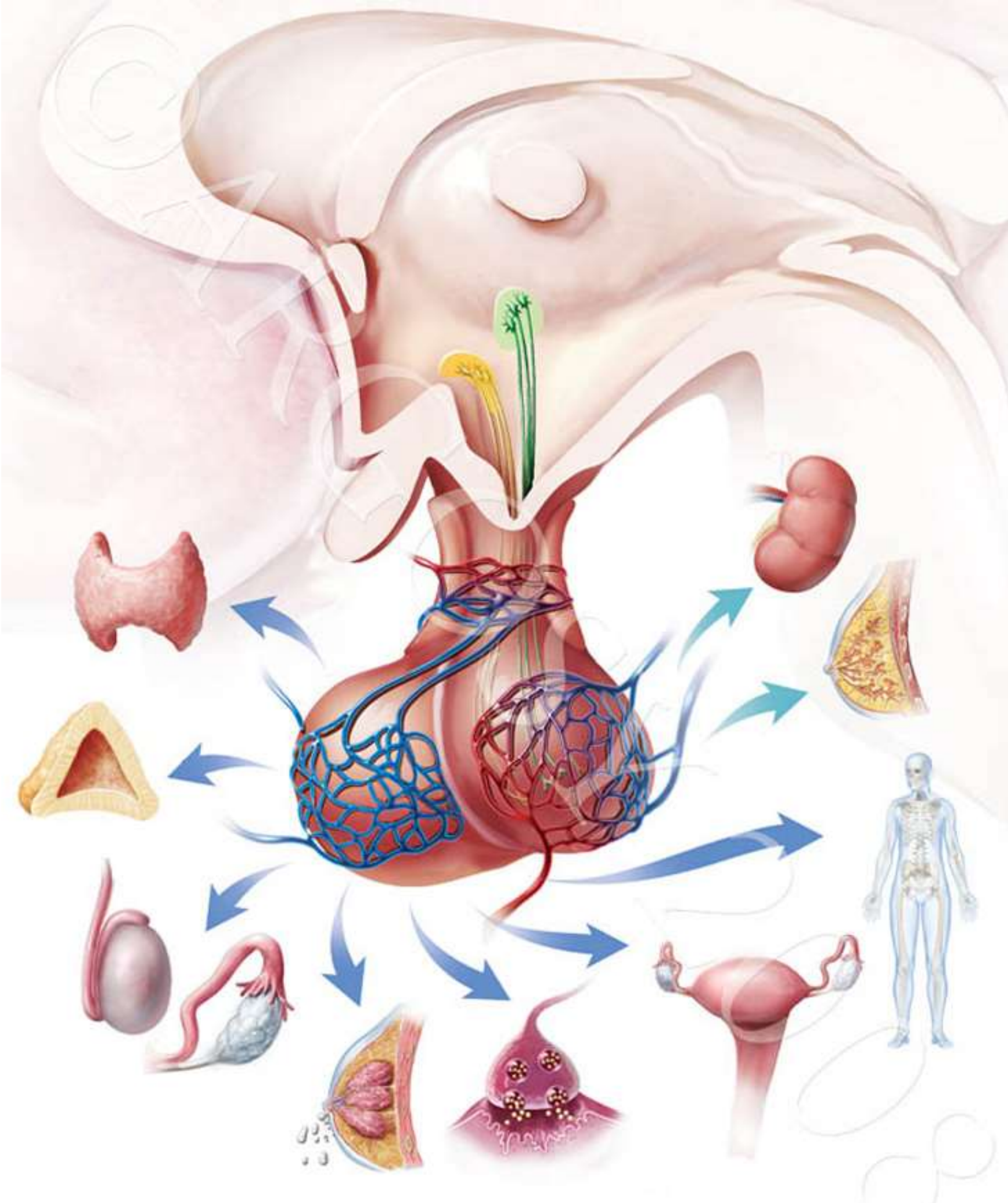
- Posterior lobe(neurohypophysis)

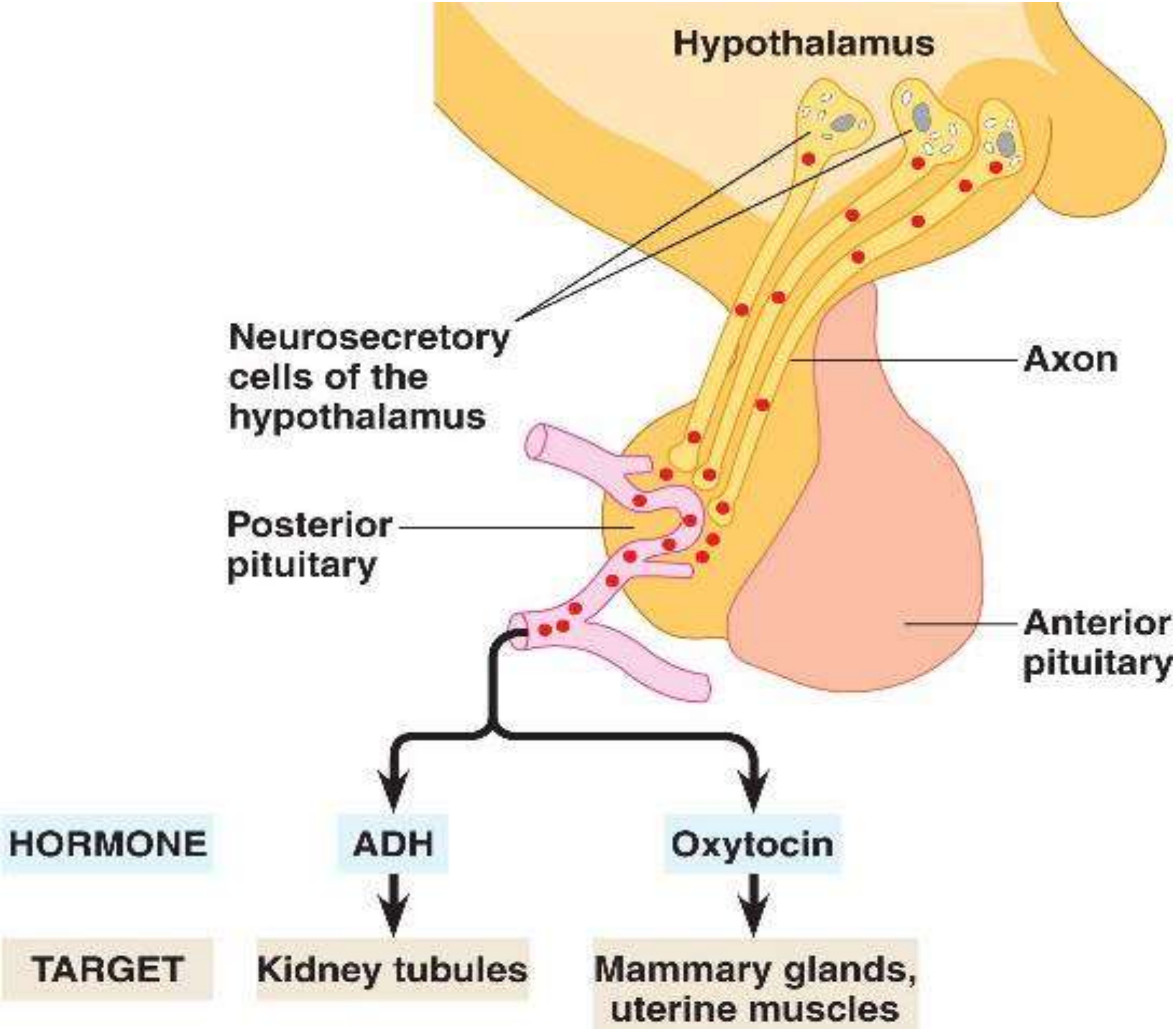

Hypothalamus
Mammillary body
Median eminence
Optic chiasm
Infundibulum
Anterior pituitary
Pars tuberalis
Pars intermedia
Pars distalis
Posterior pituitary
Infundibular stalk
Pars nervosa
Hypophyseal fossa in sella turcica of sphenoid bone


Sinus petrosus inferior
Sinus sagittalis superior
Sinus sagittalis inferior
Sinus sphenoparietalis
Vv. superiores cerebri
A. carotis interna
N. opticus [II]
V. magna cerebri
Sinus cavernosus
Sinus transversus
Hypophysis
Sinus sigmoideus
Sinus intercavernosi
Sinus rectus
Plexus basilaris
Confluens sinuum
Sinus occipitalis
Sinus marginalis
Foramen magnum
Sinus petrosus superior


Plexus choroideus ventriculi tertii
Ventriculus tertius
Adhesio interthalamica
Thalamus
Tela choroidea ventriculi tertii
Fornix, Columna
Fornix, Crus
Corpus callosum
Foramen interventriculare
Corpus callosum, Rostrum
Glandula pinealis
Commissura anterior
V. magna cerebri
Lamina terminalis
Tectum mesencephali
Hypothalamus
Culmen
Corpus mamillare
Aqueductus mesencephali
Chiasma opticum
Recessus infundibuli
Declive
Fossa interpeduncularis
Lobulus centralis
Hypophysis
Tegmentum mesencephali
Lingula cerebelli
Pons
Folium vermis
Fasciculus longitudinalis medialis
Velum medullare superius
A. basilaris
Ventriculus quartus
Fastigium
Decussatio pedunculorum cerebellarium superiorum
Tuber vermis
Sulcus bulbopontinus
Plexus choroideus ventriculi quarti
Fossa rhomboidea
Pyramis vermis
A. vertebralis
Apertura mediana ventriculi quarti
Uvula vermis
Canalis centralis
Nodulus
Tonsilla cerebelli

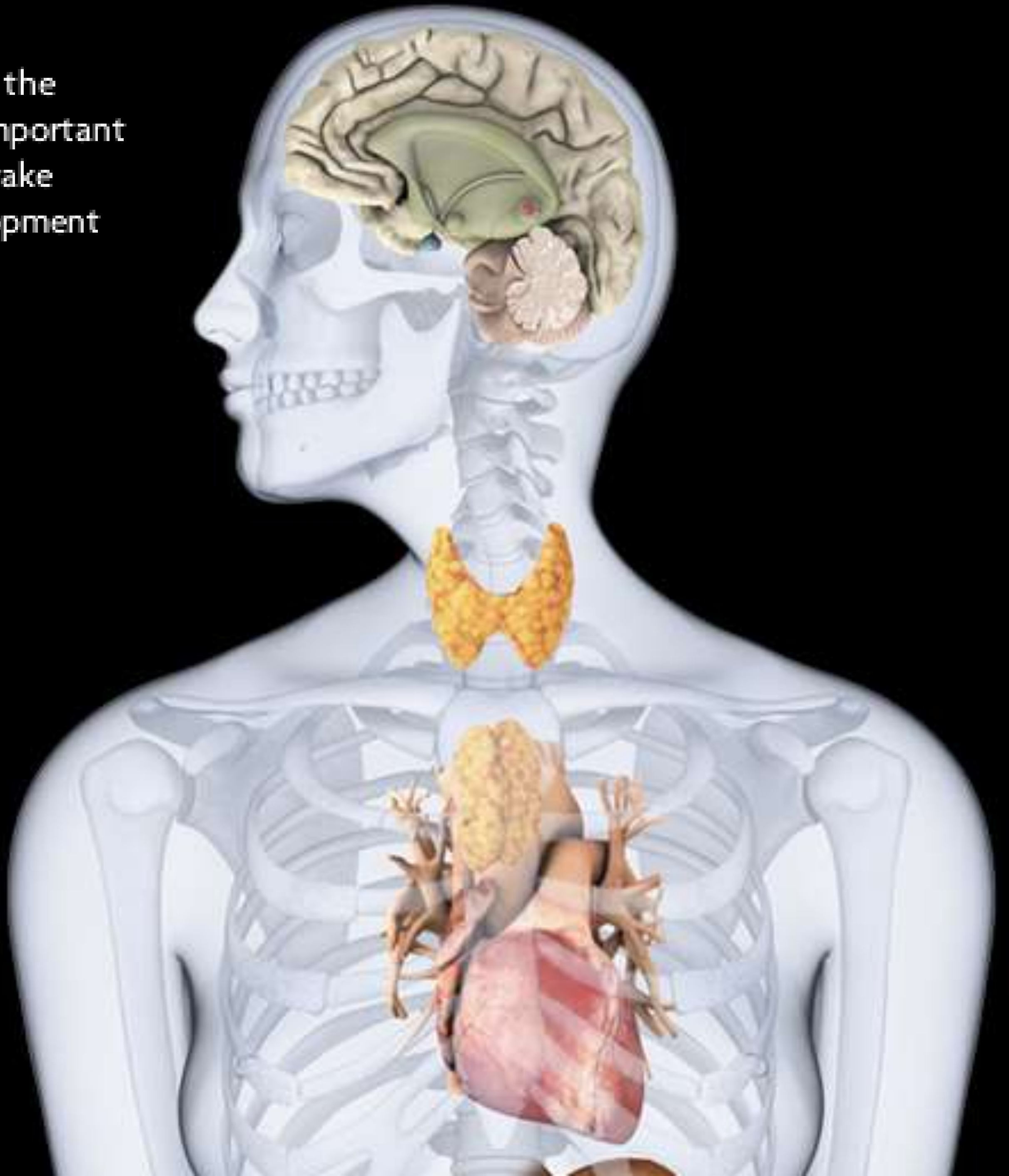

Hypothalamus
Paraventricular nucleus
Supraoptic nucleus
Median eminence
Infundibulum
Anterior pituitary
Posterior pituitary
Thyrotropic cells secrete **thyroid-stimulating hormone (TSH).**
Thyroid
Muscle
Somatotropic cells secrete **growth hormone (GH).**
Adipose tissue
Bone
Mammotropic cells secrete **prolactin (PRL).**
Mammary gland
Gonadotropic cells secrete **follicle-stimulating hormone (FSH)** and **luteinizing hormone (LH).**
Testis
Ovary
Corticotropic cells secrete **adrenocorticotropic hormone (ACTH).**
Adrenal cortex
Adrenal gland
Pars intermedia cells secrete **melanocyte-stimulating hormone (MSH).**
Melanocytes

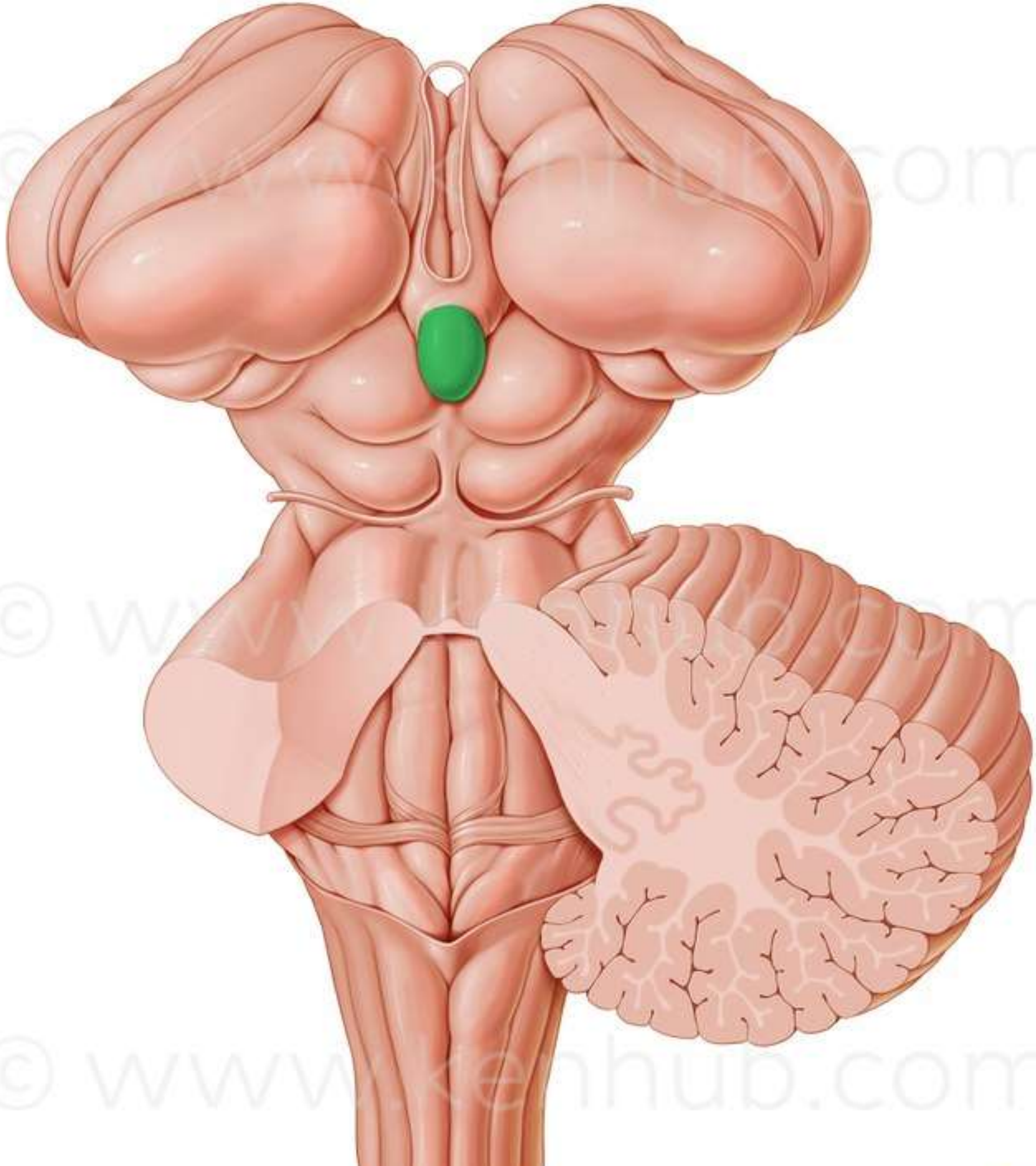



Hypothalamus
Neurosecretory cells of the hypothalamus
Axon
Posterior pituitary
Anterior pituitary
HORMONE
ADH
Oxytocin
TARGET
Kidney tubules
Mammary glands, uterine muscles

Pineal gland (pineal body)

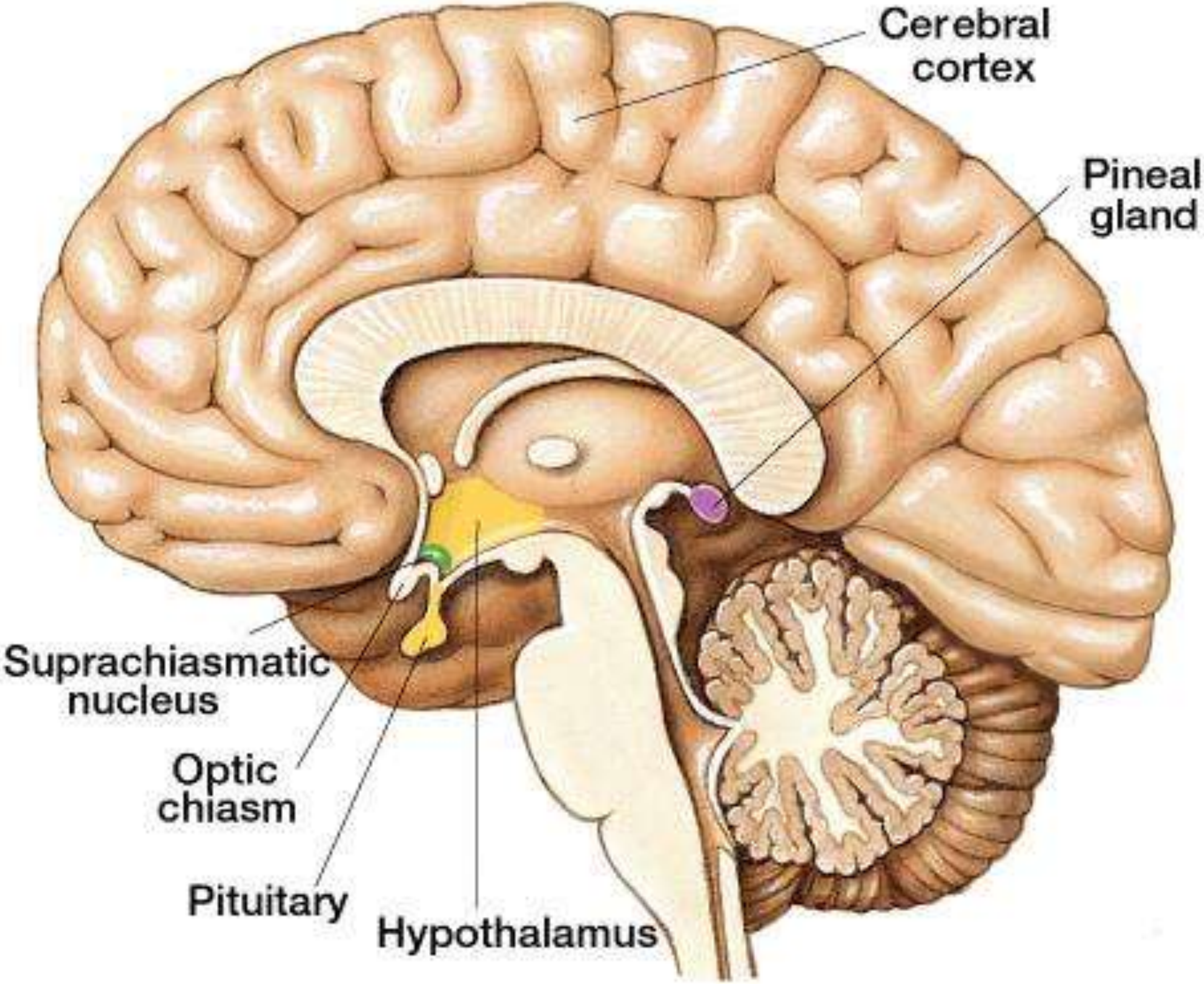
This pea-sized gland in the middle of the brain makes melatonin, a hormone important in body rhythms such as the sleep–wake cycle; it also influences sexual development




© www.kenhub.com

PINEAL GLAND

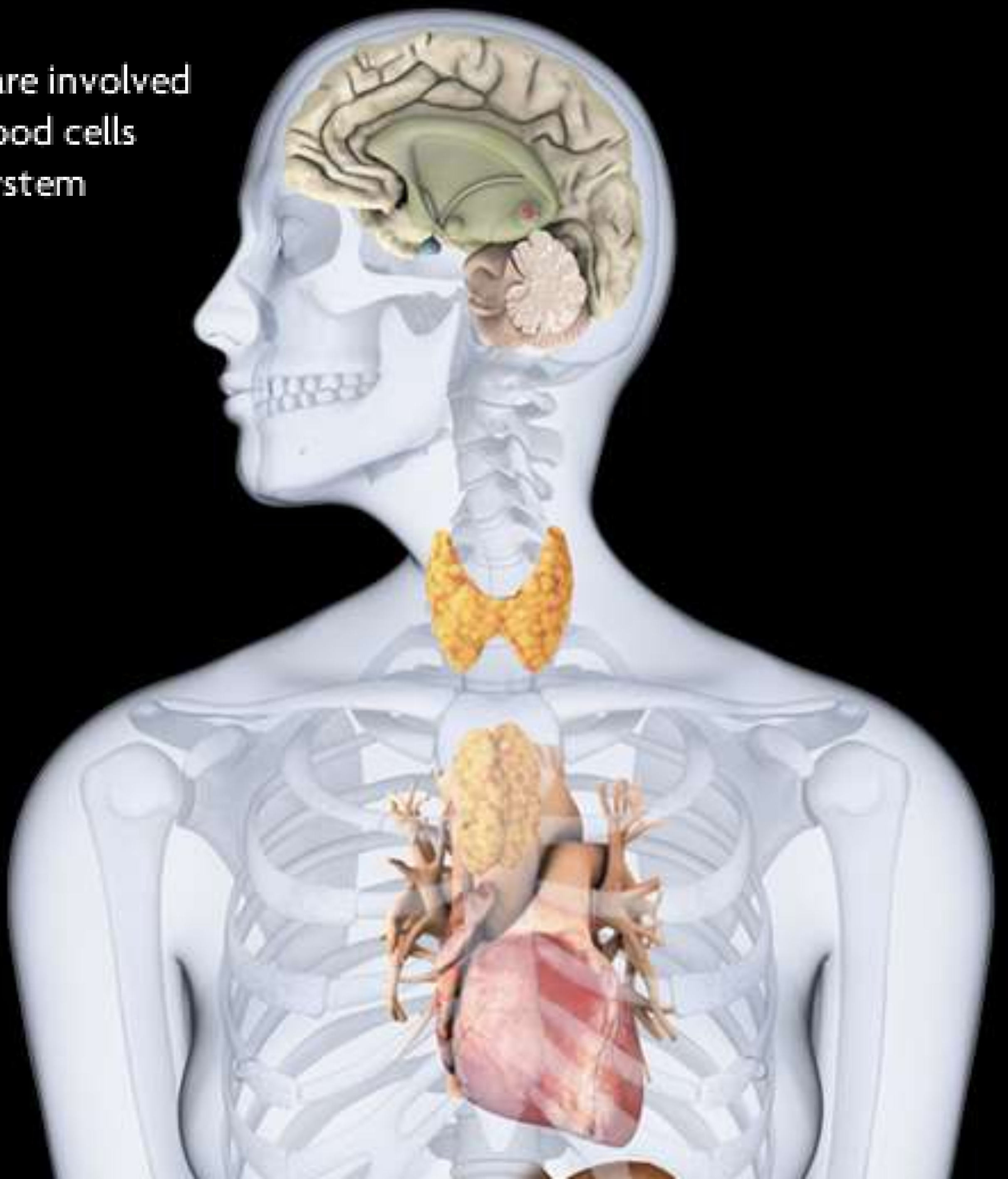
- **Location:** Between the pons and sides of the thalamus; size of a pea
- **Primary Hormones:** Melatonin
- **Functions:** Regulates sleep patterns; mating patterns; migration patterns; day and night rhythms
- **Major Disorders:** Insomnia


Cerebral
cortex
Pineal
gland
Suprachiasmatic
nucleus
Optic
chiasm
Pituitary
Hypothalamus

THE PINEAL GLAND OR EPIPHYSIS (GLANDULA PINEALIS, EPIPHYSIS) - **ANATOMICALLY BELONGS TO EPITALAMUS OF THE DIENCEPHALON. REGULATES CIRCADIAN RHYTHMS IN HUMANS, MODELING THE FUNCTIONAL ACTIVITY OF MANY ENDOCRINE GLANDS.**

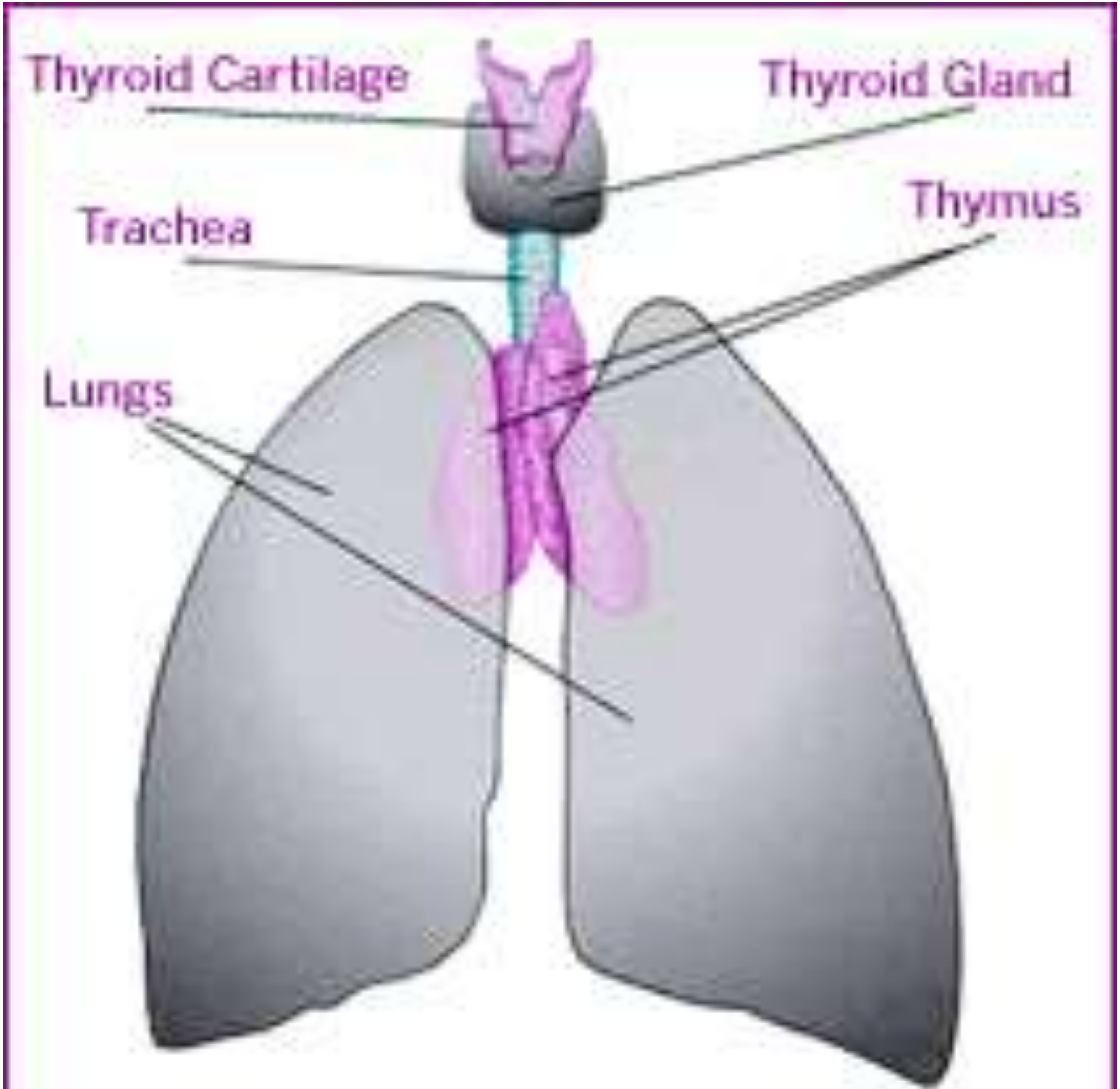
Thymus gland

Produces three hormones that are involved in the development of white blood cells called T cells, for the immune system



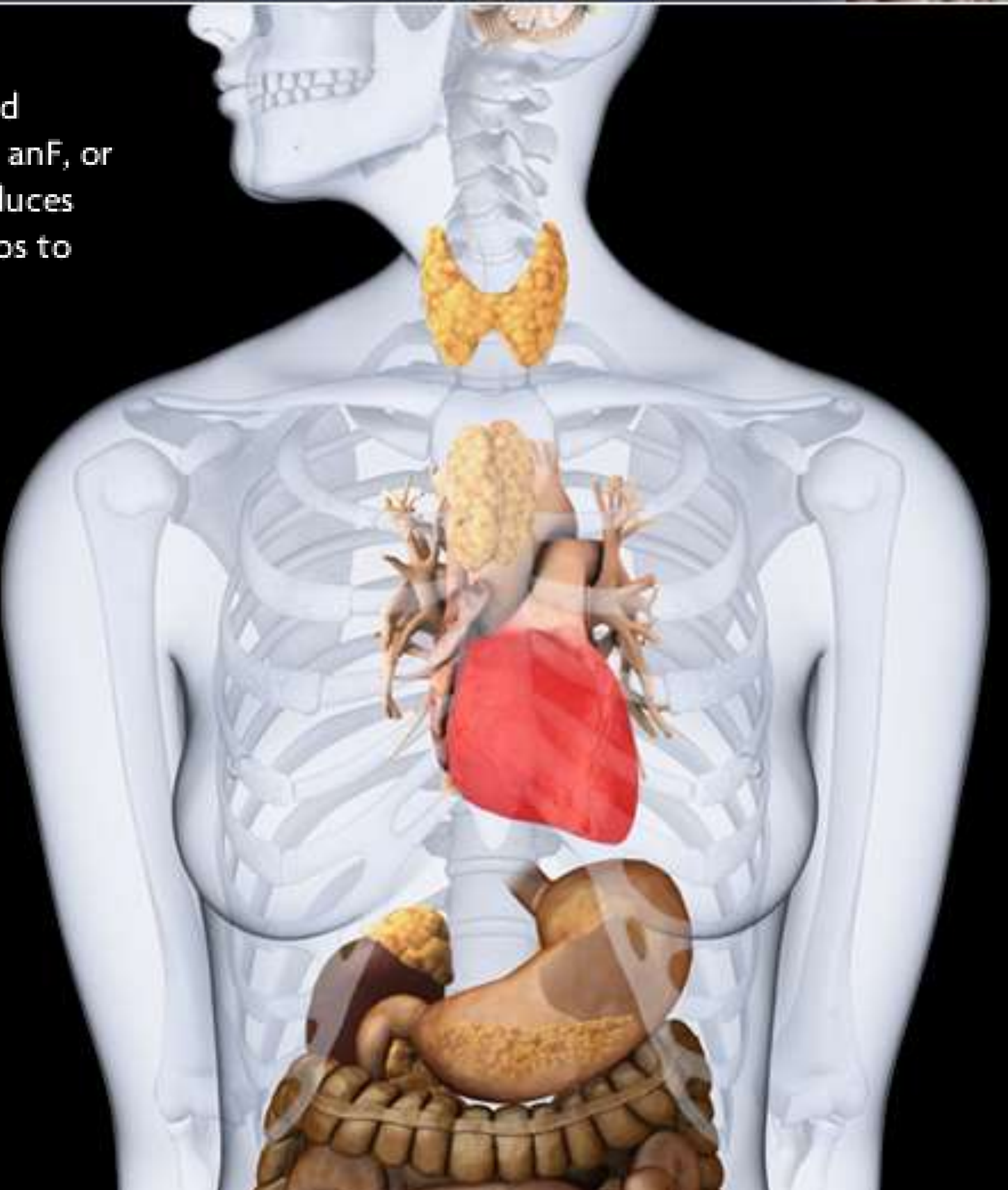
THYMUS

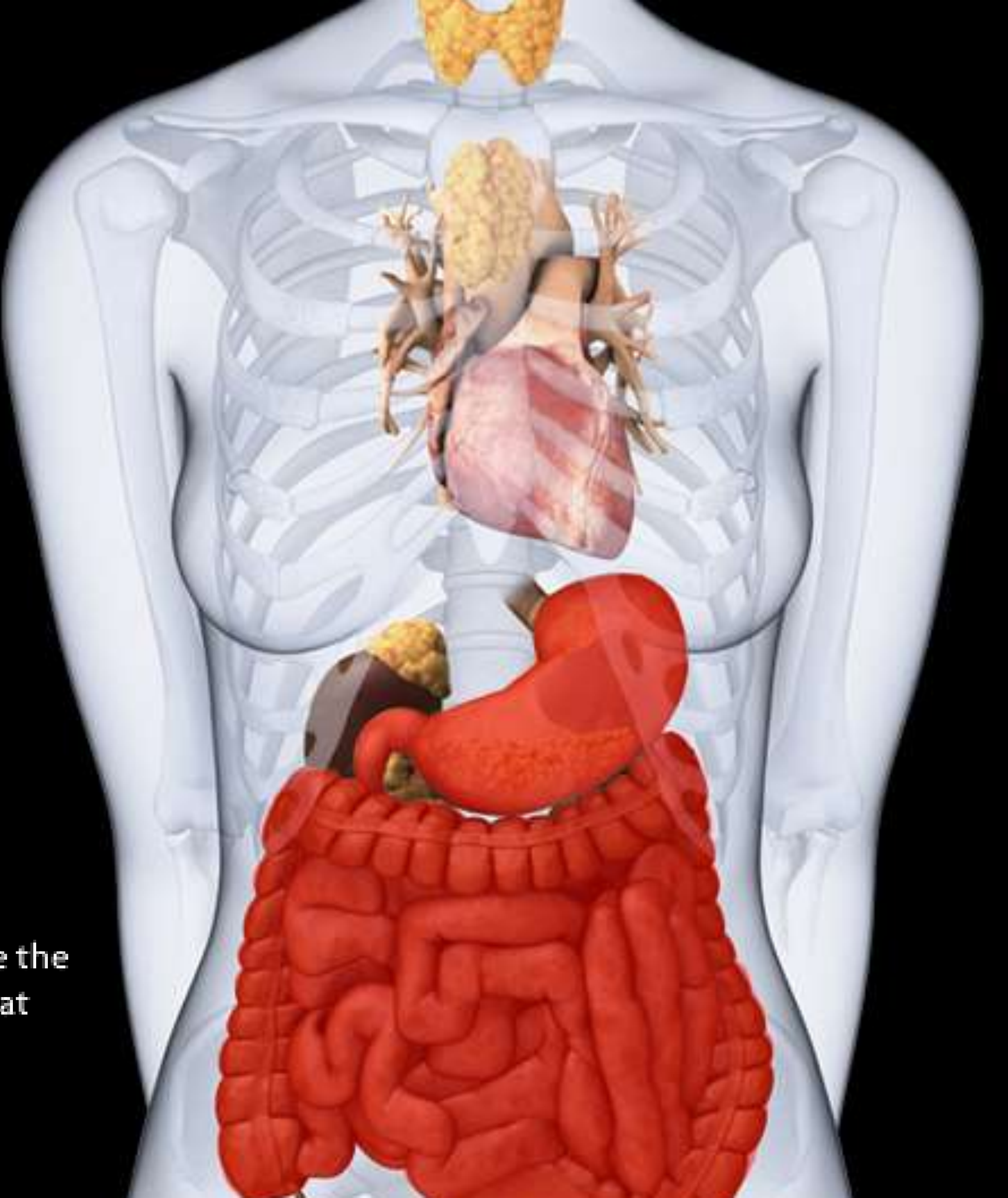
- **Location:** Thoracic cavity below the neck
- **Primary Hormones:** Thymosis
- **Functions:** T-lymphocyte education center
- **Major Disorders:** Cancers (lymphomas)


Thyroid Cartilage
Thyroid Gland
Thymus
Trachea
Lungs

Heart

The heart produces a hormone called atriopeptin (atrial natriuretic factor, anF, or atrial natriuretic peptide, anP); it reduces blood volume and pressure, and helps to regulate fluid balance

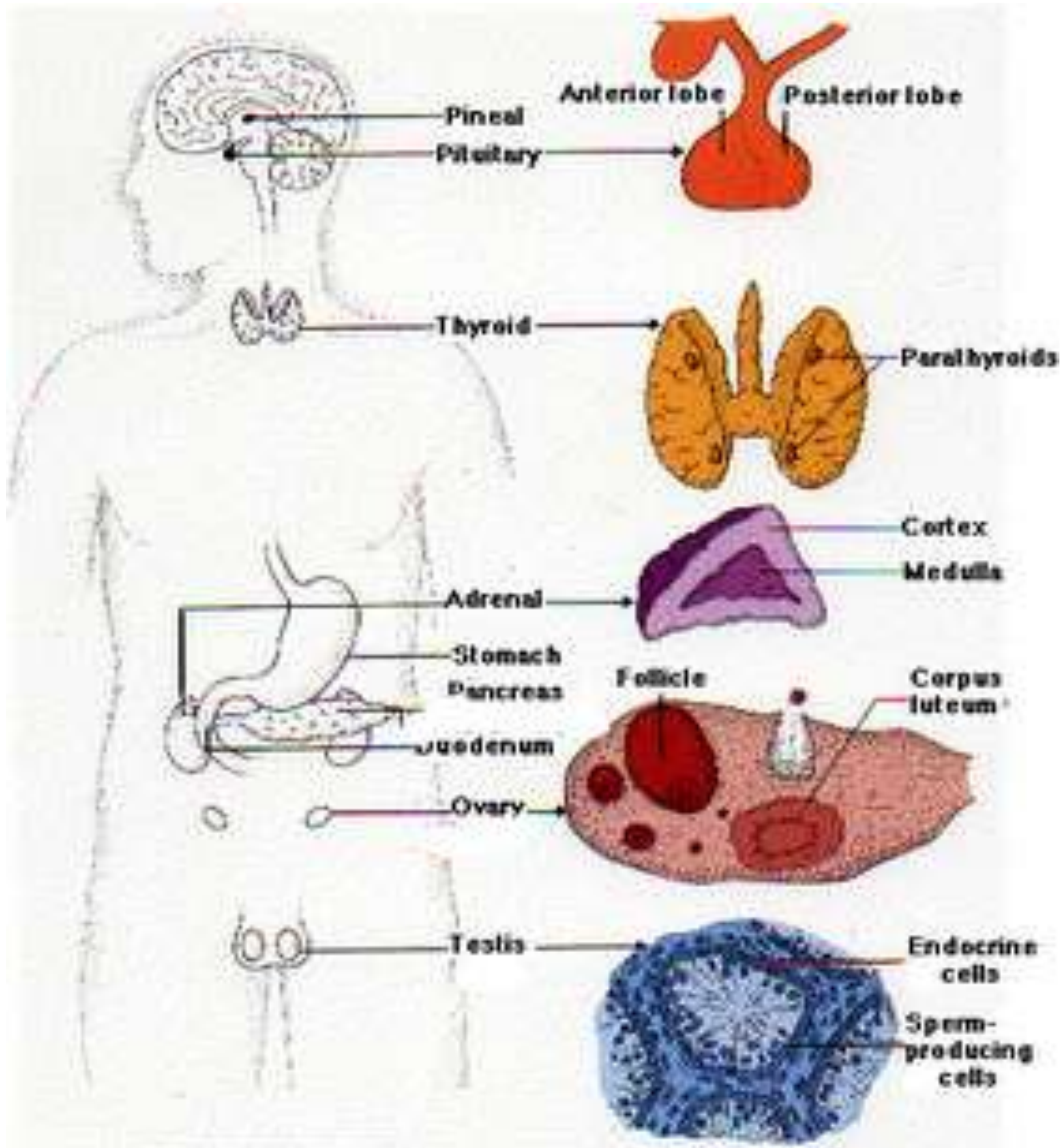


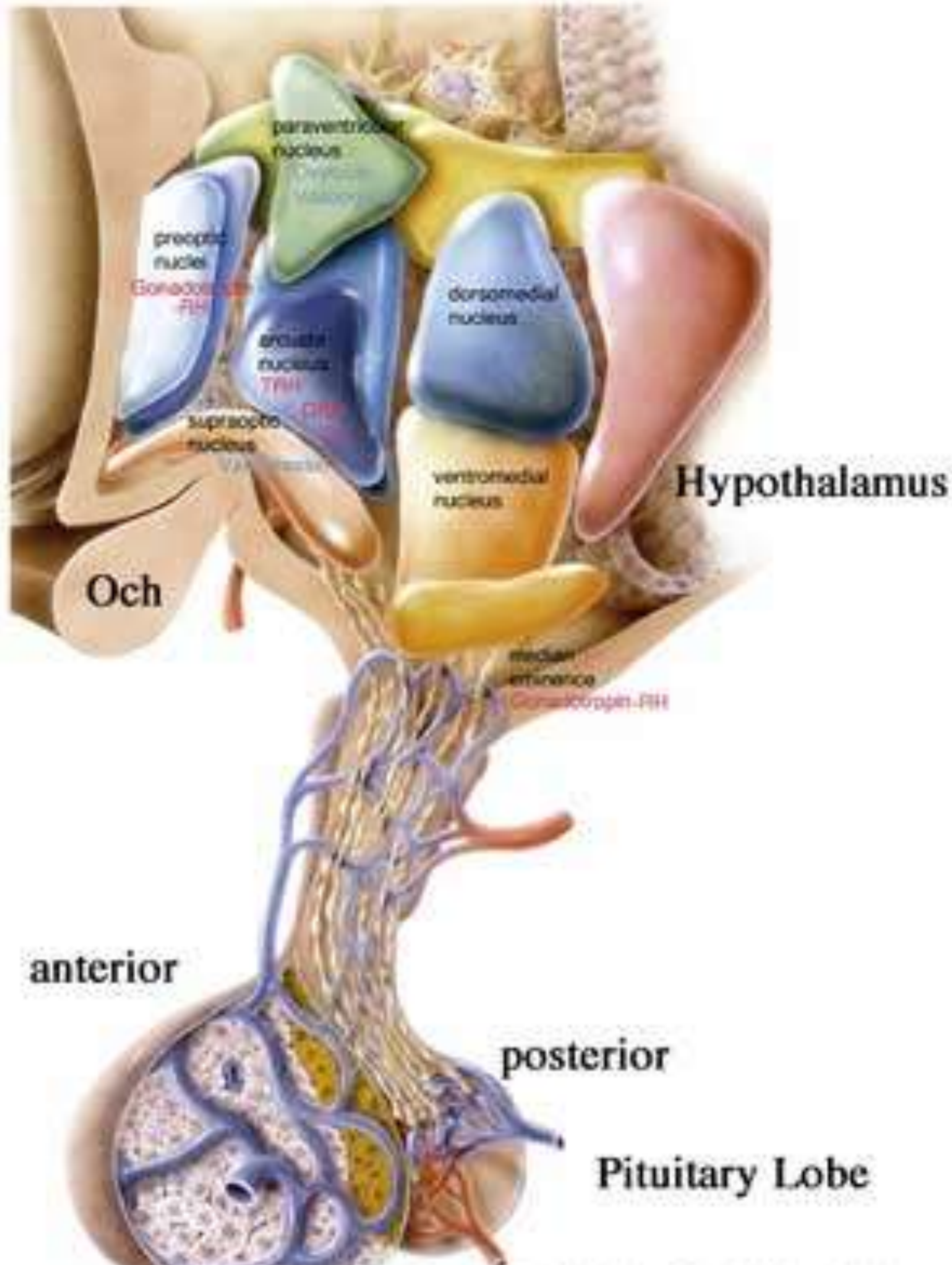


ENDOCRINE SYSTEM

Stomach and intestines

These make hormones that stimulate the production or release of enzymes that aid digestion


Anterior lobe
Posterior lobe
Pineal
Pituitary
Thyroid
Parathyroids
Cortex
Medulla
Adrenal
Stomach
Pancreas
Duodenum
Follicle
Corpus luteum
Ovary
Testis
Endocrine cells
Sperm-producing cells



Exempla Endocrinologica, Breckwoldt et al., 1991

THYROID GLAND

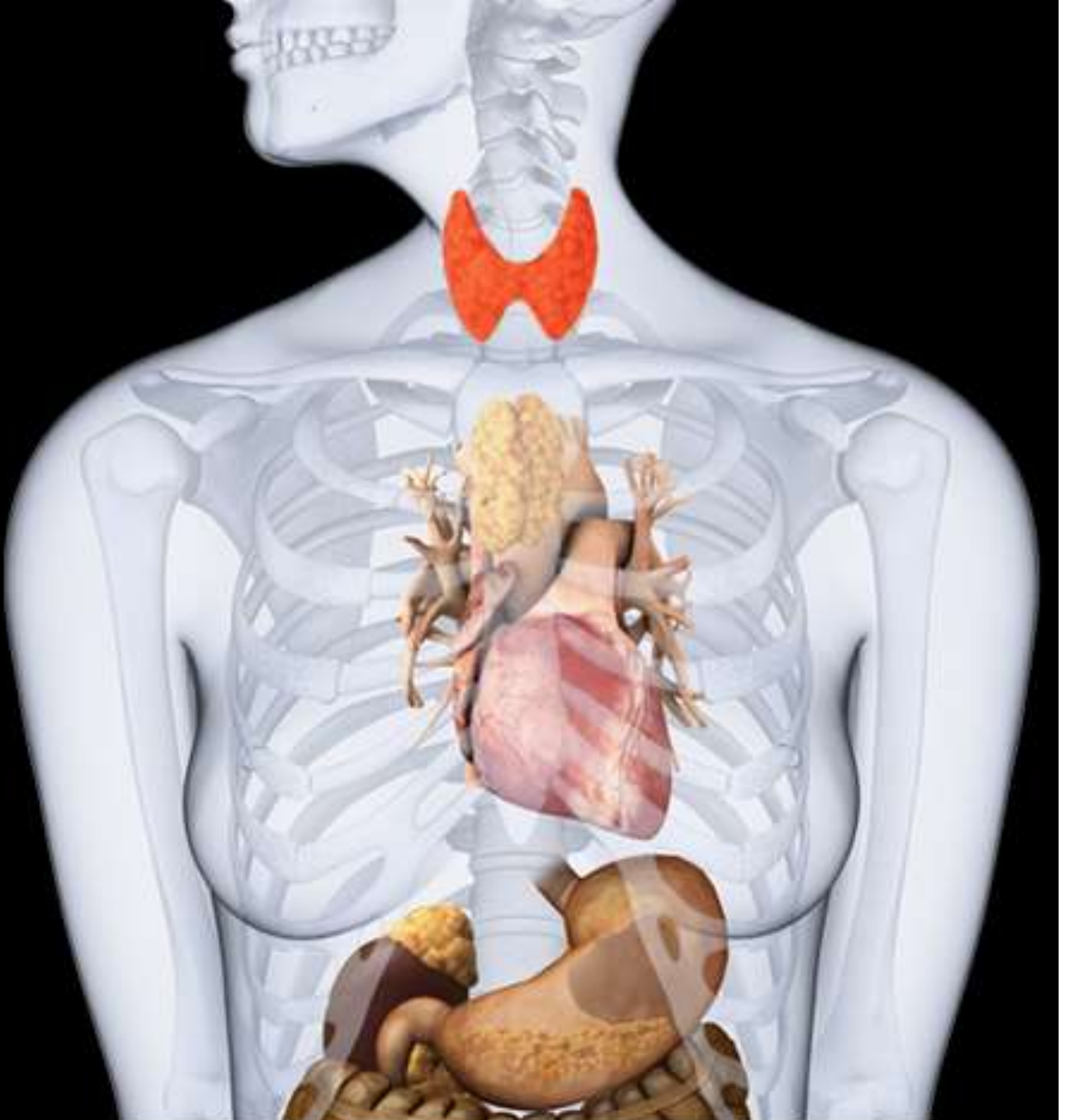
(GLANDULA THYROIDEA)

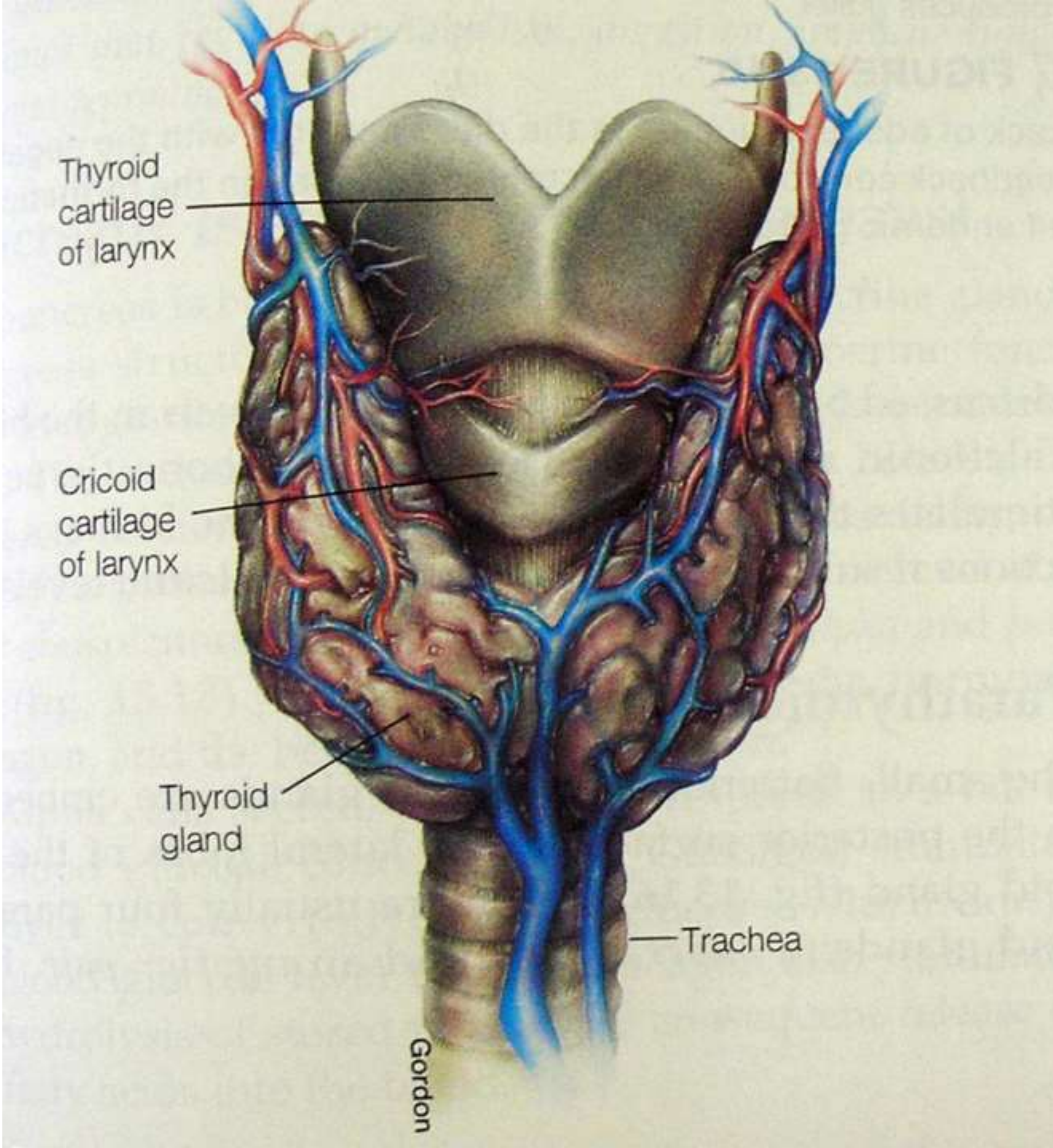
ENDOCRINE SYSTEM

The body's chemical messengers (hormones) are made by endocrine glands. These glands have no ducts but secrete their hormones directly into the blood, by which means they reach every cell in the body. Hormones affect certain target tissues or organs and regulate their activities.

Thyroid gland

Responsible for controlling aspects of metabolism, including the maintenance of body weight, the rate of energy use, and heart rate; unlike other glands, it can store its hormones

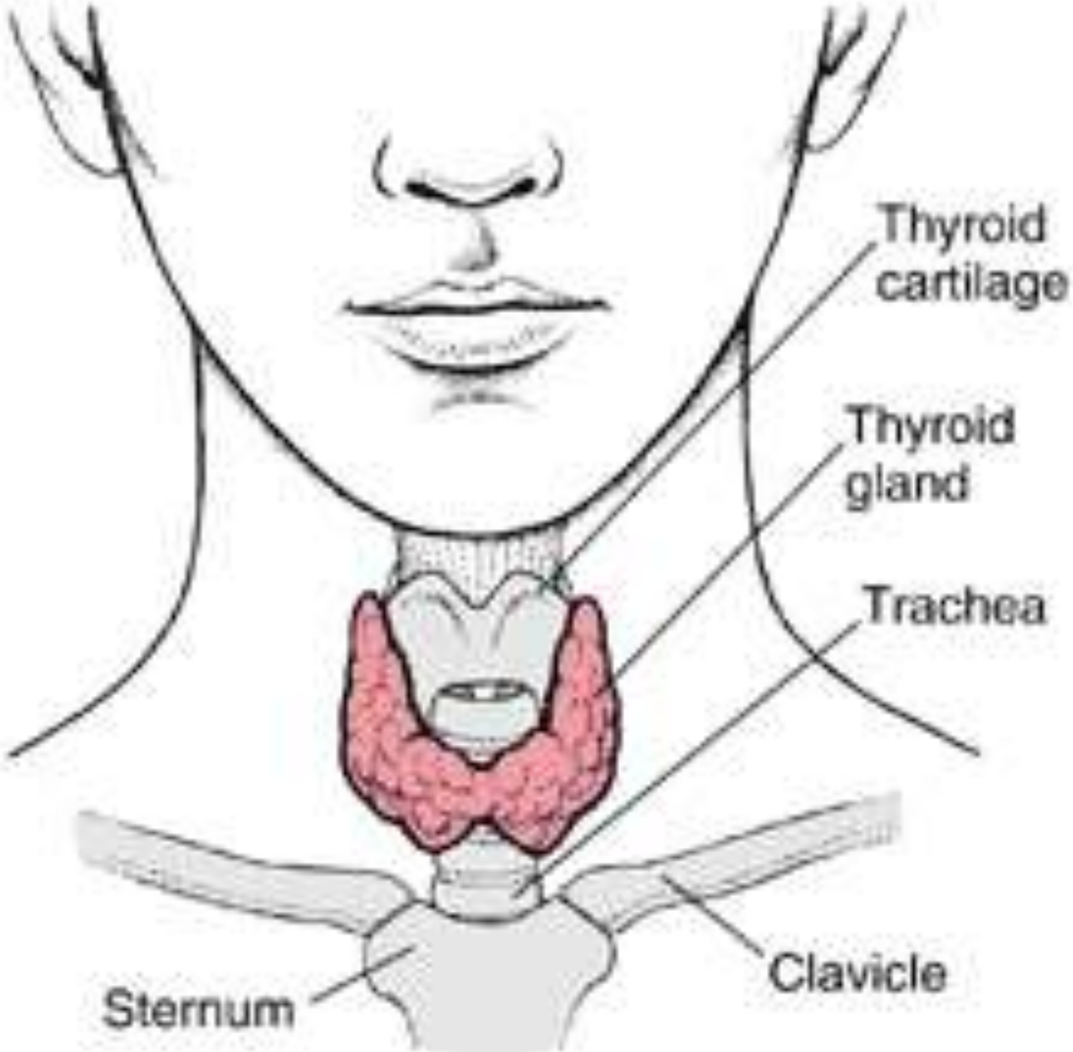


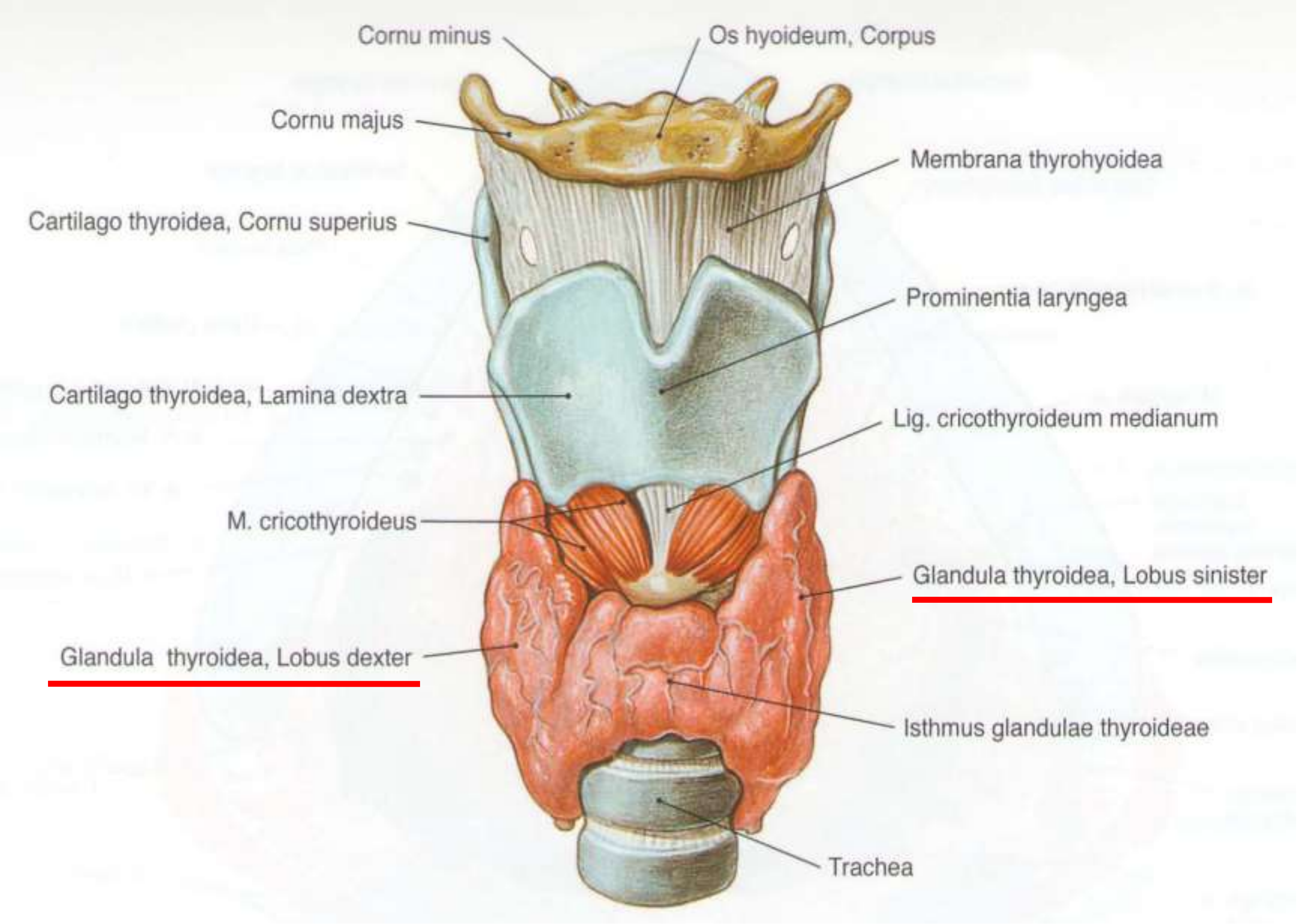

Thyroid cartilage of larynx
Cricoid cartilage of larynx
Thyroid gland
Trachea
Gordon

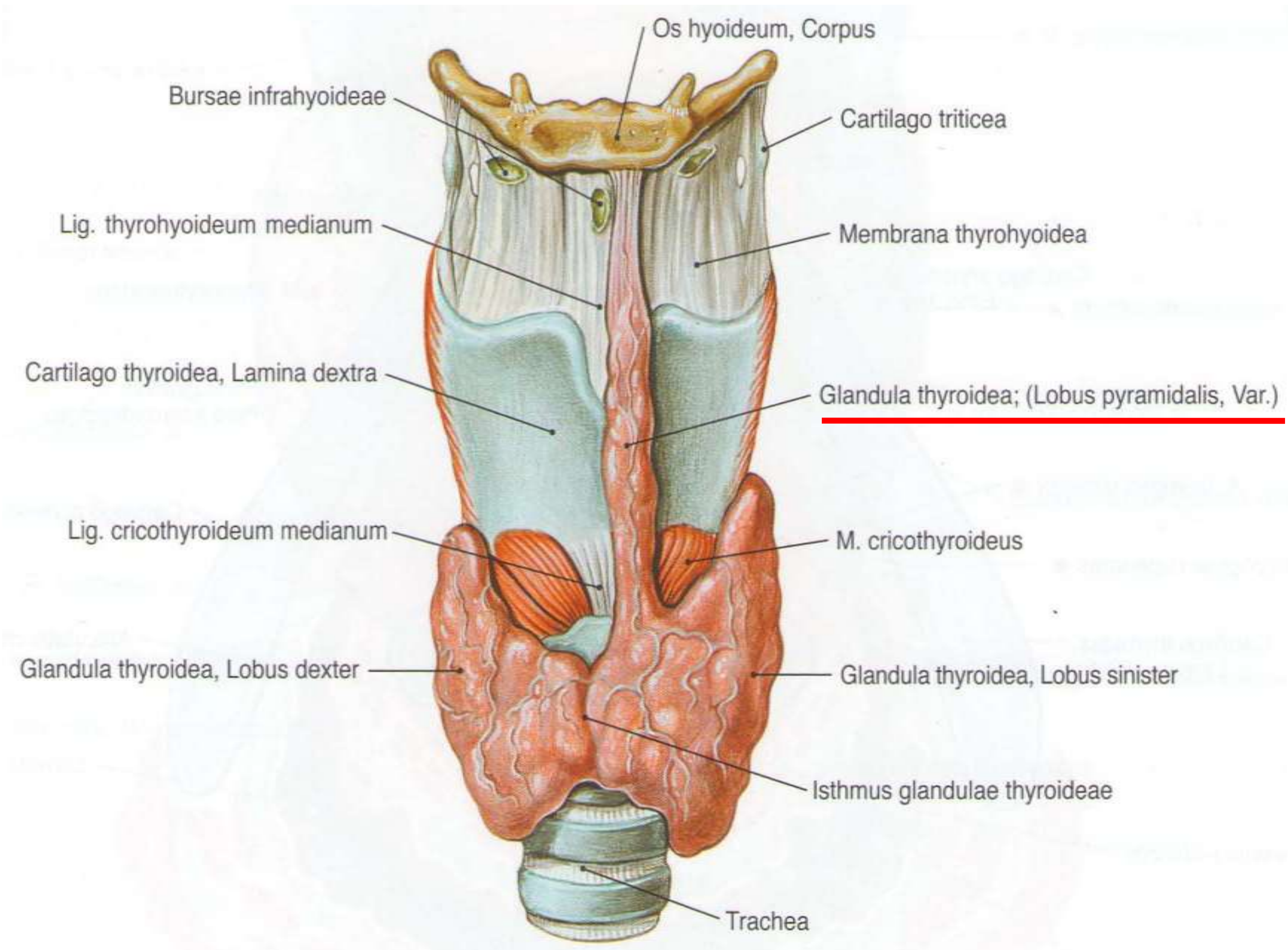
THYROID GLAND

- **Location:** Anterior region of neck, surrounding trachea
- **Primary Hormones:** Thyroxine (T-4) and Triiodothyronine (T-3)
- **Functions:** Regulate iodine; secrete TSH (thyroid stimulating hormone)
- **Major Disorders:** Goiter; Thyroid Cancer

Locating the Thyroid Gland

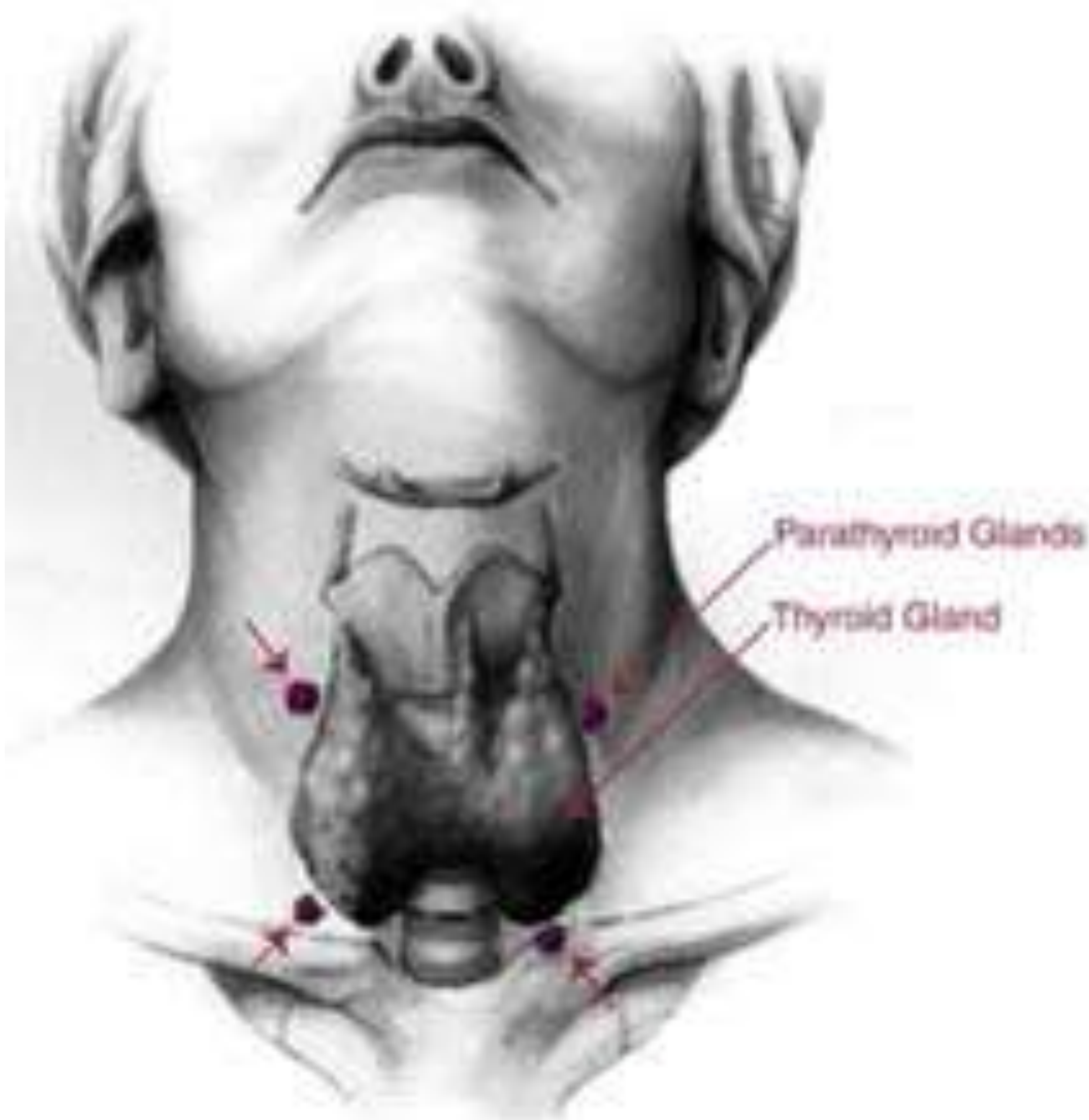


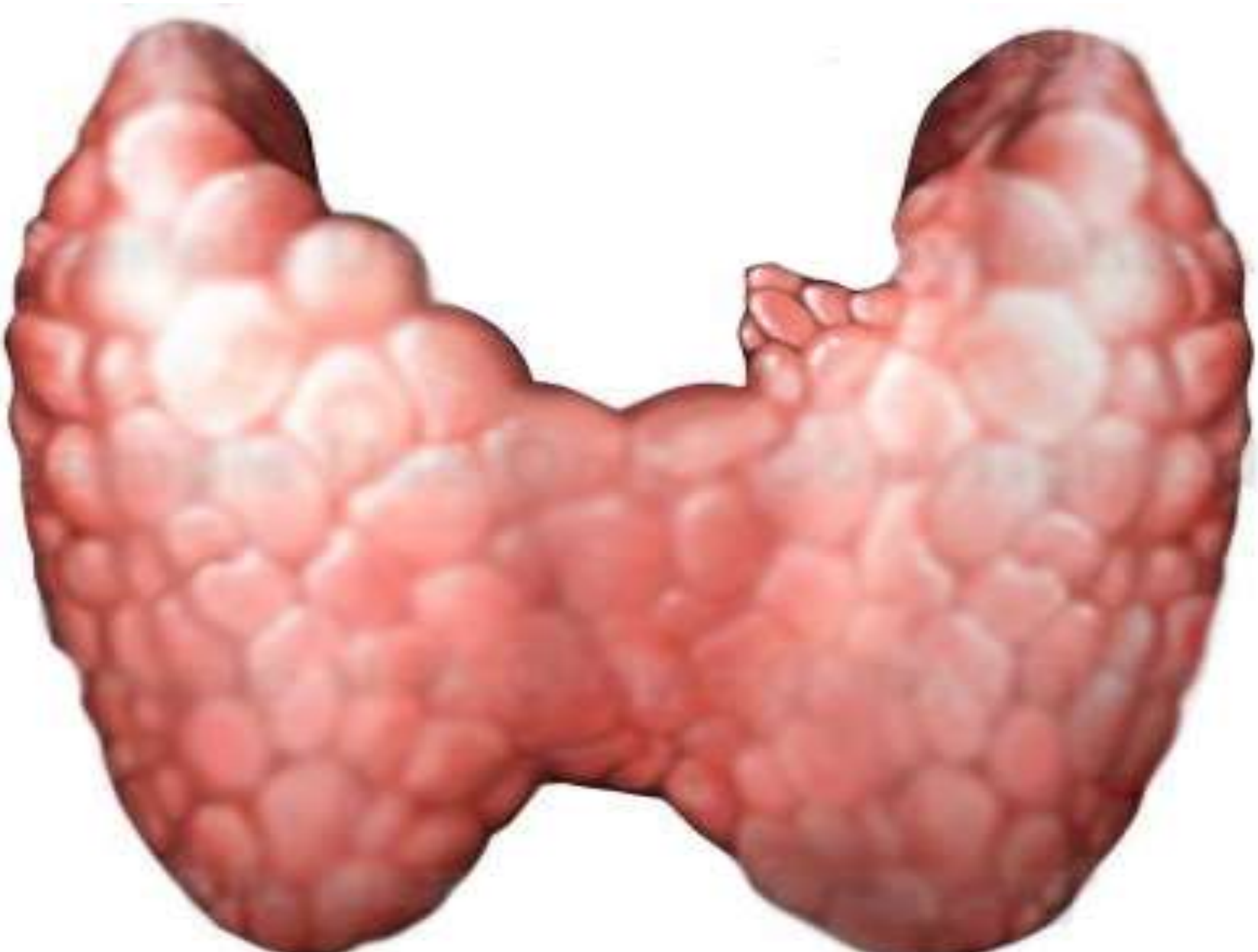

Cornu minus
Os hyoideum, Corpus
Cornu majus
Membrana thyrohyoidea
Cartilago thyroidea, Cornu superius
Prominentia laryngea
Cartilago thyroidea, Lamina dextra
Lig. cricothyroideum medianum
M. cricothyroideus
Glandula thyroidea, Lobus sinister
Glandula thyroidea, Lobus dexter
Isthmus glandulae thyroideae
Trachea

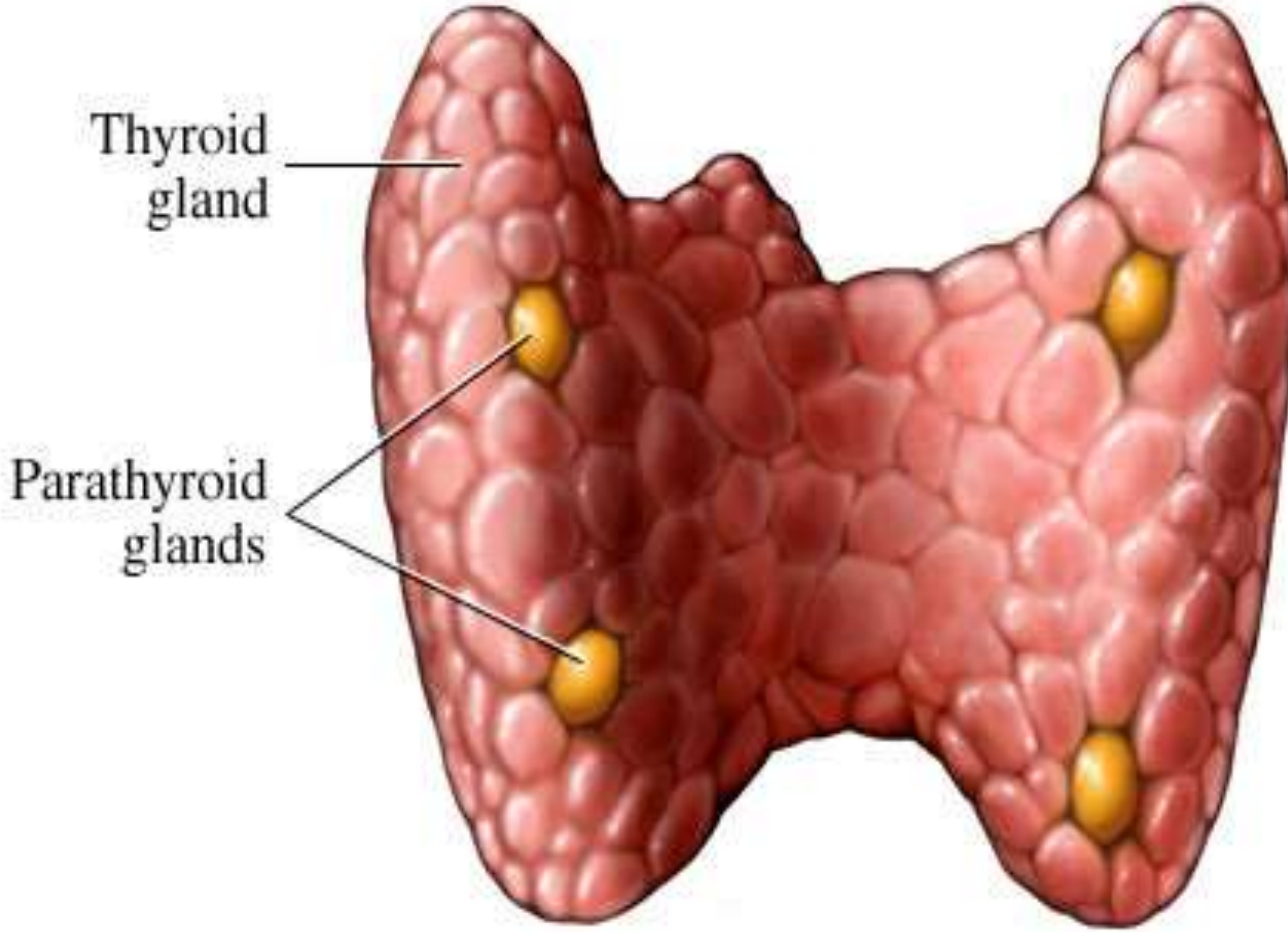

Os hyoideum, Corpus
Bursae infrahyoideae
Cartilago triticea
Lig. thyrohyoideum medianum
Membrana thyrohyoidea
Cartilago thyroidea, Lamina dextra
Glandula thyroidea; (Lobus pyramidalis, Var.)
Lig. cricothyroideum medianum
M. cricothyroideus
Glandula thyroidea, Lobus dexter
Glandula thyroidea, Lobus sinister
Isthmus glandulae thyroideae
Trachea

PARATHYROID GLAND

- **Location:** 4 glands around the thyroid
- **Primary Hormones:** PTH (parathyroid hormone)
- **Functions:** Affects the bones and kidneys; maintain calcium levels in the blood
- **Major Disorders:** Osteoporosis; Hyper- and Hypo- parathyroidism


Parathyroid Glands
Thyroid Gland

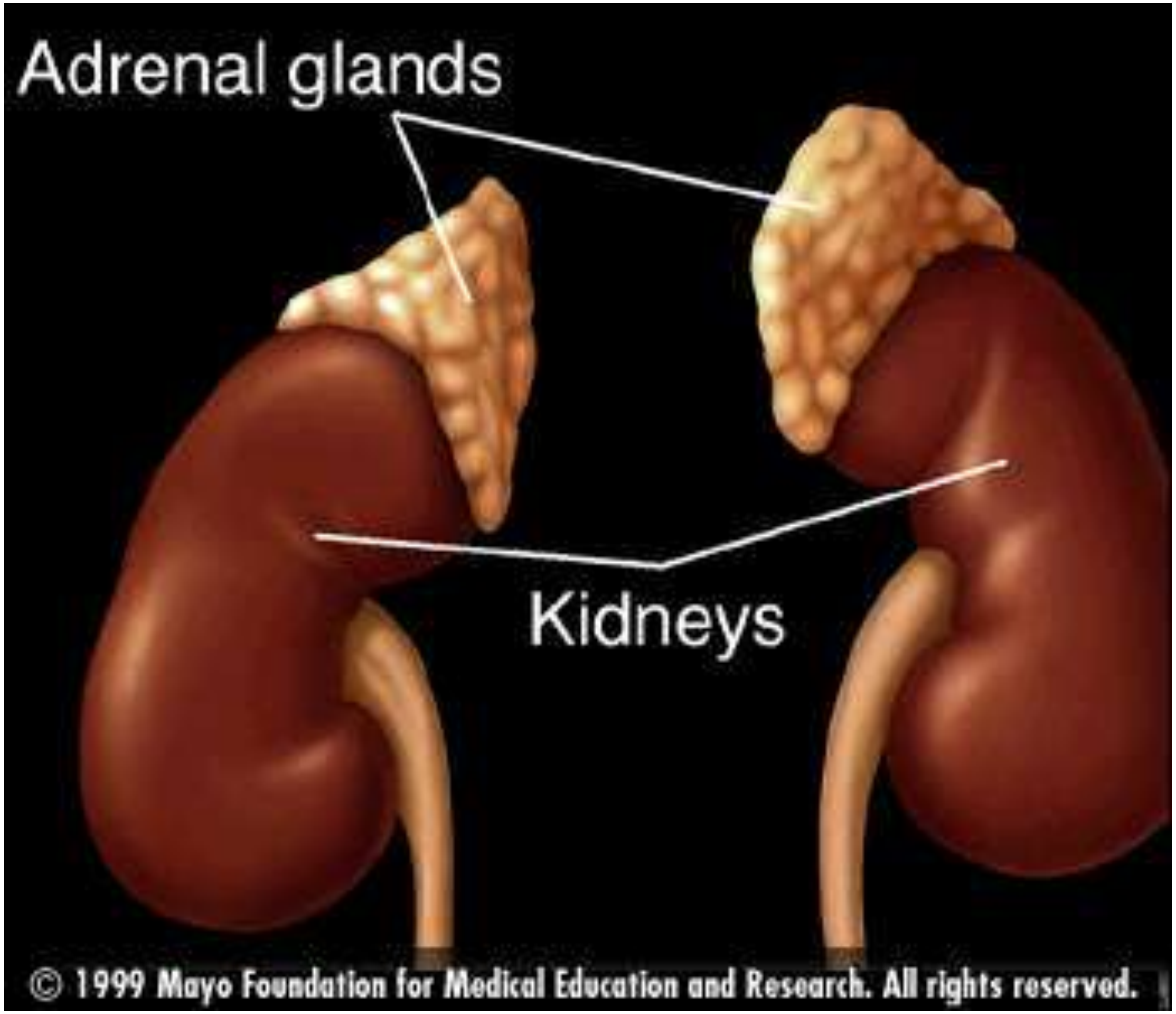



Thyroid
gland
Parathyroid
glands

ADRENAL GLAND

ADRENAL CORTEX

- **Location:** Atop the kidneys; outer layer surrounding the adrenal medulla
- **Primary Hormones:** Glococorticoids, Mineralcorticoids, and Aldosterone
- **Functions:** Maintain blood pressure, metabolism, levels of estrogen and testosterone
- **Major Disorders:** Addison's disease and Cushing's disease



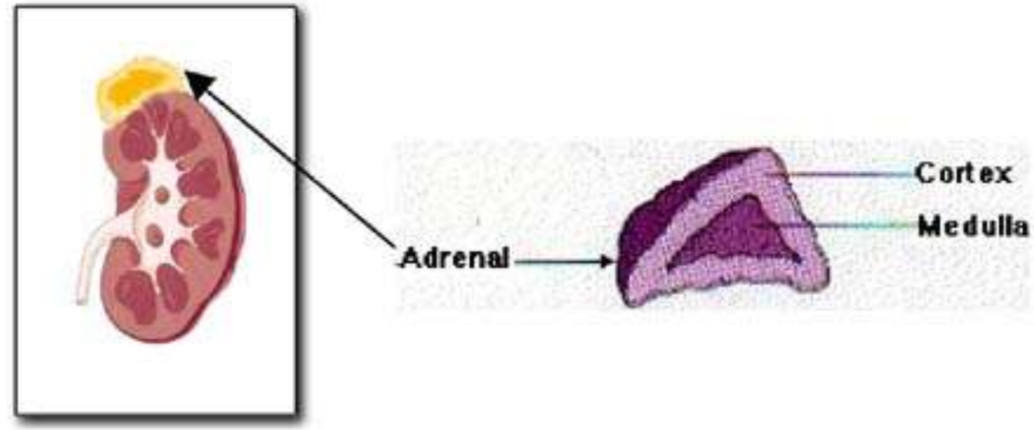
cut away of the
adrenal gland

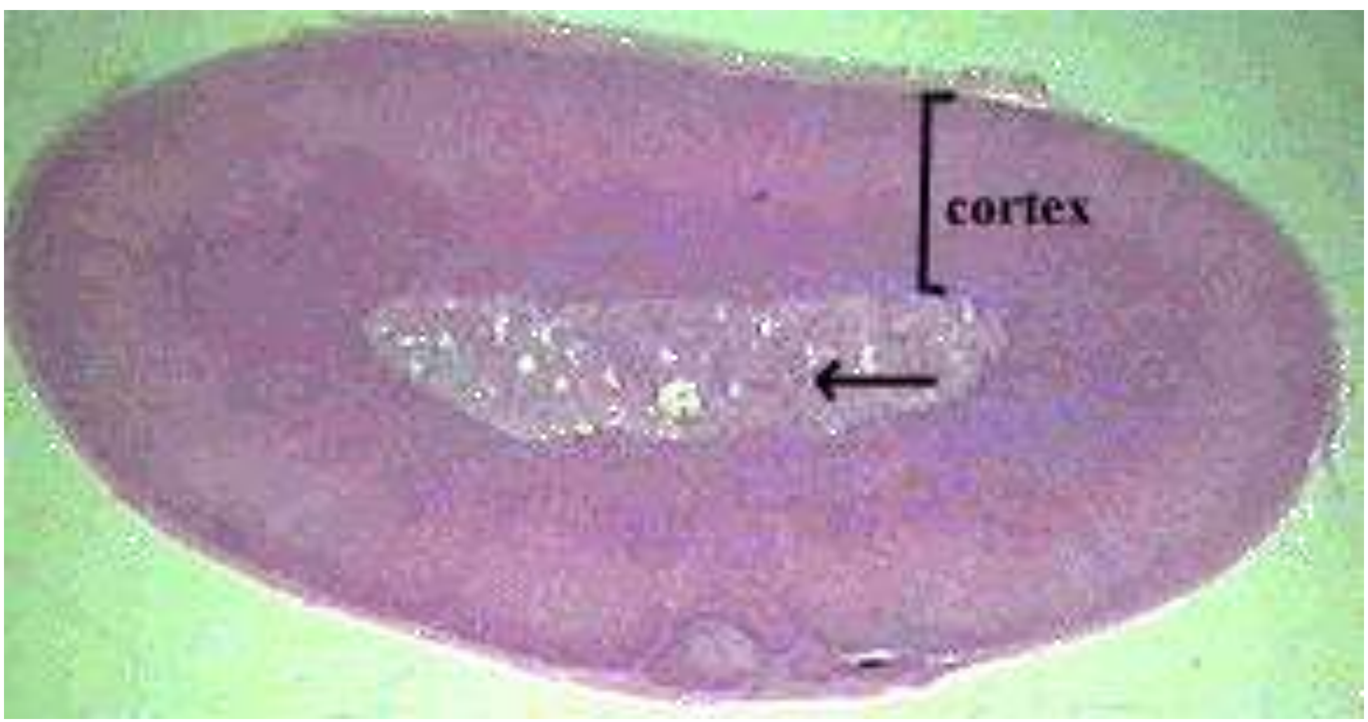
ADRENAL GLAND

ADRENAL MEDULLA

- **Location:** Atop the kidneys surrounded by the adrenal cortex; the adrenal pulp
- **Primary Hormones:** Epinephrine and Norepinephrine (a.k.a. Adrenaline)
- **Functions:** Increase blood pressure, heart rate, dilatation of pupils (adrenaline rush)
- **Major Disorders:** Pheochromocytoma

Anatomy of the adrenal glands



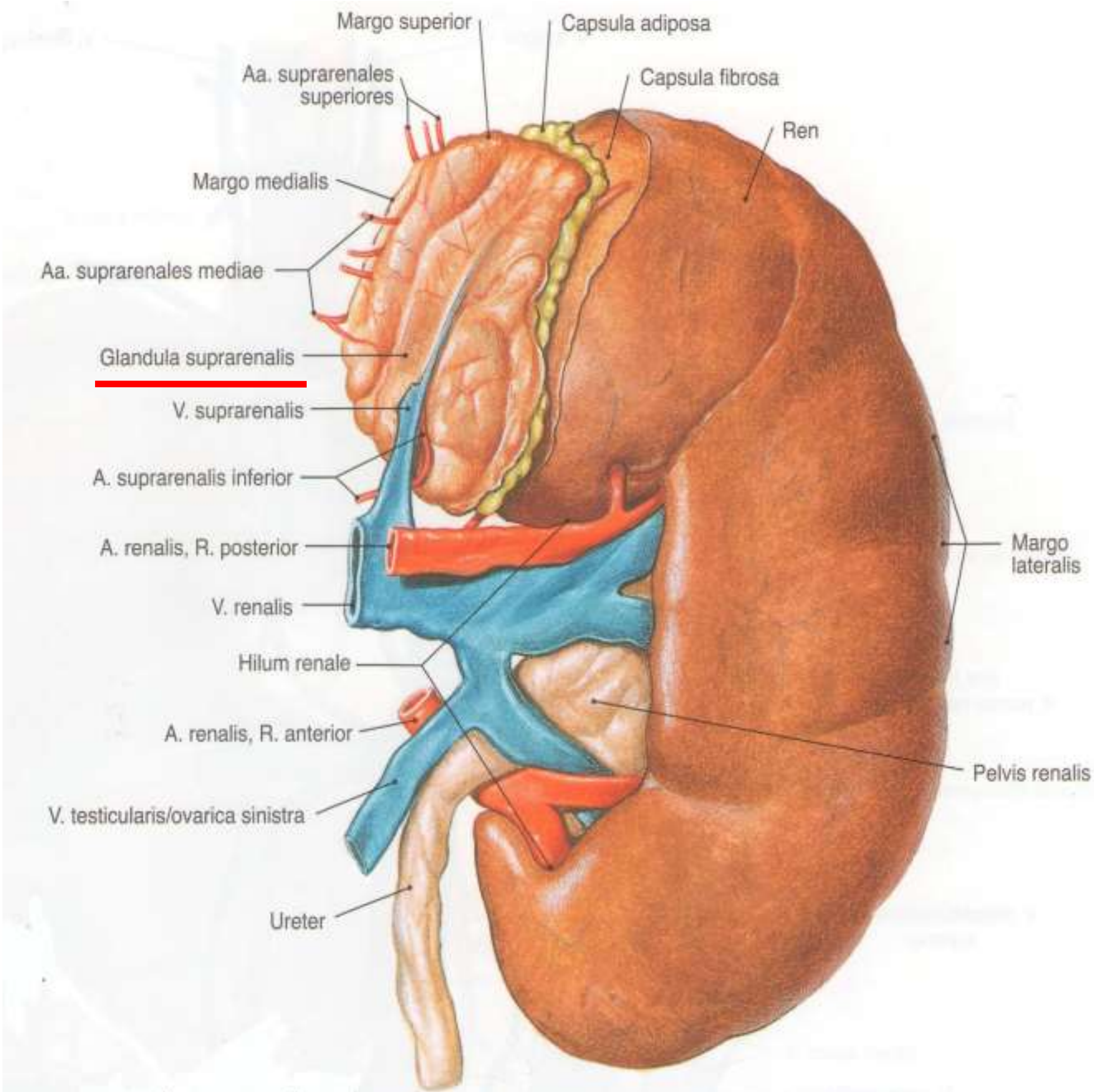


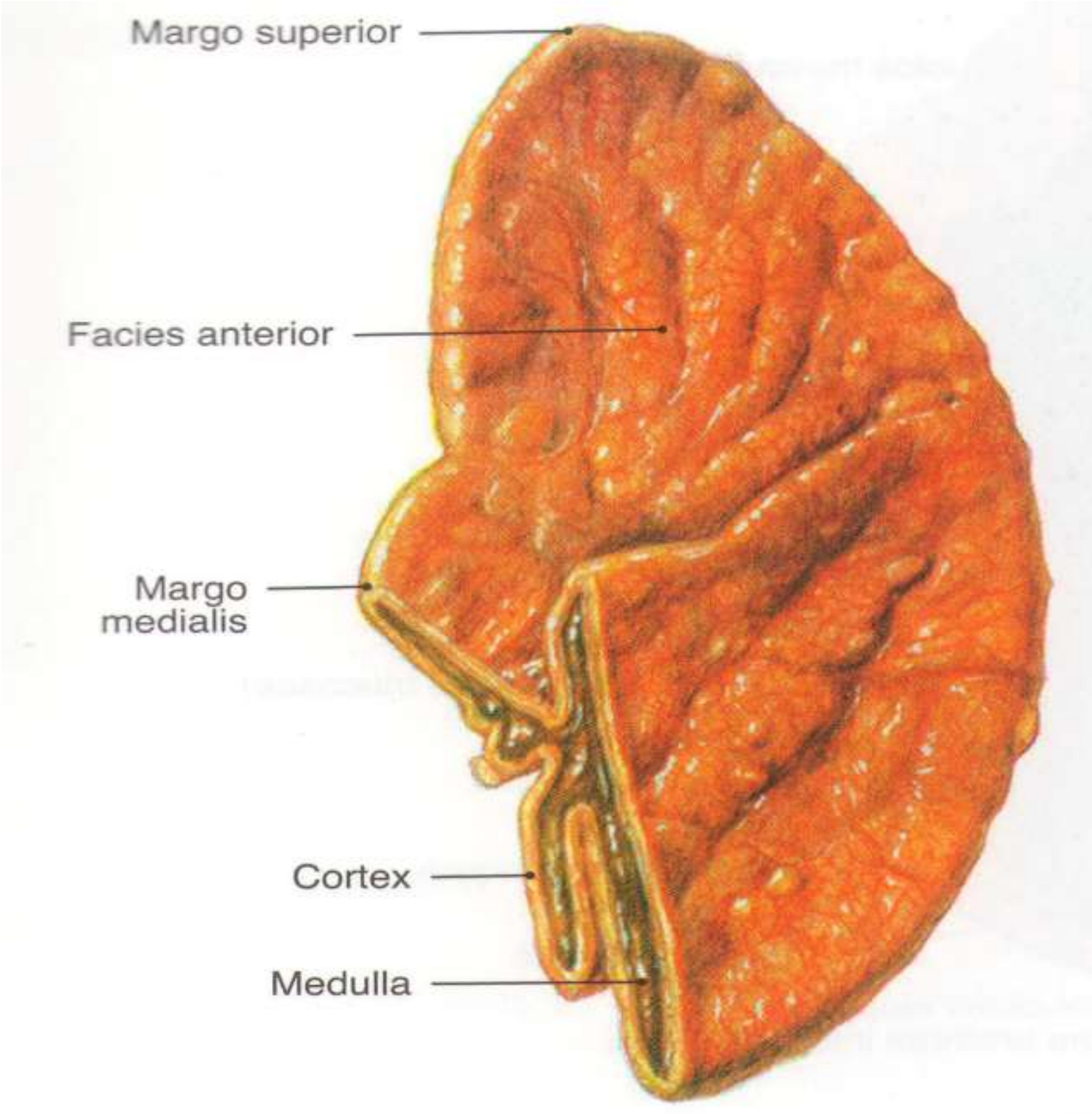
GLANDULA SUPRARENALIS

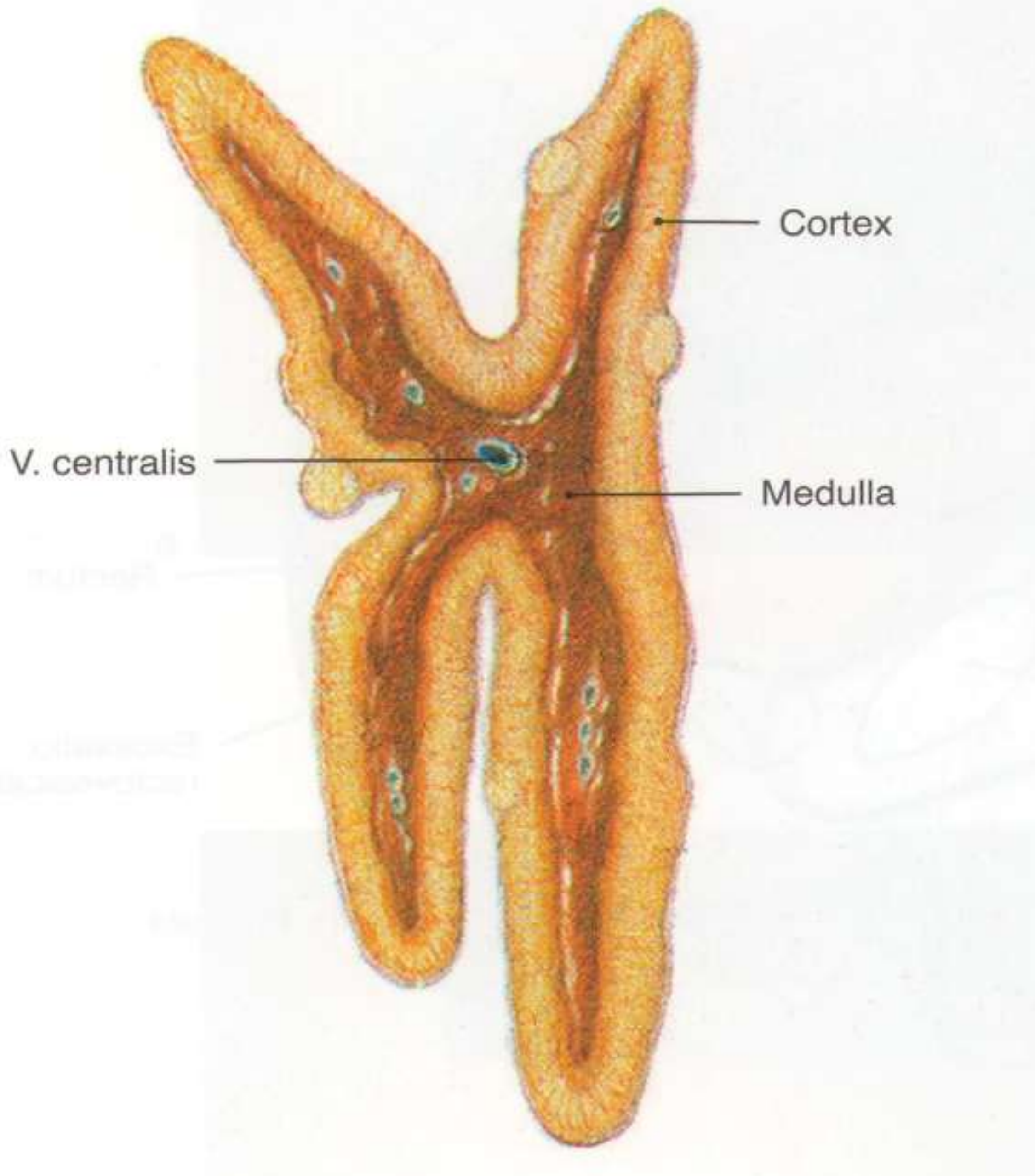
- cortex:

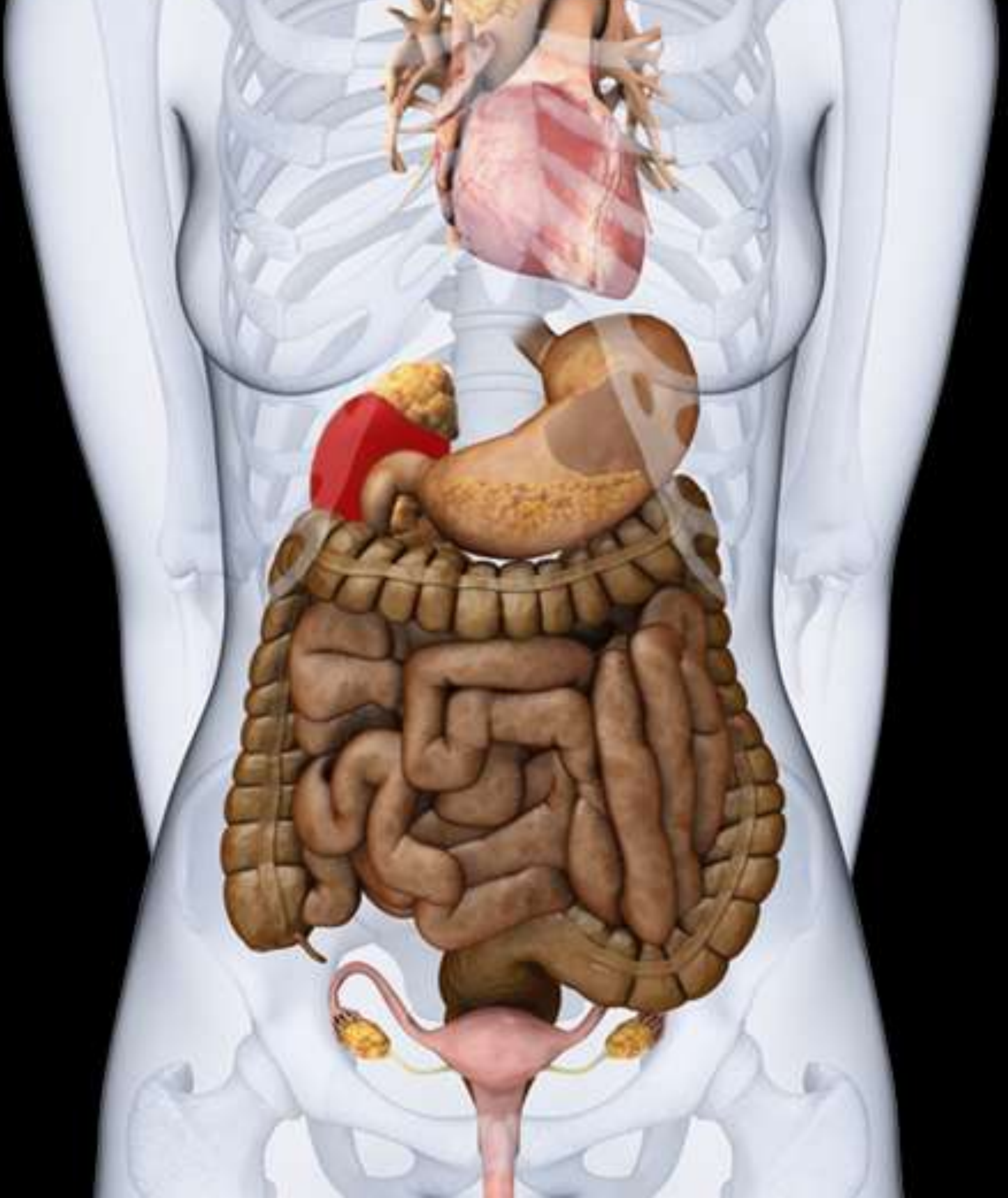
a) zona glomerulosa
b) zona fasciculata
c) zona reticularis

- medulla


Margo superior
Capsula adiposa
Aa. suprarenales superiores
Capsula fibrosa
Ren
Margo medialis
Aa. suprarenales mediae
Glandula suprarenalis
V. suprarenalis
A. suprarenalis inferior
A. renalis, R. posterior
Margo lateralis
V. renalis
Hilum renale
A. renalis, R. anterior
Pelvis renalis
V. testicularis/ovarica sinistra
Ureter


Margo superior
Facies anterior
Margo medialis
Cortex
Medulla


Cortex
V. centralis
Medulla

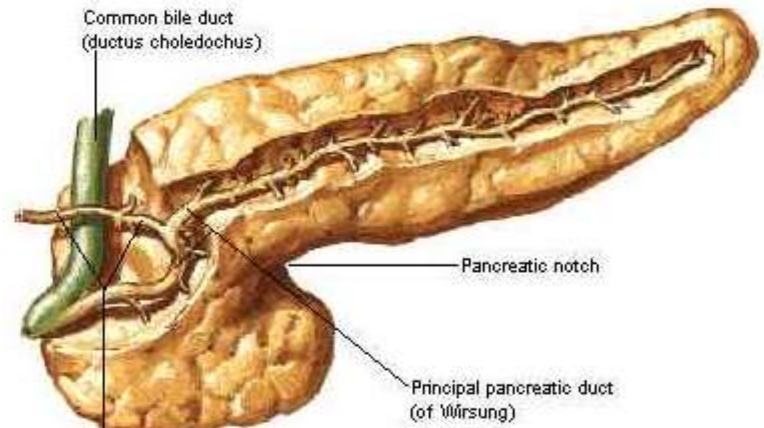


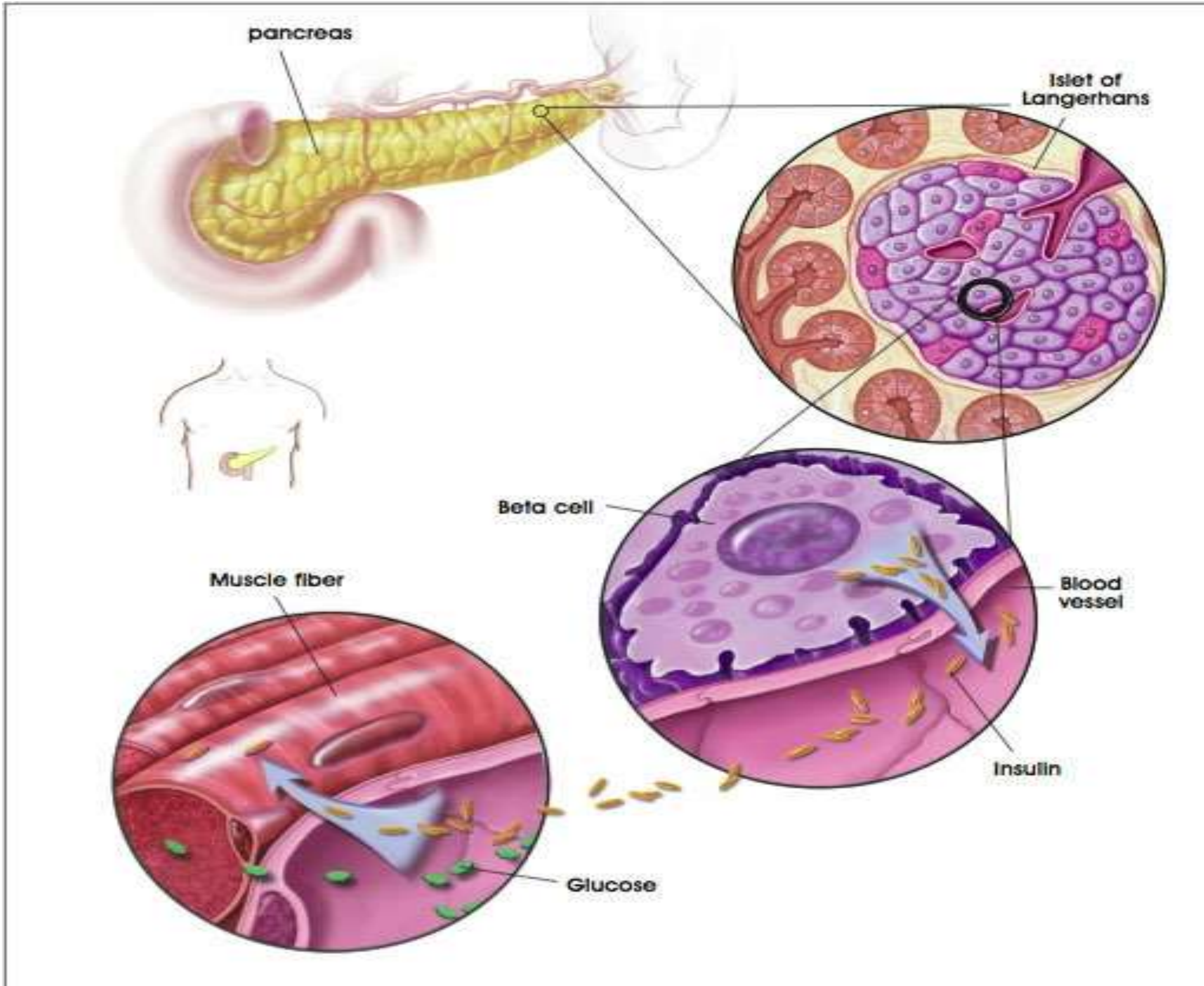
Kidney

Erythropoietin, which is secreted by the kidneys, stimulates the production of red blood cells in bone marrow.

PANCREAS

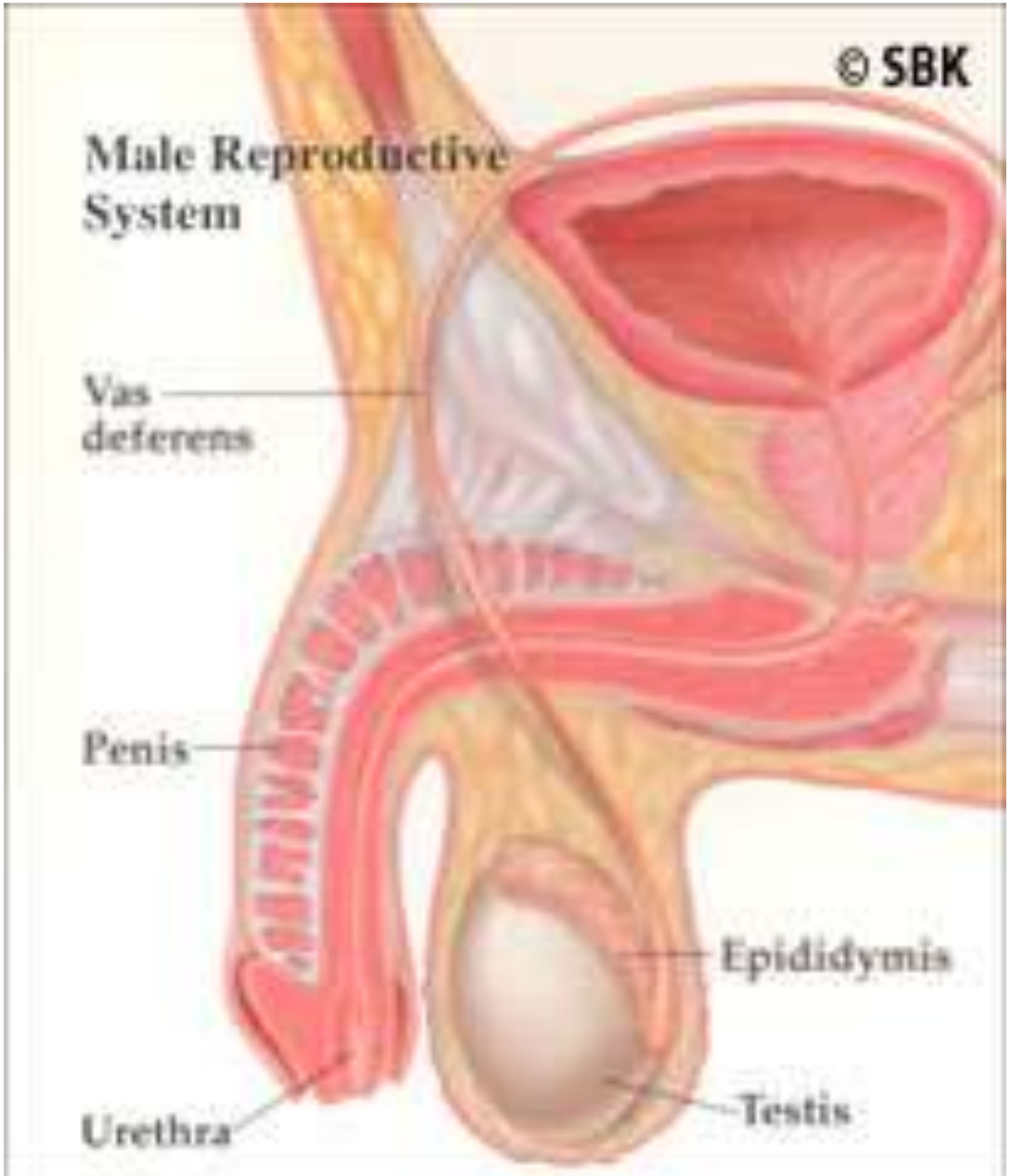
- **Location:** Around the stomach and small intestine
- **Primary Hormones:** Insulin and Glucagon (secreted by the pancreatic islets or the Islets of Langerhans)
- **Functions:** Digestion of enzymes; regulate blood-glucose levels; insulin uptake
- **Major Disorders:** Diabetes mellitus types I and II


Common bile duct
(ductus choledochus)
Pancreatic notch
Principal pancreatic duct
(of Wirsung)
Accessory pancreatic duct
(of Santorini)


pancreas
Islet of Langerhans
Beta cell
Blood vessel
Muscle fiber
Insulin
Glucose

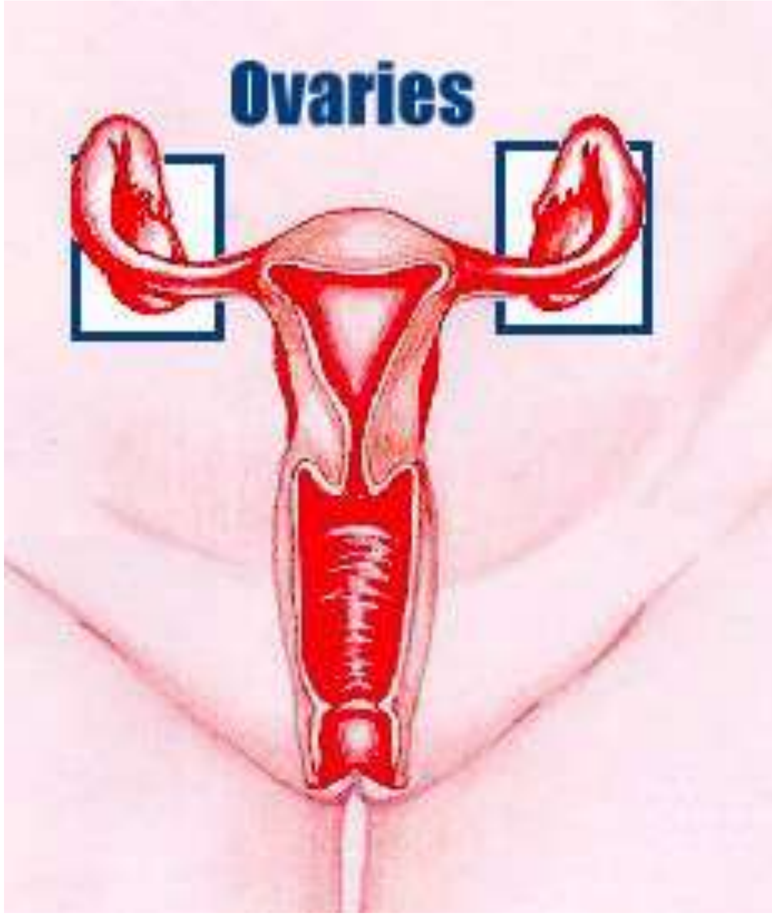
TESTES

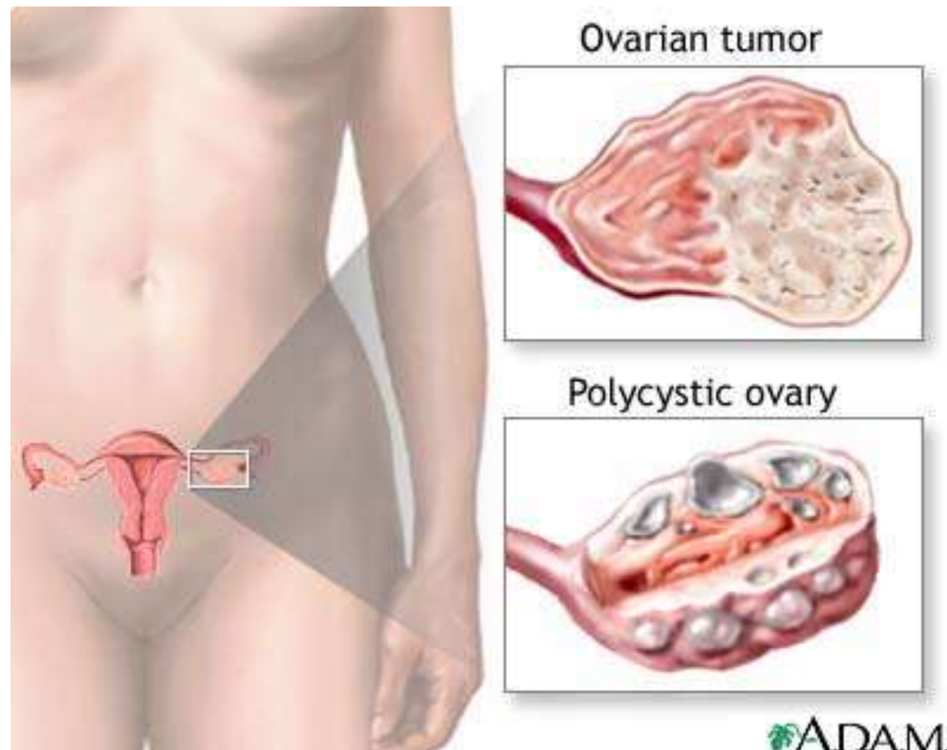
- **Location:** Within the scrotum
- **Primary Hormones:** Testosterone; ICSH
- **Functions:** Produce sperm and testosterone; primary and secondary sex characteristics
- **Major Disorders:** Testicular cancer; Germ-cell tumors

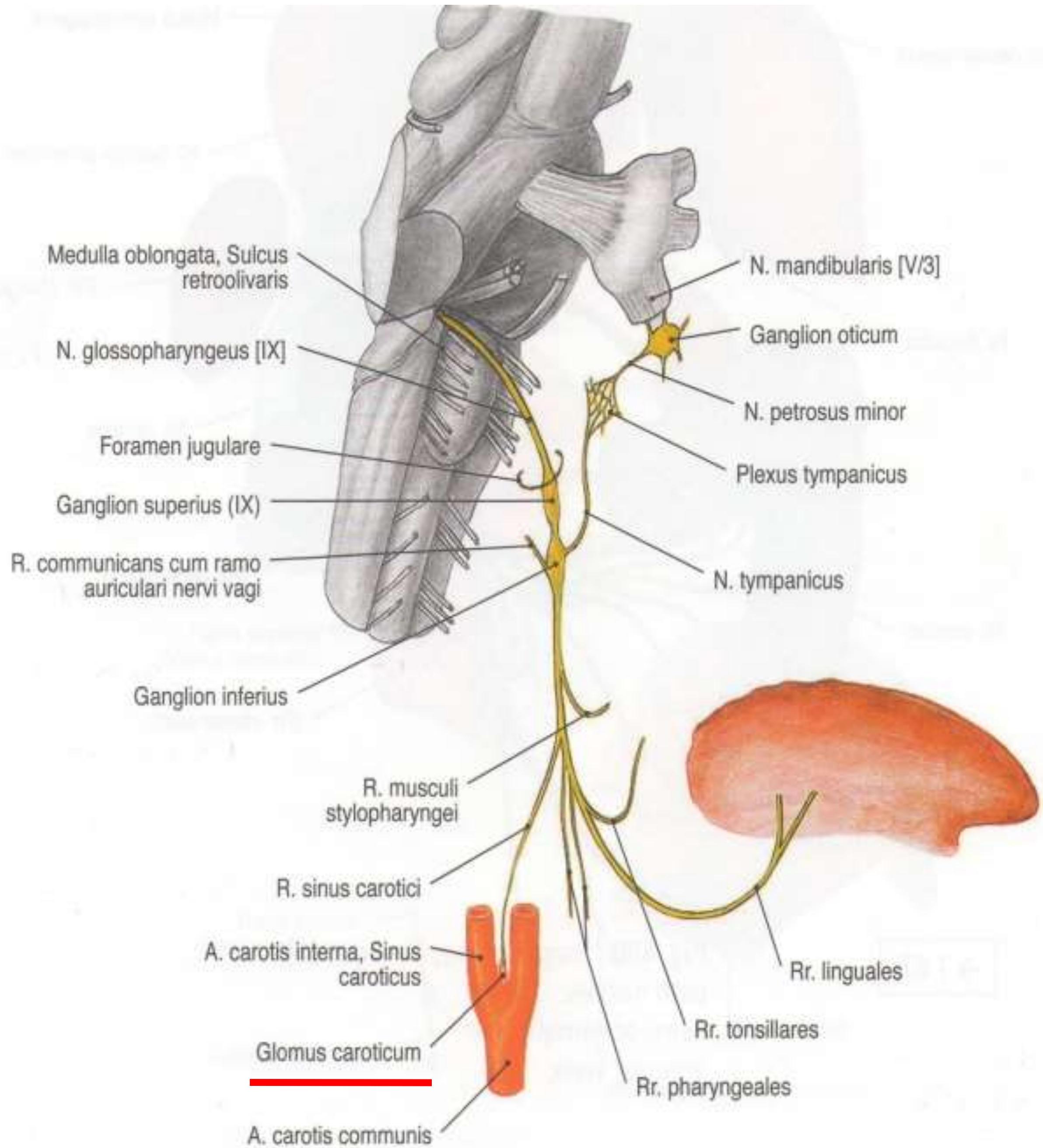

© SBK
Male Reproductive System
Vas deferens
Penis
Urethra
Epididymis
Testis

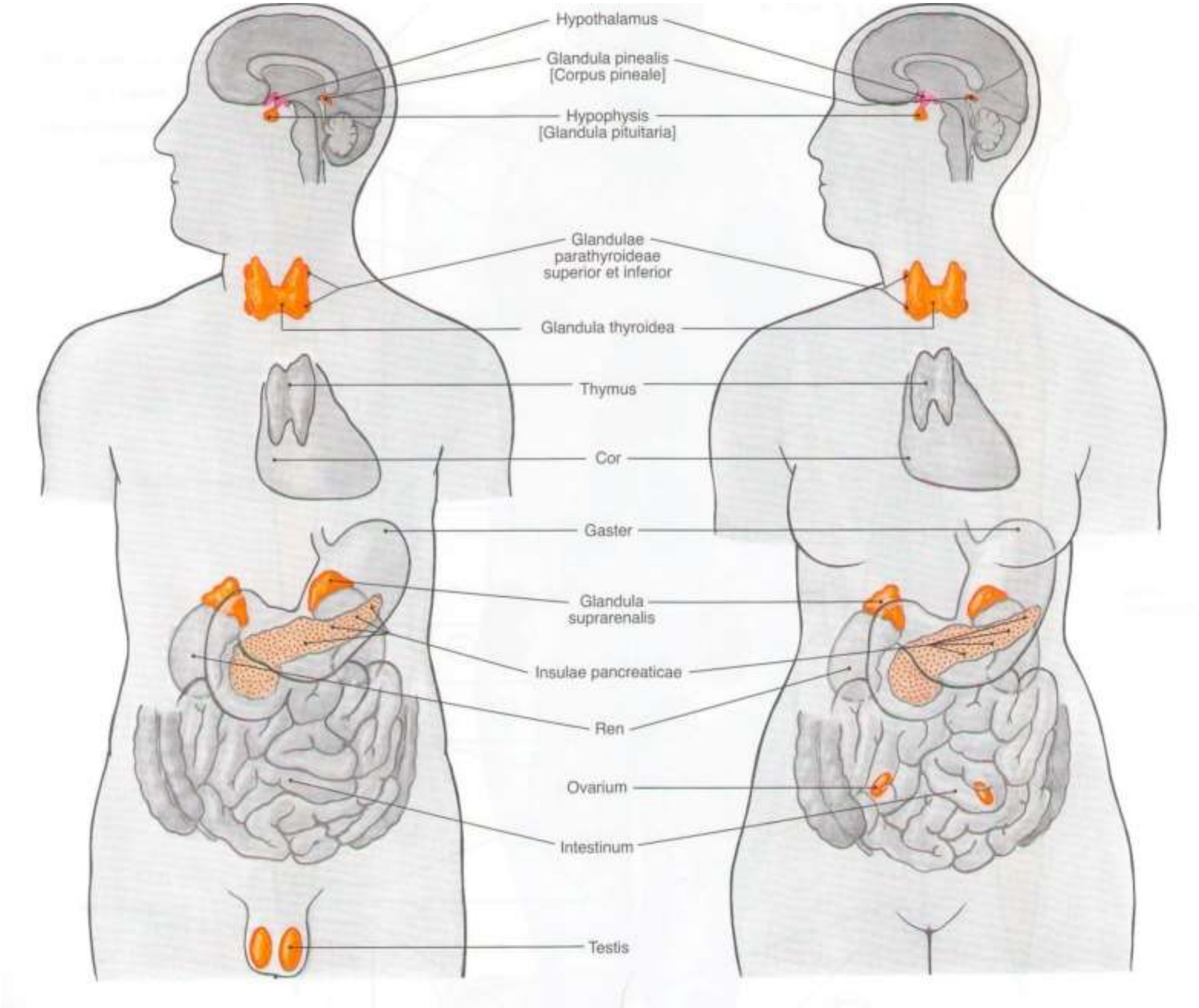
OVARIES

- **Location:** In the abdomen at the end of the fallopian tubes
- **Primary Hormones:** Estrogen, Progestins, Estradiol
- **Functions:** Produce female gametes; ova and ovum; oocytes- immature gametes
- **Major Disorders:** Ovarian Cancer


Ovaries


Ovarian tumor
Polycystic ovary
A.D.A.M.


Medulla oblongata, Sulcus retroolivaris
N. glossopharyngeus [IX]
Foramen jugulare
Ganglion superius (IX)
R. communicans cum ramo auriculari nervi vagi
Ganglion inferius
R. musculi stylopharyngei
R. sinus carotici
A. carotis interna, Sinus caroticus
Glomus caroticum
A. carotis communis
N. mandibularis [V/3]
Ganglion oticum
N. petrosus minor
Plexus tympanicus
N. tympanicus
Rr. linguales
Rr. tonsillares
Rr. pharyngeales


Hypothalamus
Glandula pinealis
[Corpus pineale]
Hypophysis
[Glandula pituitaria]
Glandulae
parathyroideae
superior et inferior
Glandula thyroidea
Thymus
Cor
Gaster
Glandula
suprarenalis
Insulae pancreaticae
Ren
Ovarium
Intestinum
Testis

THE ADRENAL GLANDS

(Glandula suprarenalis)

HUMAN BODY DVD-ROM

- CELLS AND DNA
- SKELETAL SYSTEM
- MUSCULAR SYSTEM
- NERVOUS SYSTEM
- ENDOCRINE SYSTEM
- CARDIOVASCULAR SYSTEM
- RESPIRATORY SYSTEM
- SKIN, HAIR, AND NAILS
- LYMPH AND IMMUNITY
- DIGESTIVE SYSTEM
- URINARY SYSTEM
- REPRODUCTIVE SYSTEM
- DEVELOPMENT
- ANIMATIONS

SYSTEM

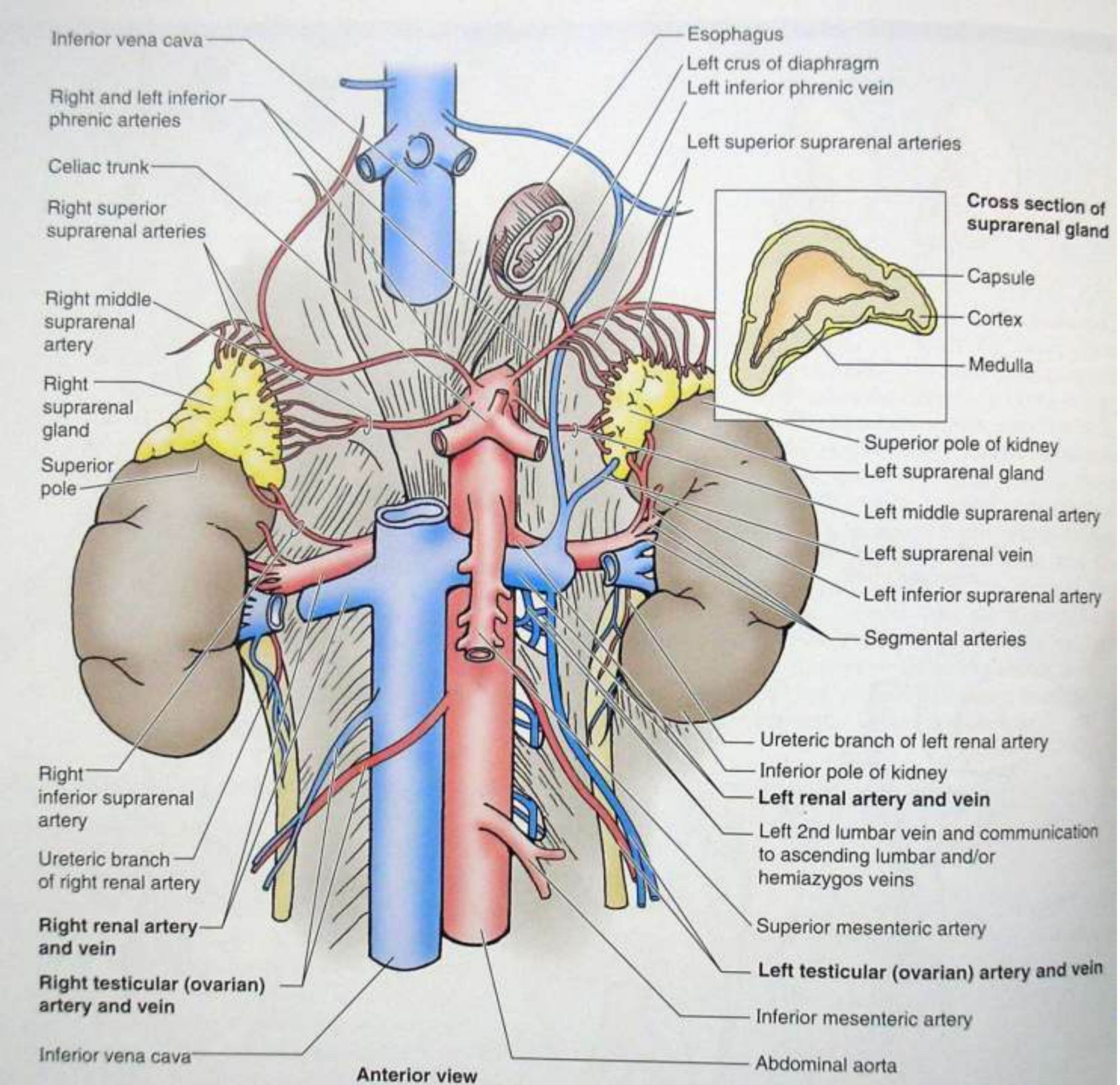
ENDOCRINE SYSTEM

The body's chemical messengers (hormones) are made by endocrine glands. These glands have no ducts but secrete their hormones directly into the blood, by which means they reach every cell in the body. Hormones affect certain target tissues or organs and regulate their activities. For more detail, see *The Human Body Book*, pp.122–23.

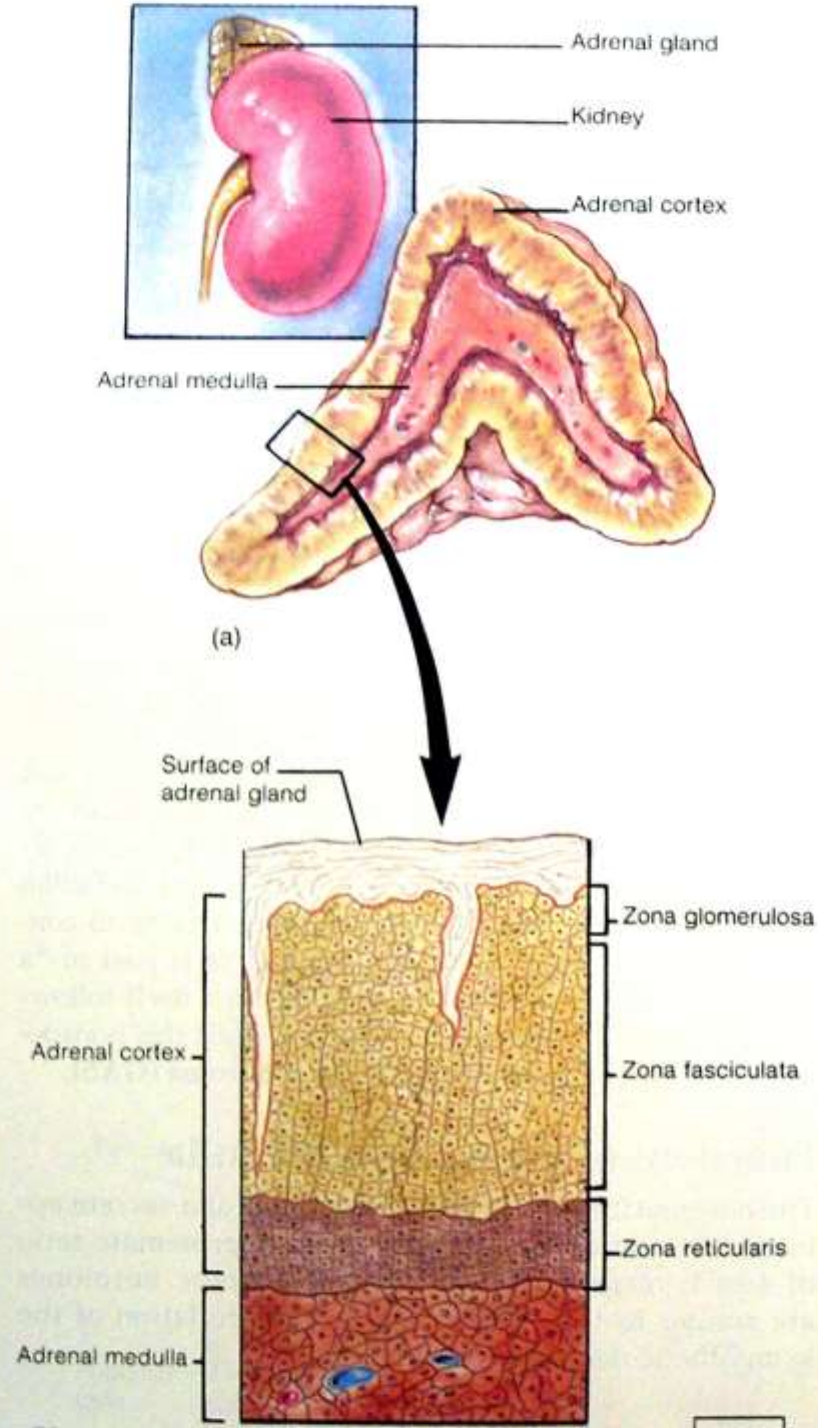
Adrenal gland

The cortex (outer layer) manufactures steroid hormones that regulate metabolism of glucose, sodium, potassium and fluid balance; the medulla (inner layer) produces adrenaline

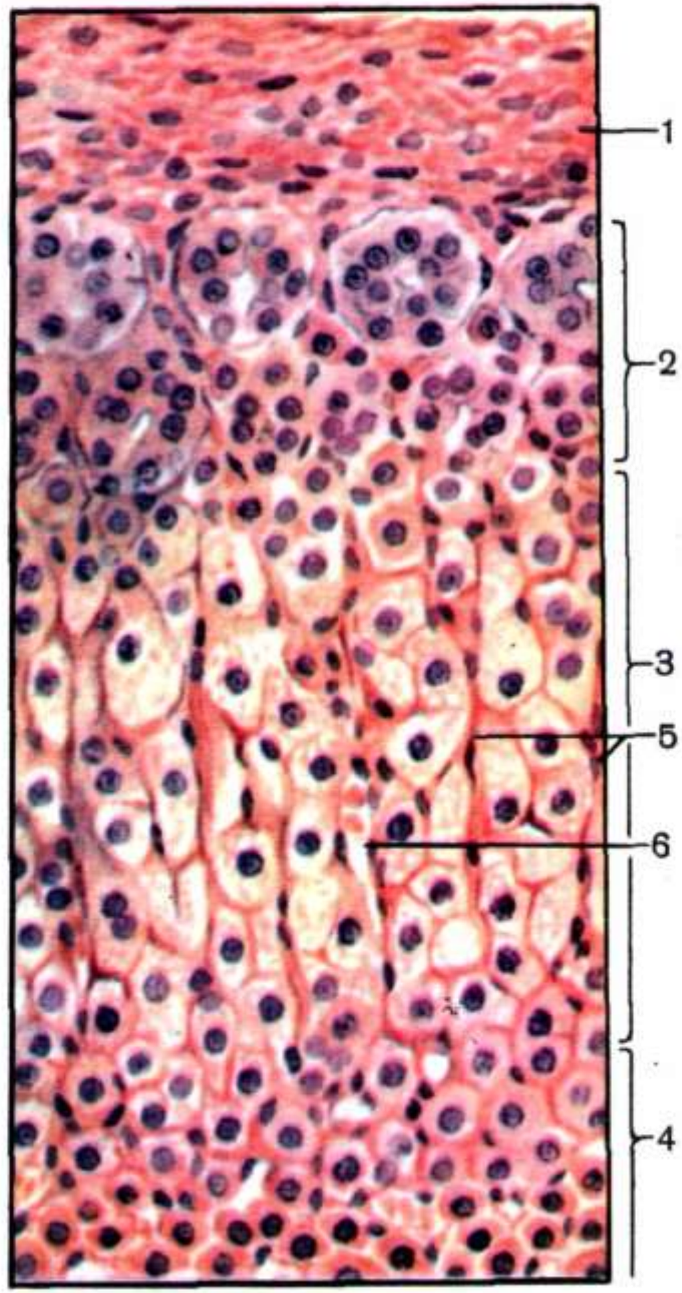
HOME | QUIT



Inferior vena cava
Right and left inferior phrenic arteries
Celiac trunk
Right superior suprarenal arteries
Right middle suprarenal artery
Right suprarenal gland
Superior pole
Right inferior suprarenal artery
Ureteric branch of right renal artery
Right renal artery and vein
Right testicular (ovarian) artery and vein
Inferior vena cava
Anterior view
Esophagus
Left crus of diaphragm
Left inferior phrenic vein
Left superior suprarenal arteries
Cross section of suprarenal gland
Capsule
Cortex
Medulla
Superior pole of kidney
Left suprarenal gland
Left middle suprarenal artery
Left suprarenal vein
Left inferior suprarenal artery
Segmental arteries
Ureteric branch of left renal artery
Inferior pole of kidney
Left renal artery and vein
Left 2nd lumbar vein and communication to ascending lumbar and/or hemiazygos veins
Superior mesenteric artery
Left testicular (ovarian) artery and vein
Inferior mesenteric artery
Abdominal aorta


Adrenal gland
Kidney
Adrenal cortex
Adrenal medulla
(a)
Surface of adrenal gland
Zona glomerulosa
Zona fasciculata
Zona reticularis
Adrenal cortex
Adrenal medulla

THE ADRENAL GLANDS - A PAIRED ORGAN LOCATED ON THE UPPER POLE OF THE KIDNEY.



Гістологічна будова надниркової залози

Adrenal cortex consists of three zones:

- **Zona glomerulosa,**
- **Zona fasciculata,**
- **Zona reticularis.**

PARAGANGLIA

The group of glands of the suprarenal system also include paraganglia or chromaffin cells.

The term "paraganglia" was first used by Kohn in 1903.

Paraganglia:

corpora paraaortica, aortic body

located on the all sides and above the aortic bifurcation. The largest of the aortic paraganglia is paragangliy, which is located at the beginning of the lower mesenteric artery - (Zuckerkandl's body);

glomus caroticum - located at the bifurcation of the common carotid artery:

glomus coccygeum - located the end of a. sacralis mediana;

paraganglion supracardiacum - located in the heart area, two of them – on the top and bottom.

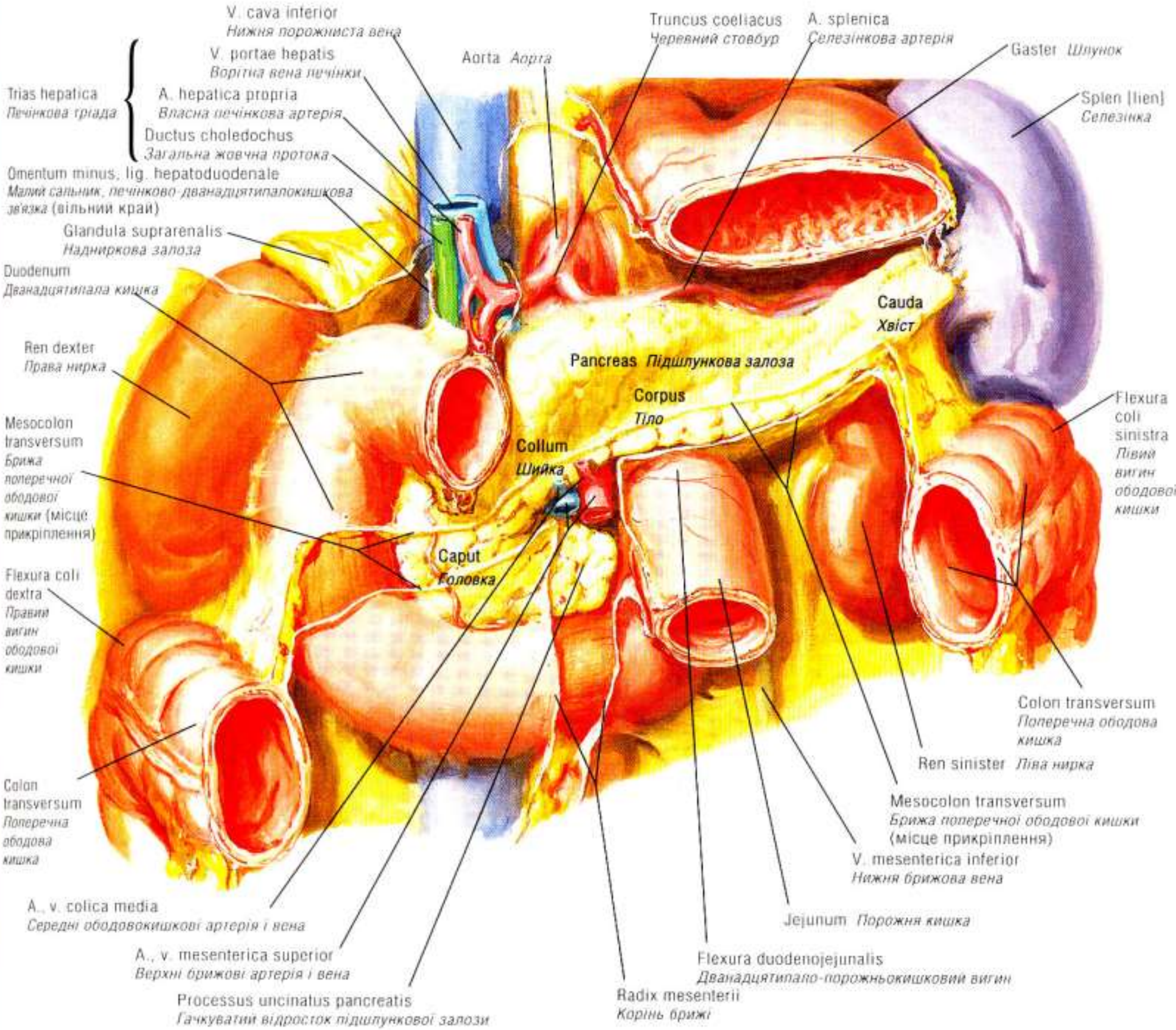
PANCREAS

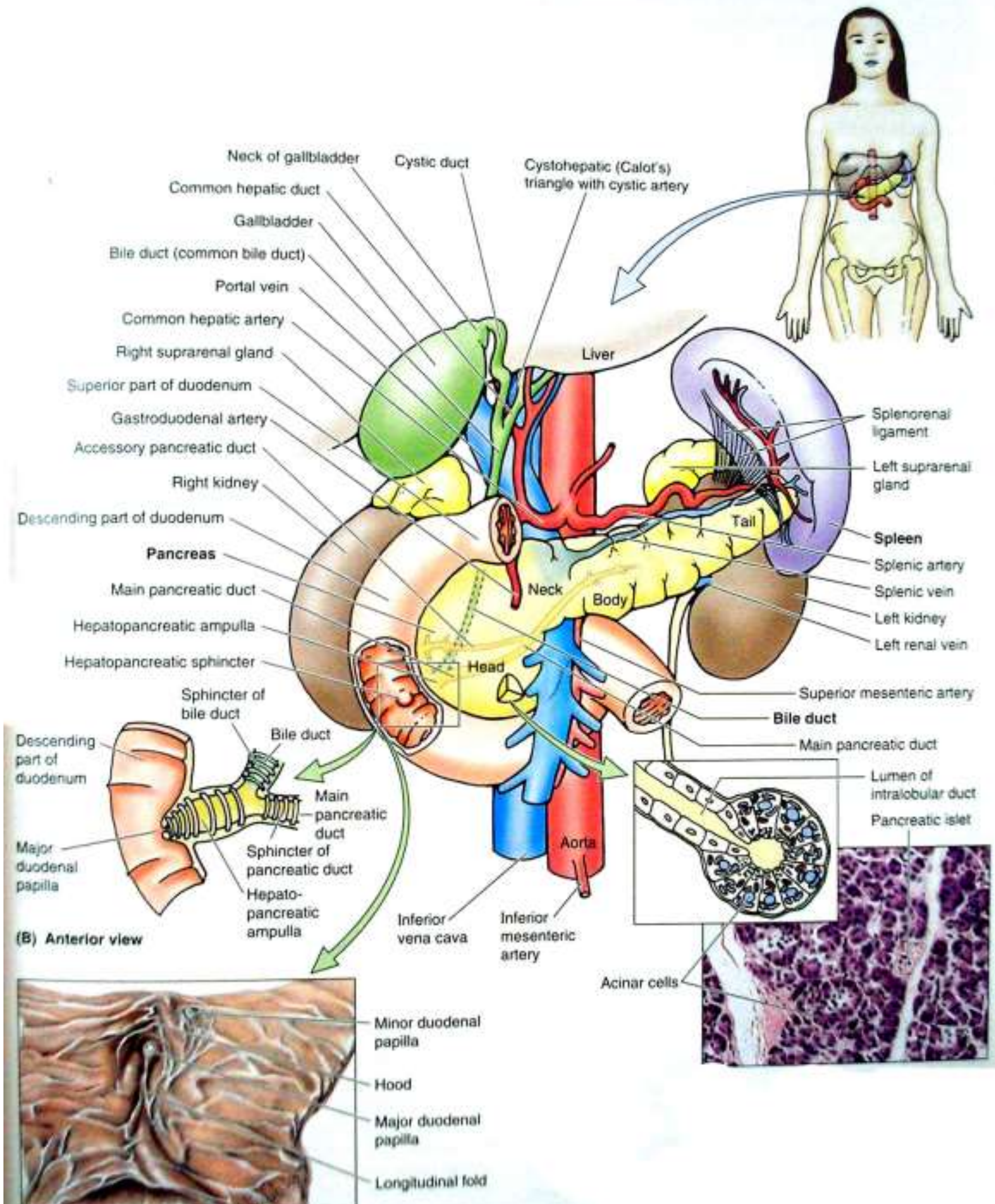
ENDOCRINE SYSTEM

The body's chemical messengers (hormones) are made by endocrine glands. These glands have no ducts but secrete their hormones directly into the blood, by which means they reach every cell in the body. Hormones affect certain target tissues or organs and regulate their activities. For more detail, see *The Human Body Book*, pp.122–23.

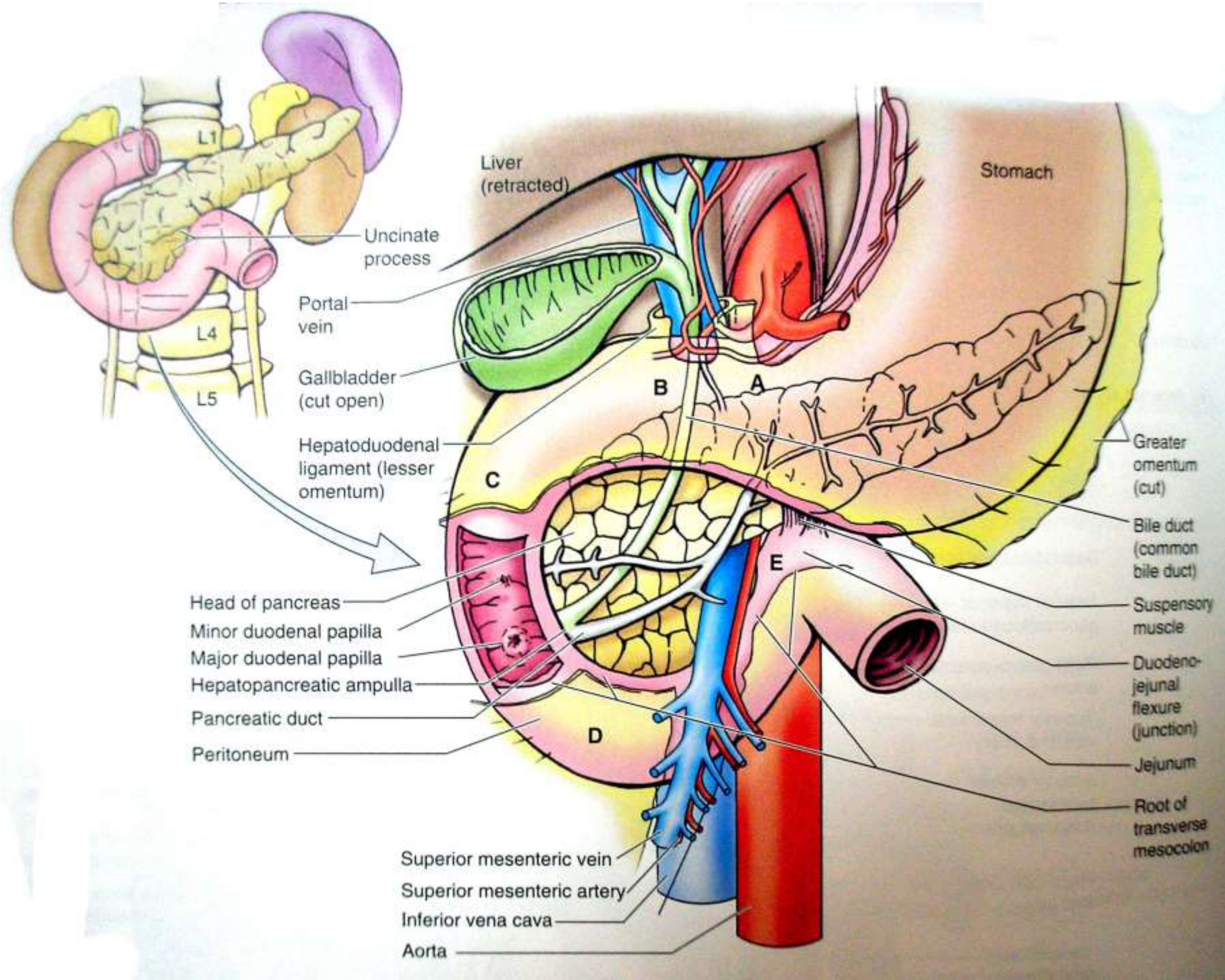
Pancreas

Clusters of cells known as islets of Langerhans produce two hormones, insulin and glucagon, that respectively lower and raise blood glucose levels in the body's energy control mechanism

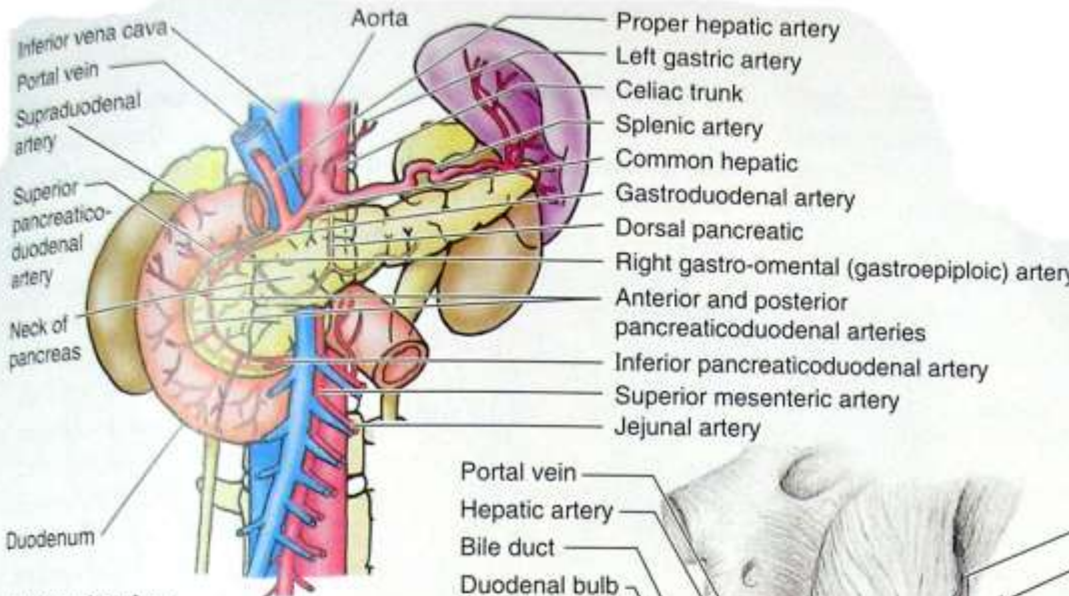

V. cava inferior
Нижня порожниста вена
Trias hepatica
Печінкова тріада
V. portae hepatis
Ворітна вена печінки
A. hepatica propria
Власна печінкова артерія
Ductus choledochus
Загальна жовчна протока
Omentum minus, lig. hepatoduodenale
Малий сальник, печінково-дванадцятипалокишкова зв'язка (вільний край)
Glandula suprarenalis
Надниркова залоза
Duodenum
Дванадцятипала кишка
Ren dexter
Права нирка
Mesocolon transversum
Брижа поперечної ободової кишки (місце прикріплення)
Flexura coli dextra
Правий вигин ободової кишки
Colon transversum
Поперечна ободова кишка
Aorta Аорта
Truncus coeliacus
Черевний стовбур
A. splenica
Селезінкова артерія
Gaster Шлунок
Splen [lien]
Селезінка
Cauda
Хвіст
Pancreas Підшлункова залоза
Corpus
Тіло
Collum
Шийка
Caput
Головка
Flexura coli sinistra
Лівий вигин ободової кишки
Colon transversum
Поперечна ободова кишка
Ren sinister Ліва нирка
Mesocolon transversum
Брижа поперечної ободової кишки (місце прикріплення)
V. mesenterica inferior
Нижня брижова вена
Jejunum Порожня кишка
Flexura duodenojejunalis
Дванадцятипало-порожньокишковий вигин
Radix mesenterii
Корінь брижі
Processus uncinatus pancreatis
Гачкуватий відросток підшлункової залози
A., v. mesenterica superior
Верхні брижові артерія і вена
A., v. colica media
Середні ободовокишкові артерія і вена



(B) Anterior view

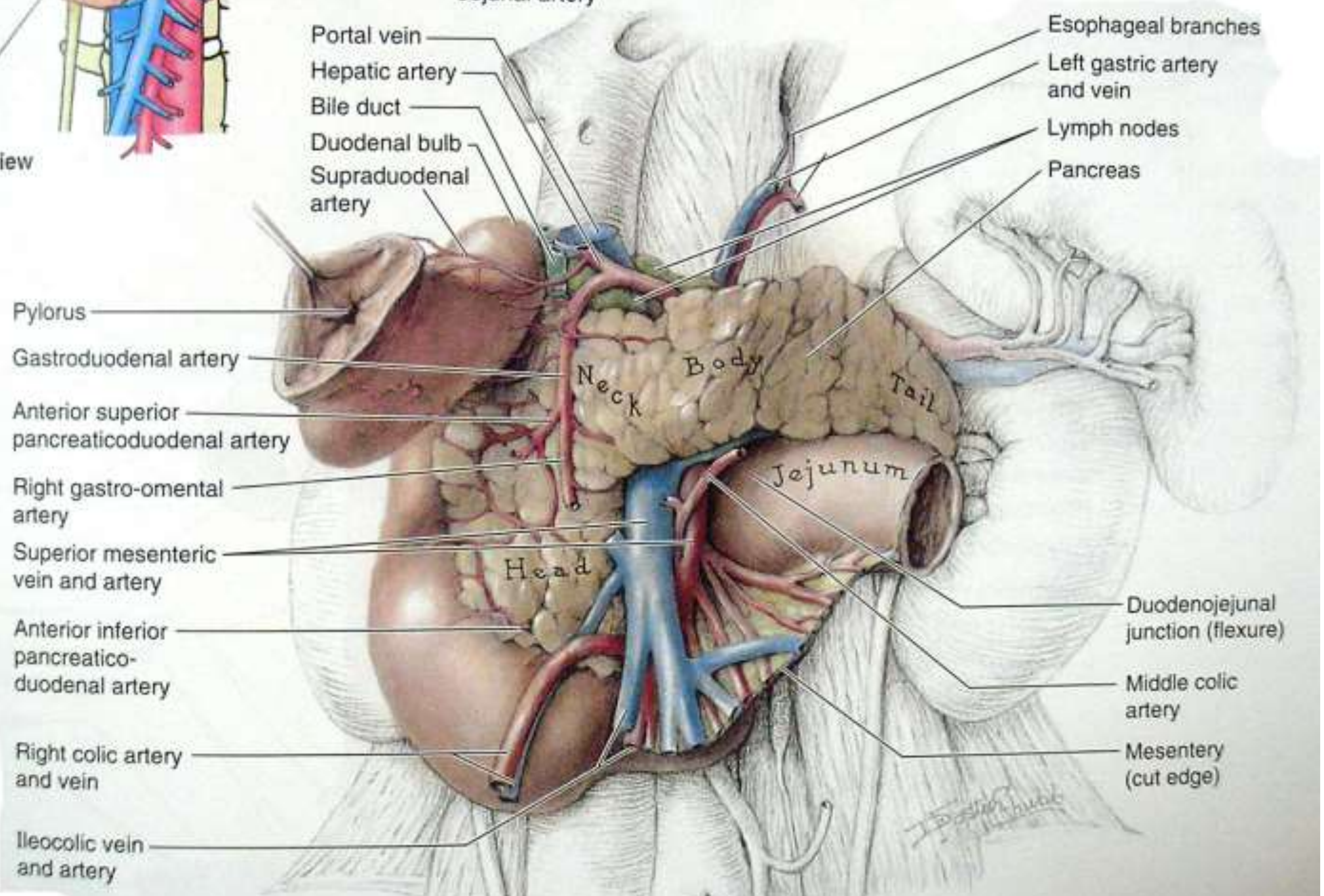


L1
L4
L5
Uncinate process
Liver (retracted)
Stomach
Portal vein
Gallbladder (cut open)
Hepatoduodenal ligament (lesser omentum)
A
B
C
D
E
Greater omentum (cut)
Bile duct (common bile duct)
Suspensory muscle
Duodeno-jejunal flexure (junction)
Jejunum
Root of transverse mesocolon
Head of pancreas
Minor duodenal papilla
Major duodenal papilla
Hepatopancreatic ampulla
Pancreatic duct
Peritoneum
Superior mesenteric vein
Superior mesenteric artery
Inferior vena cava
Aorta

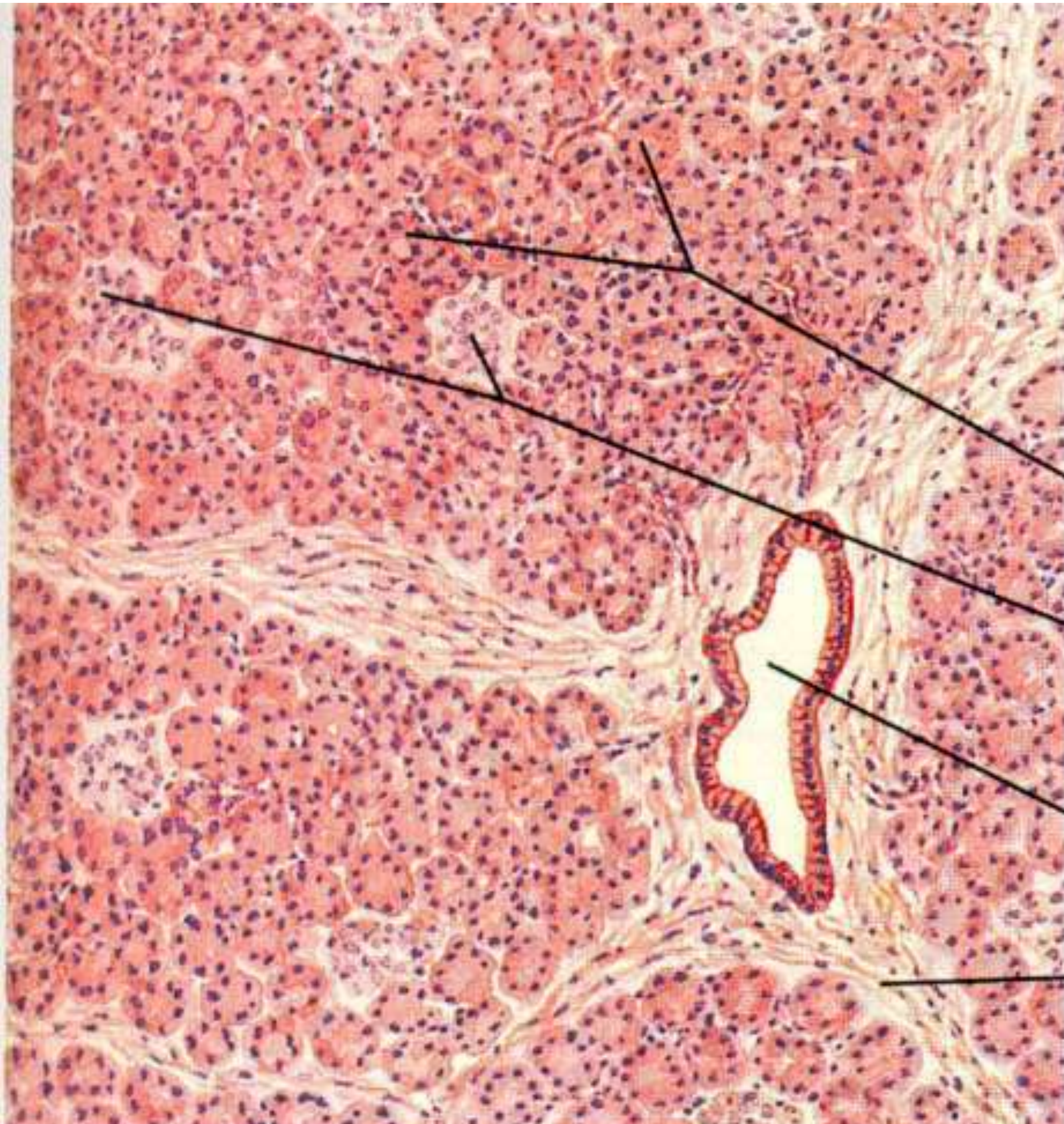


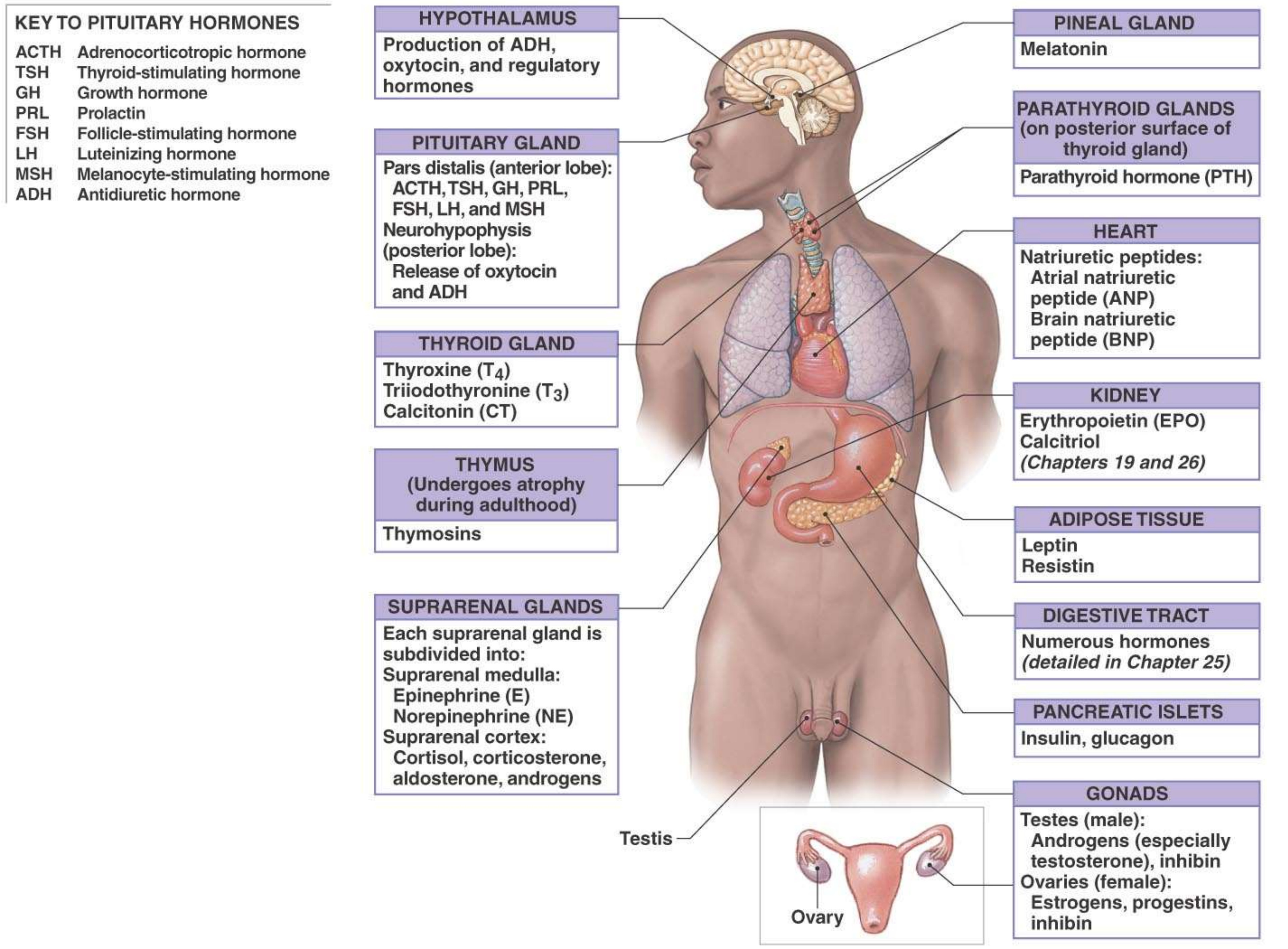
(A) Anterior view

Anterior views



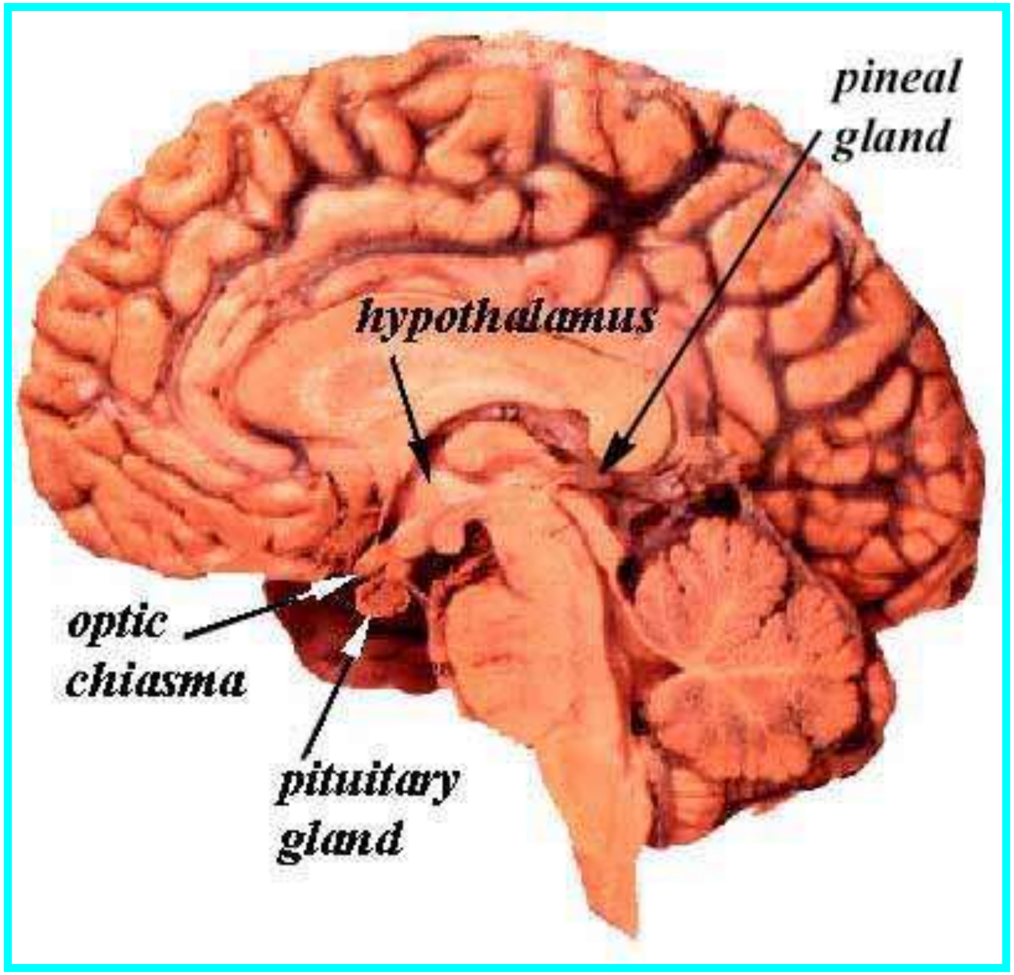
Мікроскопічна будова підшлункової залози




KEY TO PITUITARY HORMONES
ACTH Adrenocorticotropic hormone
TSH Thyroid-stimulating hormone
GH Growth hormone
PRL Prolactin
FSH Follicle-stimulating hormone
LH Luteinizing hormone
MSH Melanocyte-stimulating hormone
ADH Antidiuretic hormone
HYPOTHALAMUS
Production of ADH, oxytocin, and regulatory hormones
PITUITARY GLAND
Pars distalis (anterior lobe): ACTH, TSH, GH, PRL, FSH, LH, and MSH
Neurohypophysis (posterior lobe): Release of oxytocin and ADH
THYROID GLAND
Thyroxine (T_4)
Triiodothyronine (T_3)
Calcitonin (CT)
THYMUS
(Undergoes atrophy during adulthood)
Thymosins
SUPRARENAL GLANDS
Each suprarenal gland is subdivided into:
Suprarenal medulla:
Epinephrine (E)
Norepinephrine (NE)
Suprarenal cortex:
Cortisol, corticosterone, aldosterone, androgens
PINEAL GLAND
Melatonin
PARATHYROID GLANDS (on posterior surface of thyroid gland)
Parathyroid hormone (PTH)
HEART
Natriuretic peptides:
Atrial natriuretic peptide (ANP)
Brain natriuretic peptide (BNP)
KIDNEY
Erythropoietin (EPO)
Calcitriol
(Chapters 19 and 26)
ADIPOSE TISSUE
Leptin
Resistin
DIGESTIVE TRACT
Numerous hormones
(detailed in Chapter 25)
PANCREATIC ISLETS
Insulin, glucagon
GONADS
Testes (male):
Androgens (especially testosterone), inhibin
Ovaries (female):
Estrogens, progestins, inhibin
Testis
Ovary

HYPOTHALAMUS

- ‘Master Gland’
- Function : Control centre
- Attached to roof of third ventricle, near thalamus
- Continuously receive information on status of body systems via nerve impulses
- Monitors composition & temperature of blood
- Messages interpreted, evaluated : outgoing messages dispatched via nerves / hormones
- Plays role in feedback systems that govern secretions of endocrine system



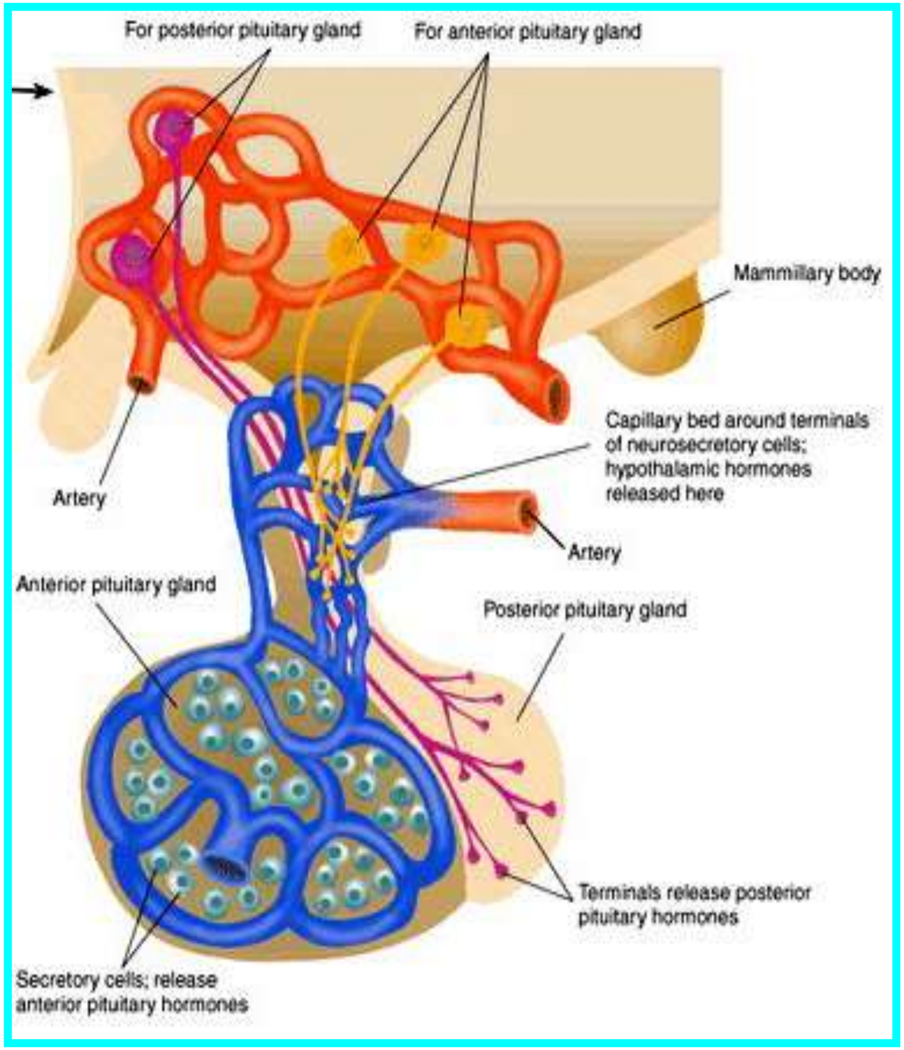
2. PITUITARY GLAND

- The mass of glandular tissue with the size of a pea
- Lies in sella turcica
- Slender stalk: Infundibulum connects pituitary gland to hypothalamus
- 2 parts : Neurohypophysis

  Adenohypophysis

Indirectly controls :

- Growth
- Metabolism
- Sexual reproduction
- Lactation

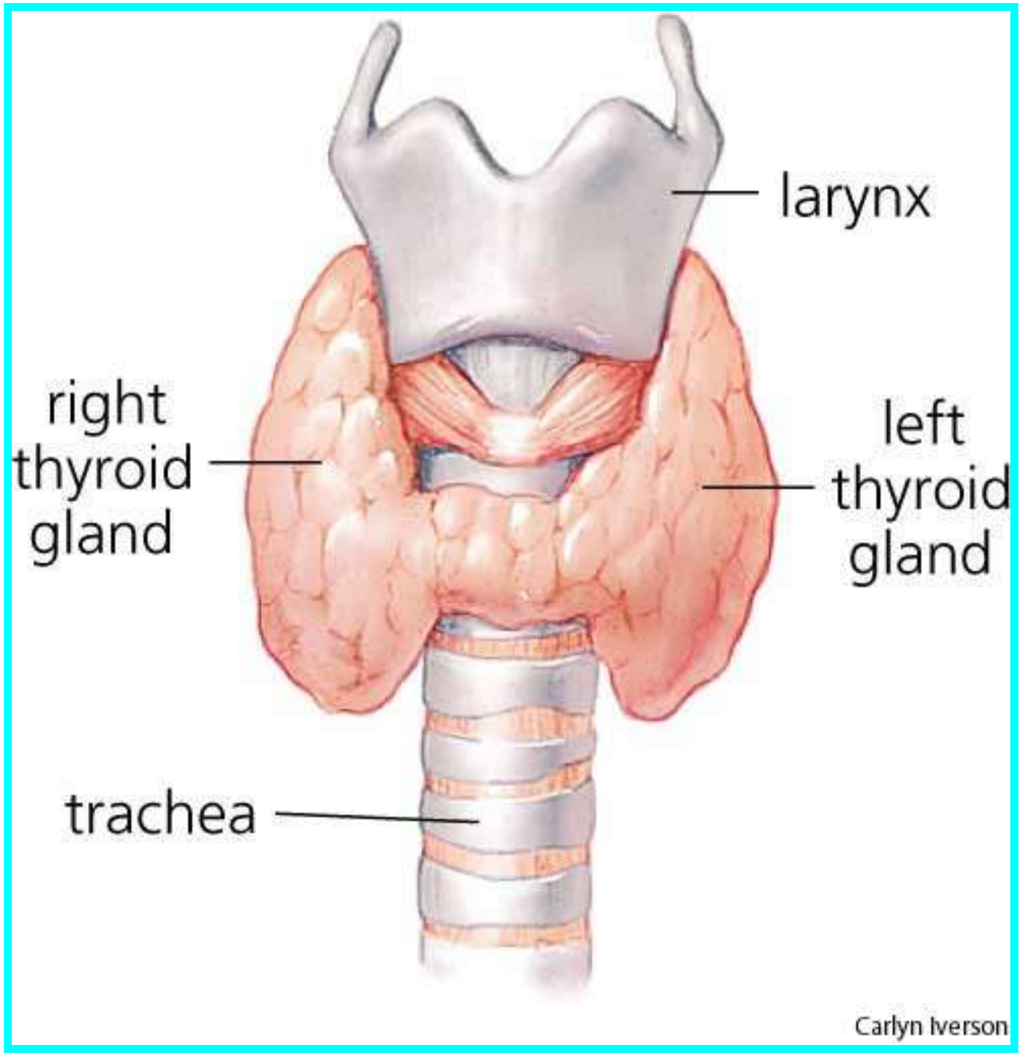


2. PITUITARY GLAND: PARTS

Neurohypophysis	**Adenohypophysis**
Small posterior lobe	Large anterior lobe
Stores hormones	Releases hormones
Oxytocin	Growth hormone (GH)
Anti Diuretic hormone (ADH)	Thyroid Stimulating hormone (TSH)
	Adenocorticotropic hormone (ACTH)
	Lutenizing hormone (LH)
	Follicle stimulating hormone (FSH)
	Melanocyte stimulating hormone (MSH)
	Prolactin (PRL)

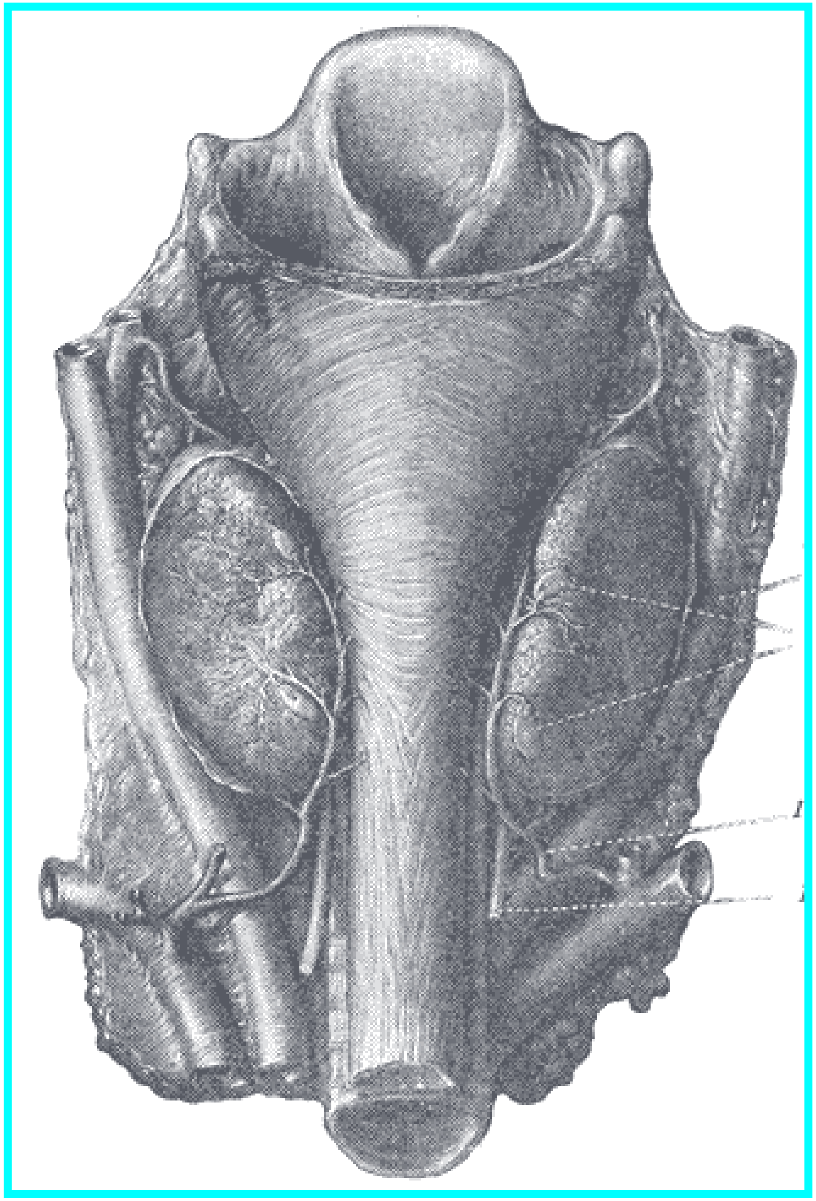
3. THYROID GLAND

- **Located in middle anterior part of the neck: below larynx, in front of trachea**
- **"Butterfly" shape**
- **2 lobes connected by isthmus**
- **↑ in size : puberty & pregnancy**
- **Rich blood supply: able to deliver high levels of hormones in short period of time**
- **Produces Thyroxin (T4) & Tri-iodothyronine (T3)**
- **Calcitonin : involved in calcium & phosphate homeostasis**



4. PARATHYROID GLAND

- Small rounded mass
- Attached to the posterior surface of thyroid gland
- Produces Parathyroid hormone (PTH)
- Controls homeostasis of calcium & phosphate in blood by activating Vitamin D

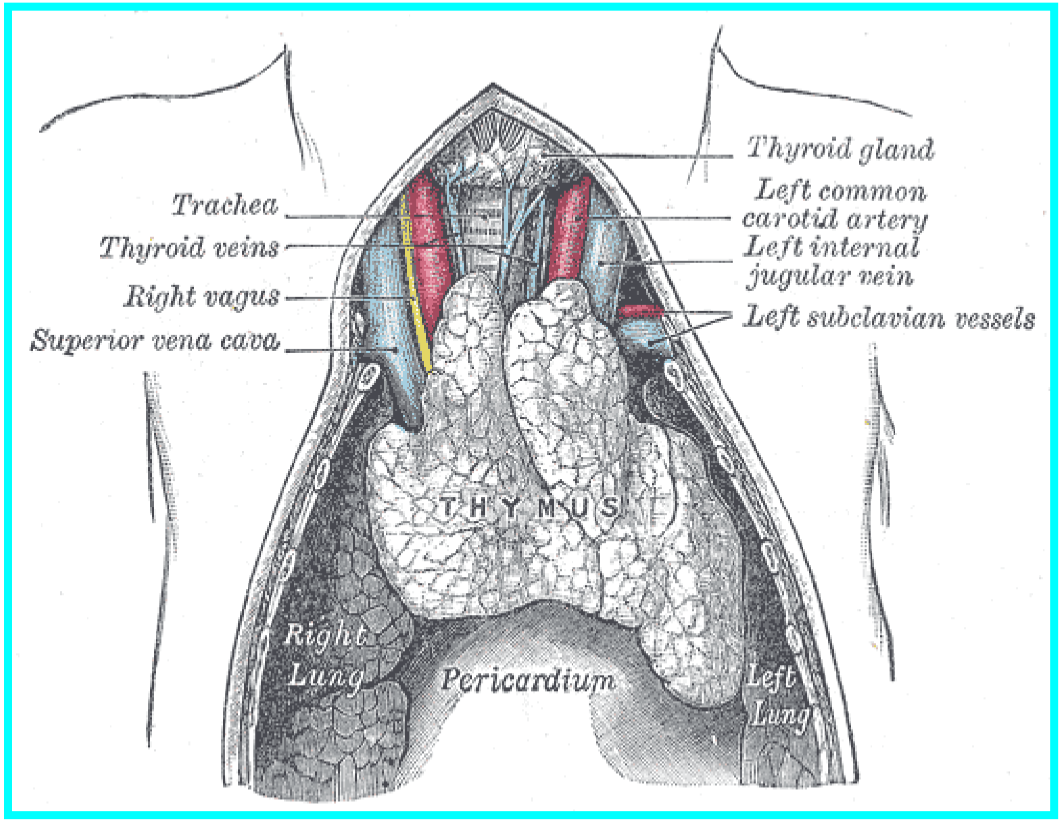


5. THYMUS

- Take a part in the immune system protection

- Produces thymosin, thymic humaral factor & thymic factor

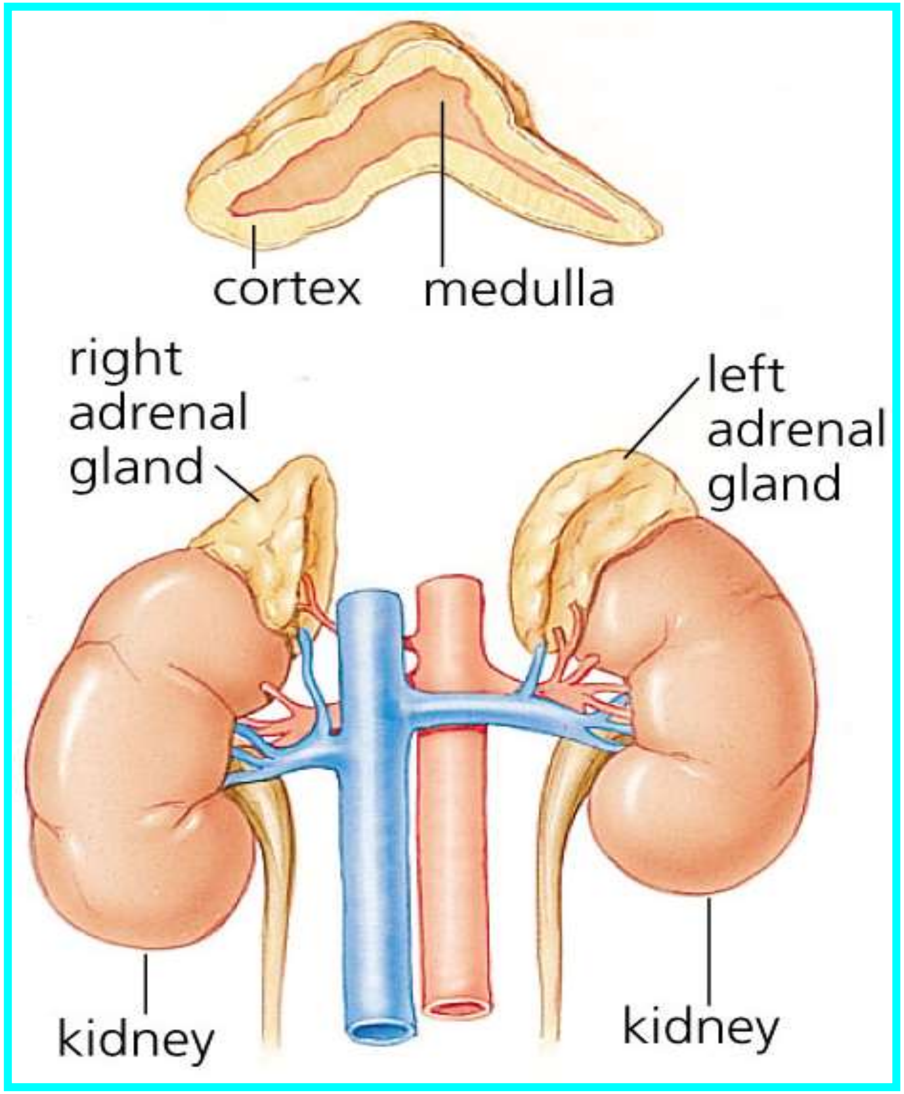
- Responsible for maturation of T-lymphocytes



6. ADRENAL GLAND

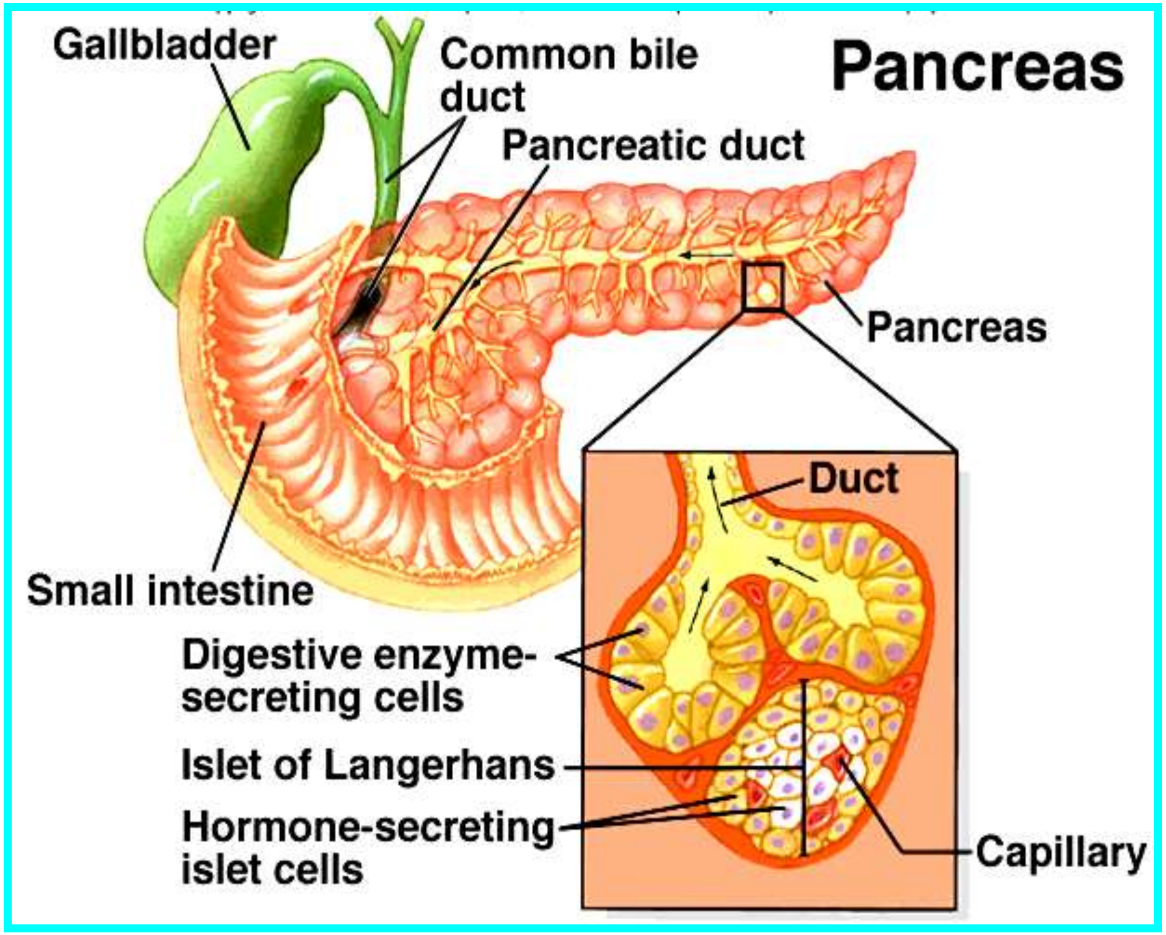
- **Located on the superior extremity of the kidney**
- **Divided into: (i) outer cortex**

  (ii) inner medulla



7. PANCREAS

- **Flattened organ**
- **Lies retroperitoneally & transversely across posterior abdominal wall**
- **Posterior to stomach, between duodenum on right & spleen on left**
- **Classified as exocrine & endocrine**



Hormones:

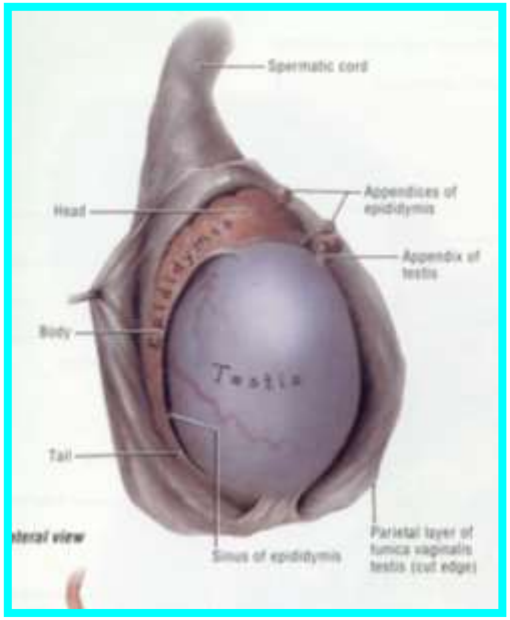
- **Islets of Langerhans secrete: Glucagon, α cells : ↑ blood glucose**

  Insulin, β cells: ↓ blood glucose
- **Growth harmone inhibiting hormone (GHIH), δ cells : inhibits glucagon & insulin**

12 & 13. TESTES & OVARIES

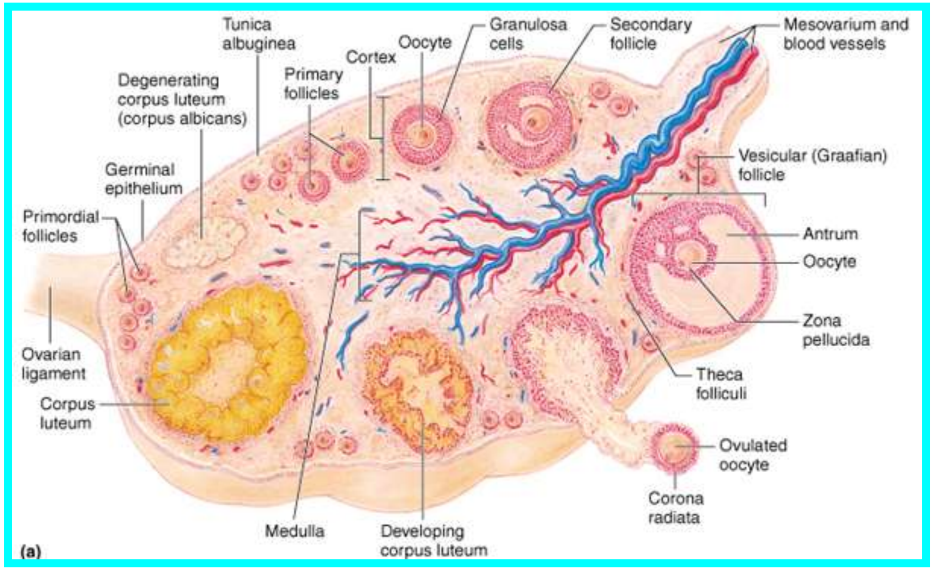
TESTES:

- **Located inside the scrotum**
- **Produce testosterone**
- **Stimulates development of male sexual characteristics**



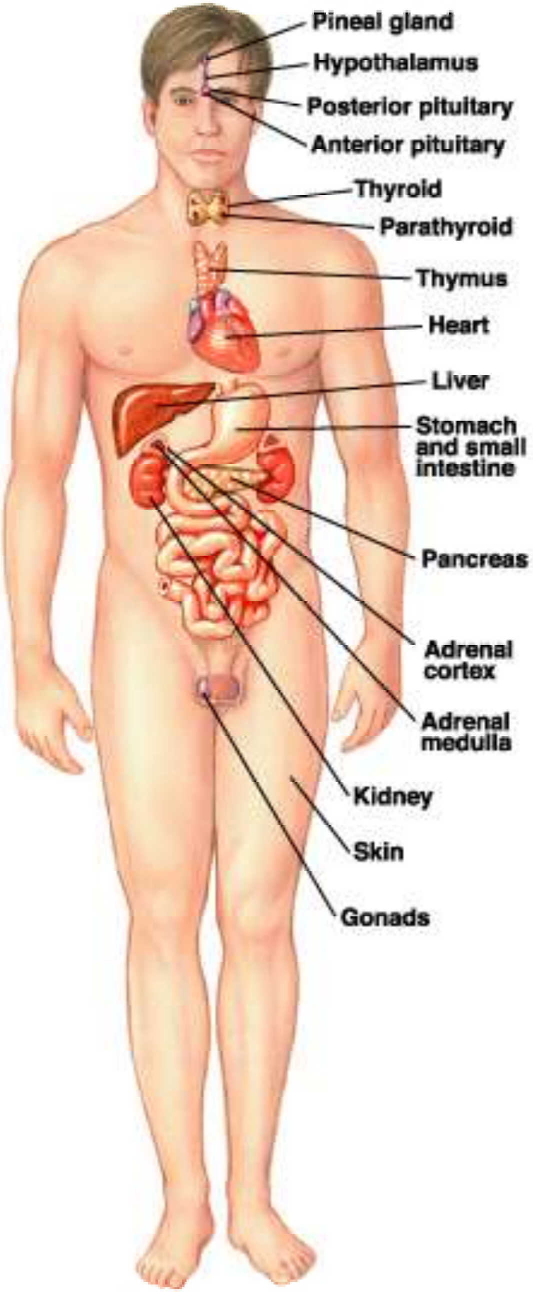
OVARIES:

- **Located in the pelvic cavity**
- **Produce estrogen & progesterone**
- **Responsible for development & maintenance of female characteristics & menstrual cycle**



Endocrine System

- The endocrine system includes all organs of the body which called endocrine glands.
- An endocrine gland secretes hormones.
- Hormones are molecules that are secreted into the blood.
- Hormones are substances that are secreted by one group of cells that affects the physiology of another group of cells (organs). The endocrine system is controlled by the pituitary gland and the hypothalamus.
- Compared to most other organs in the body, endocrine organs are well vascularized.
- VIDEO http://www.youtube.com/watch?v=HrMi4GikWwQ



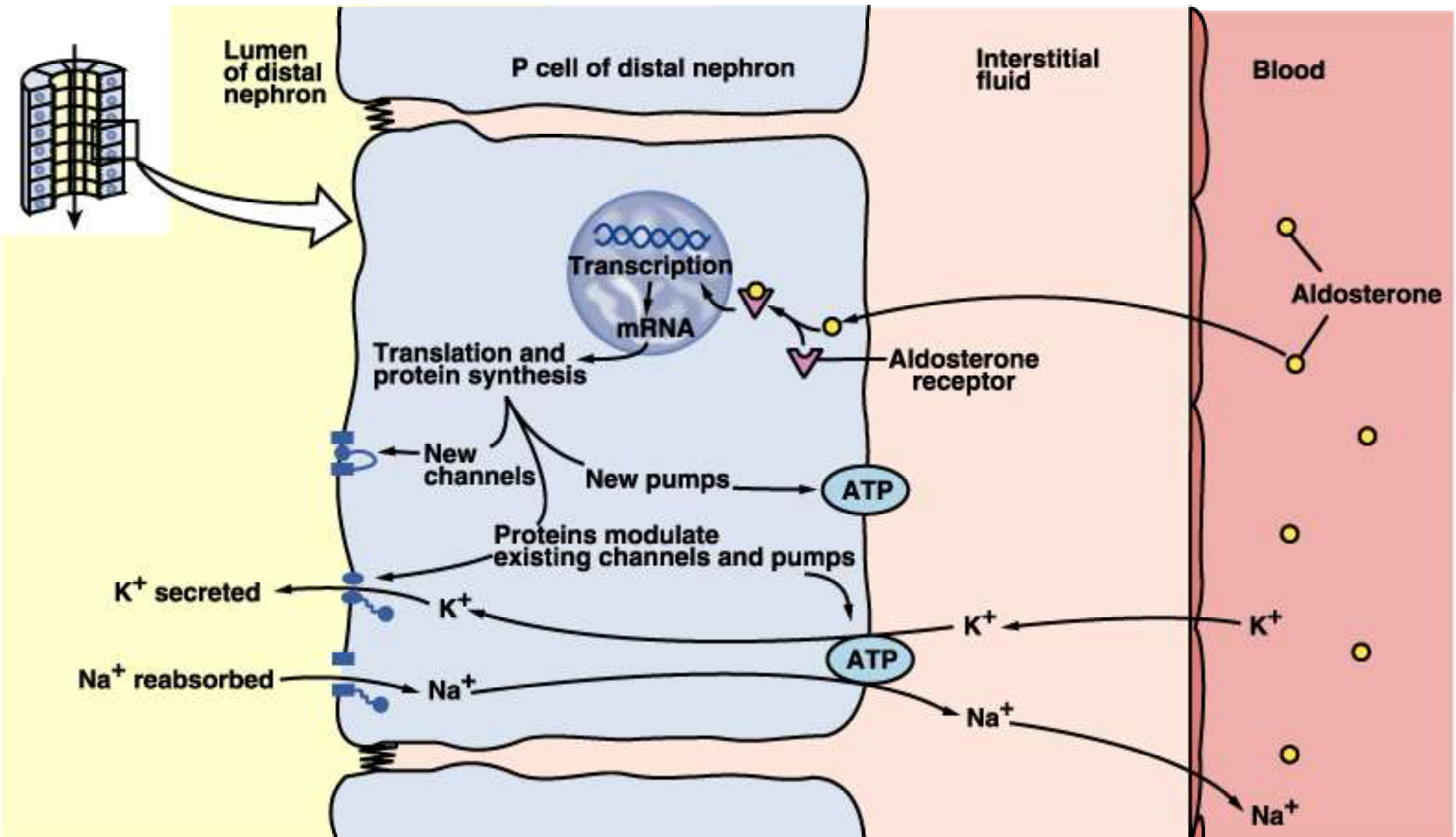
Location	Gland or Cell?	Chemical Class
Pineal gland	Gland	Amine
Hypothalamus	Clusters of neurons	Peptides
Posterior pituitary	Extensions of hypothalamic neurons	Peptides
Anterior pituitary	Gland	Peptides
Thyroid	Gland	Iodinated amines Peptide
Parathyroid	Gland	Peptide
Thymus	Gland	Peptides
Heart	Cells	Peptide
Liver	Cells	Peptides
Stomach and small intestine	Cells	Peptides
Pancreas	Gland	Peptide
Adrenal cortex	Gland	Steroids
Adrenal medulla	Gland	Amines
Kidney	Cells	Peptide Steroid
Skin	Cells	Steroid
Testes (male)	Glands	Steroids Peptide
Ovaries (female)	Glands	Steroids Peptide
Adipose tissue	Cells	Peptide
Placenta (pregnant females only)	Gland	Steroids Peptide

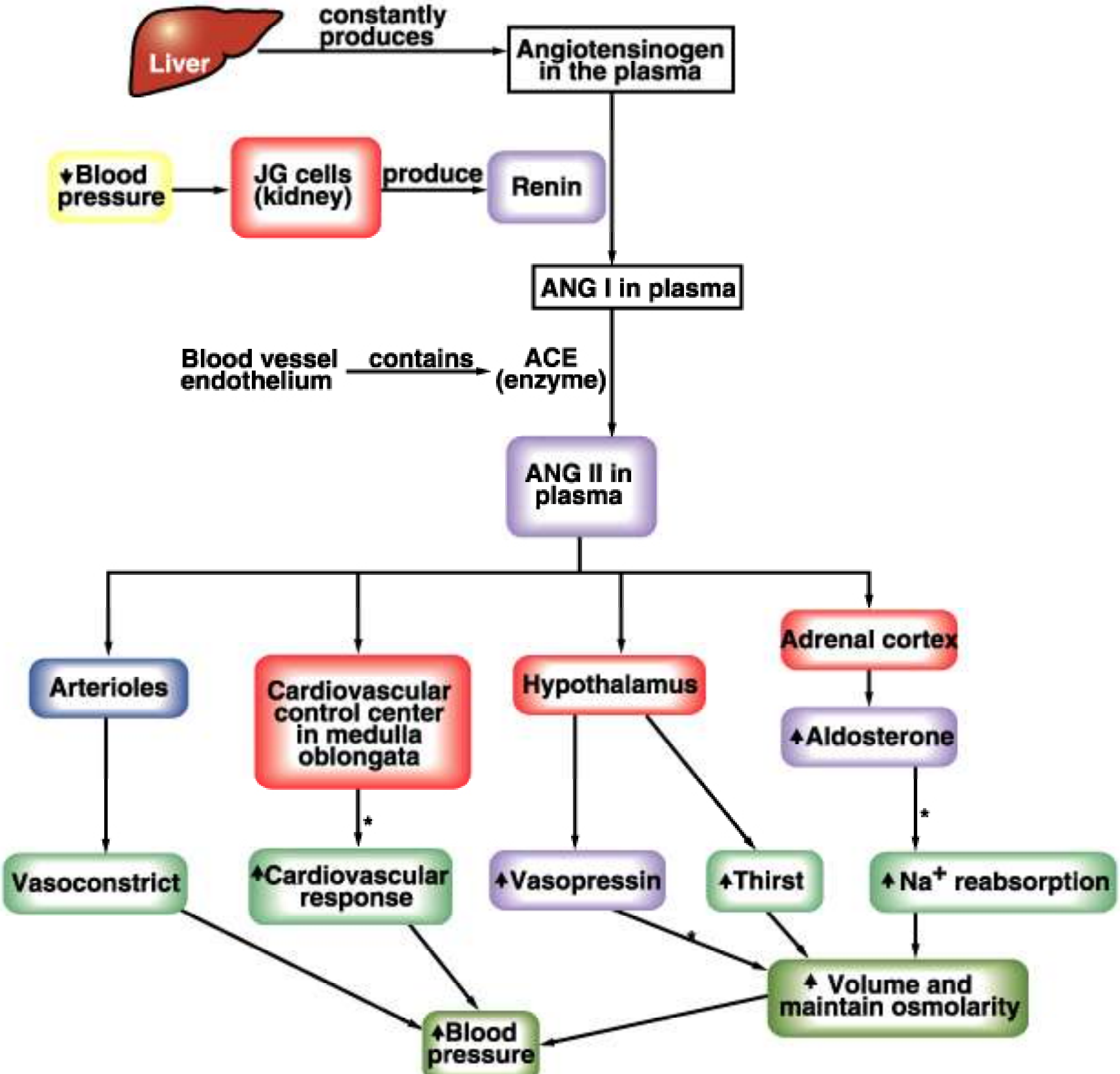
Long-term regulation of Na+

- Under the control of aldosterone; it increases Na+ reabsorption into the blood from the kidney filtrate

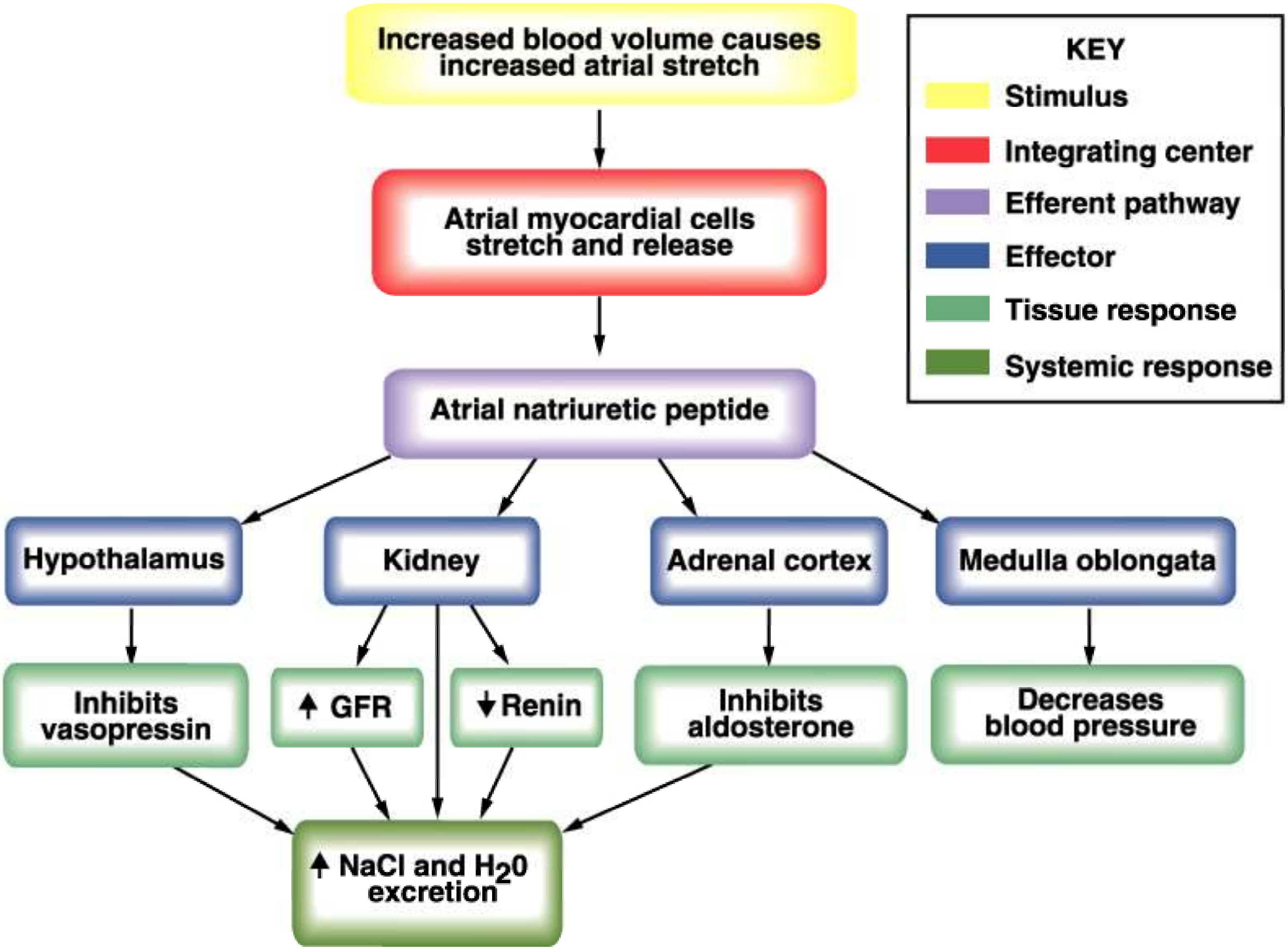
What will happen to plasma [K+]?

What will be the overall effect on plasma osmolarity?





Liver
constantly produces
Angiotensinogen in the plasma
↓Blood pressure
JG cells (kidney)
produce
Renin
ANG I in plasma
Blood vessel endothelium
contains
ACE (enzyme)
ANG II in plasma
Arterioles
Cardiovascular control center in medulla oblongata
Hypothalamus
Adrenal cortex
↑Aldosterone
*
*
*
Vasoconstrict
↑Cardiovascular response
↑Vasopressin
↑Thirst
↑Na+ reabsorption
↑ Volume and maintain osmolarity
↑Blood pressure



Increased blood volume causes increased atrial stretch
Atrial myocardial cells stretch and release
Atrial natriuretic peptide
Hypothalamus
Kidney
Adrenal cortex
Medulla oblongata
Inhibits vasopressin
↑ GFR
↓ Renin
Inhibits aldosterone
Decreases blood pressure
↑ NaCl and H20 excretion
KEY
Stimulus
Integrating center
Efferent pathway
Effector
Tissue response
Systemic response

What is a “receptor”?

- It is a protein made by the target cell (protein synthesis after gene expression)
- The protein is made, then inserted into plasma membrane, or found in cytoplasm or nucleoplasm
- The active site on the protein “fits” the hormone
- Acts to convert the signal into a response

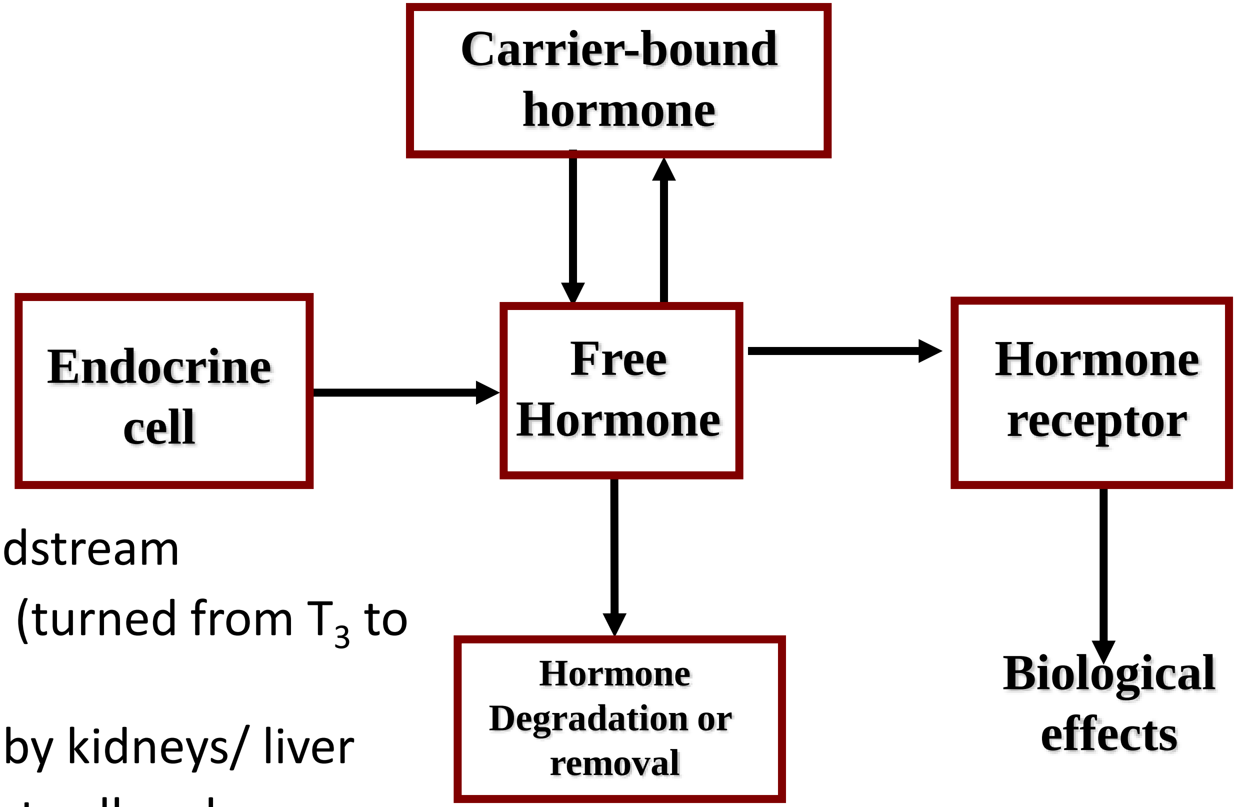
What would happen if there were a gene defect in the DNA code for a receptor?

What would happen if the receptor protein was denatured?

Hormones

- Endocrine glands secrete hormones into the plasma. Then, several different events could occur.
- It could bind to its receptor on the target cell, causing a change.
- Or, it could be destroyed by enzymes in the plasma.
- It could land in the kidneys and be filtered out before reaching its target.

What happens with a hormone once it's secreted?



- Degraded in bloodstream
- May be activated (turned from T_3 to T_4)
- May be excreted by kidneys/ liver
- May reach a target cell and cause a cell response
- May need carrier to reach target cell

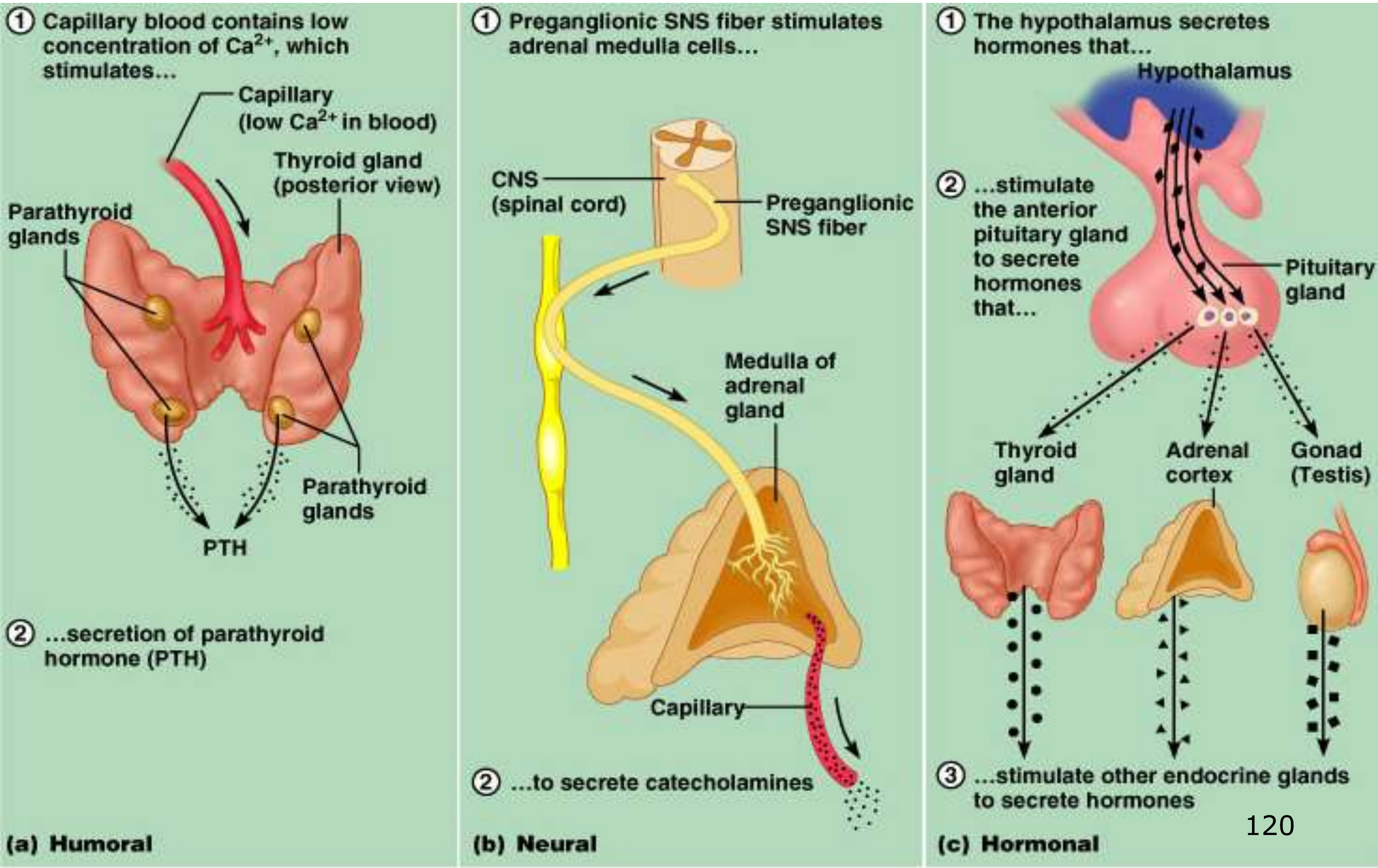
Control of Hormone Secretion

- The endocrine system is controlled by the pituitary gland and the hypothalamus.
- Always controlled by feedback loops
- Concentration declines below a minimum: more hormone is secreted
- Concentration exceeds maximum: Hormone production is halted

MECHANISMS OF HORMONE SECRETION

- **Humoral Trigger**
- Something in the blood is being monitored. When the level of that substance is too high or low, it stimulates the release of the hormone.
- **Neuronal Trigger**
- A neuron directly stimulates the gland to cause secretion of the hormone.
- **Hormonal Trigger**
- One endocrine gland releases a hormone that stimulates another endocrine gland to release its hormone.
- This is how thyroid hormone is secreted. The hypothalamus releases a hormone that causes the pituitary gland to release TSH, which causes the thyroid gland to release thyroid hormone.

Control of Hormones Release: Three Mechanisms



Humoral Trigger

- Everything in the blood is being monitored. When the level of that substance is too high or low, it stimulates the release of the hormone or stop its production.
- Examples are **insulin, glucagon, parathyroid** hormone, and **aldosterone**.
- When you eat, glucose gets high, releases insulin, which makes cells to take the sugar. Excess sugar is then converted to glycogen, which is the storage form.
- When glucose is low, glycogen is broken back down to glucose and released into the blood.
- When blood calcium is low, parathyroid gland hormone tells the intestinal cells to absorb more calcium, and kidneys to reabsorb more Ca^{++}, and stimulates osteoclasts to degrade bone matrix so calcium goes into the blood.

Neuronal Trigger

- Examples are **oxytocin, ADH** (neurohypophysis hormones) **and Epinephrine** (adrenal medulla hormone)

Hormonal Trigger

- This is when one endocrine gland releases a hormone that stimulates another endocrine gland to release its hormone.
- Examples are any of the hypothalamus or anterior pituitary hormones, and also the adrenal cortex (steroid) hormones (except aldosterone) and thyroid hormone.

Hypothalamus

- This is located at the base of the brain. It is part of the limbic system, which controls the autonomic nervous system and the endocrine systems.
- The hypothalamus controls the endocrine system by controlling the pituitary gland.
  - Secretes **releasing hormones** to cause the pituitary to release hormones
  - Secretes **inhibiting hormones** to turn off secretion of pituitary hormones

The Pituitary Gland

- This is located in the sella tursica (totally encased in bone), which gives you a clue as to how important this gland is.
- The adenohypophysis portion of the pituitary gland (anterior lobe) actually develops from an embryonic pouch that grows upward from the ectoderm of the pharynx!
- One type of diabetes (insipidus) can be caused by trauma to the pituitary gland.
- A tumor of the pituitary gland can lead to blindness because it is so close to the optic chiasma.

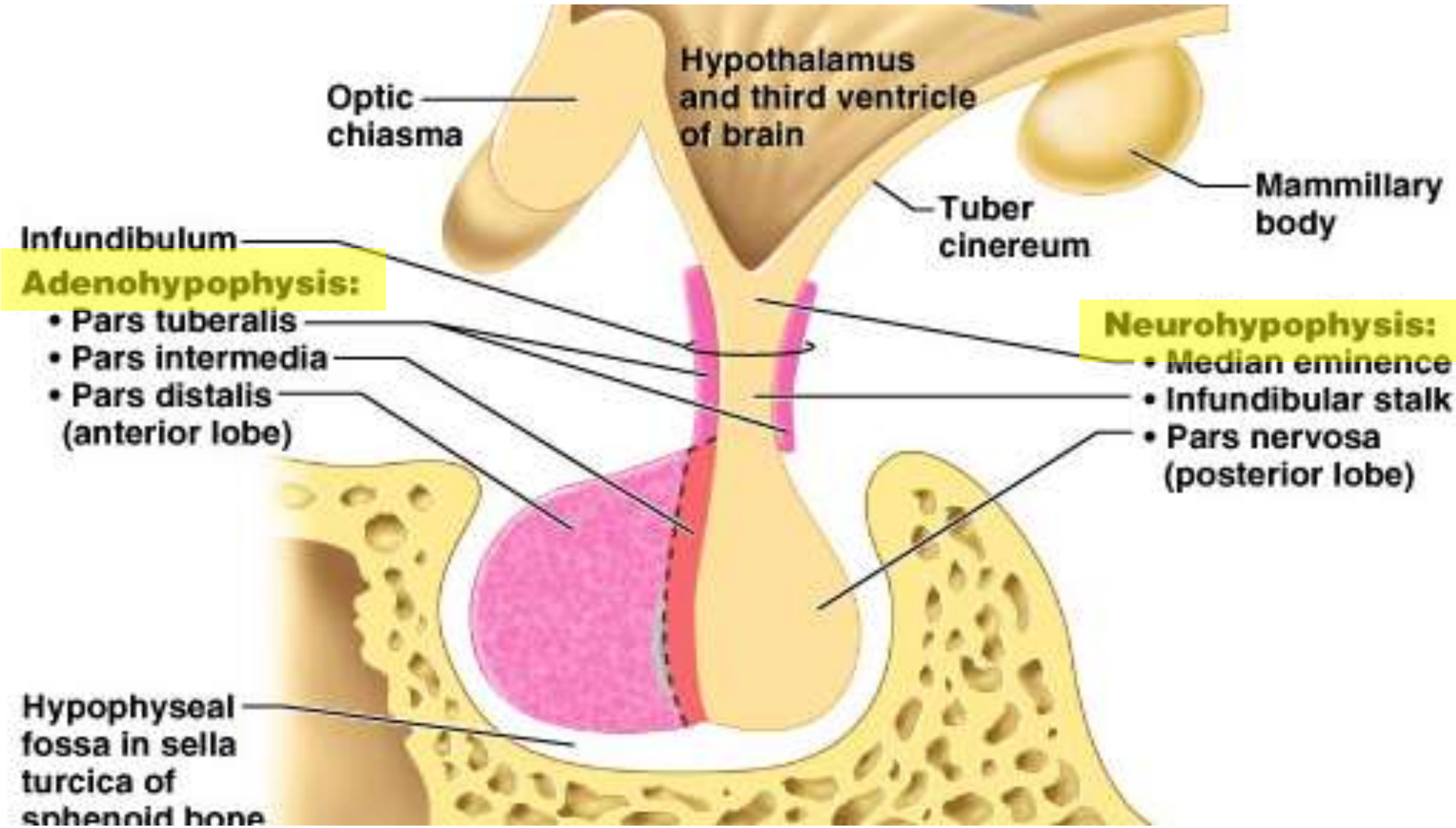
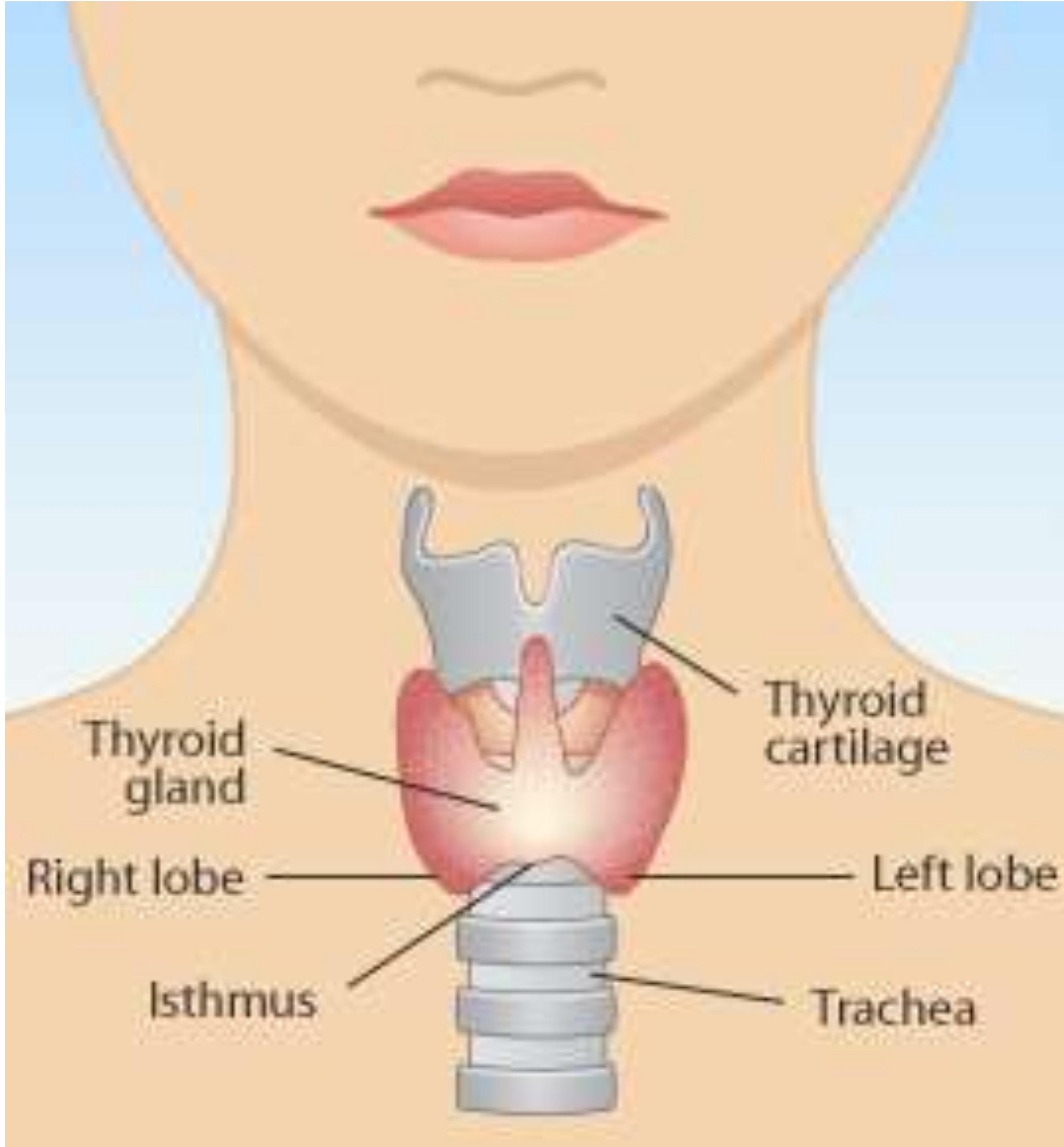


Figure 25.3a-c


Thyroid cartilage
Thyroid gland
Right lobe
Left lobe
Isthmus
Trachea

The Thyroid Gland

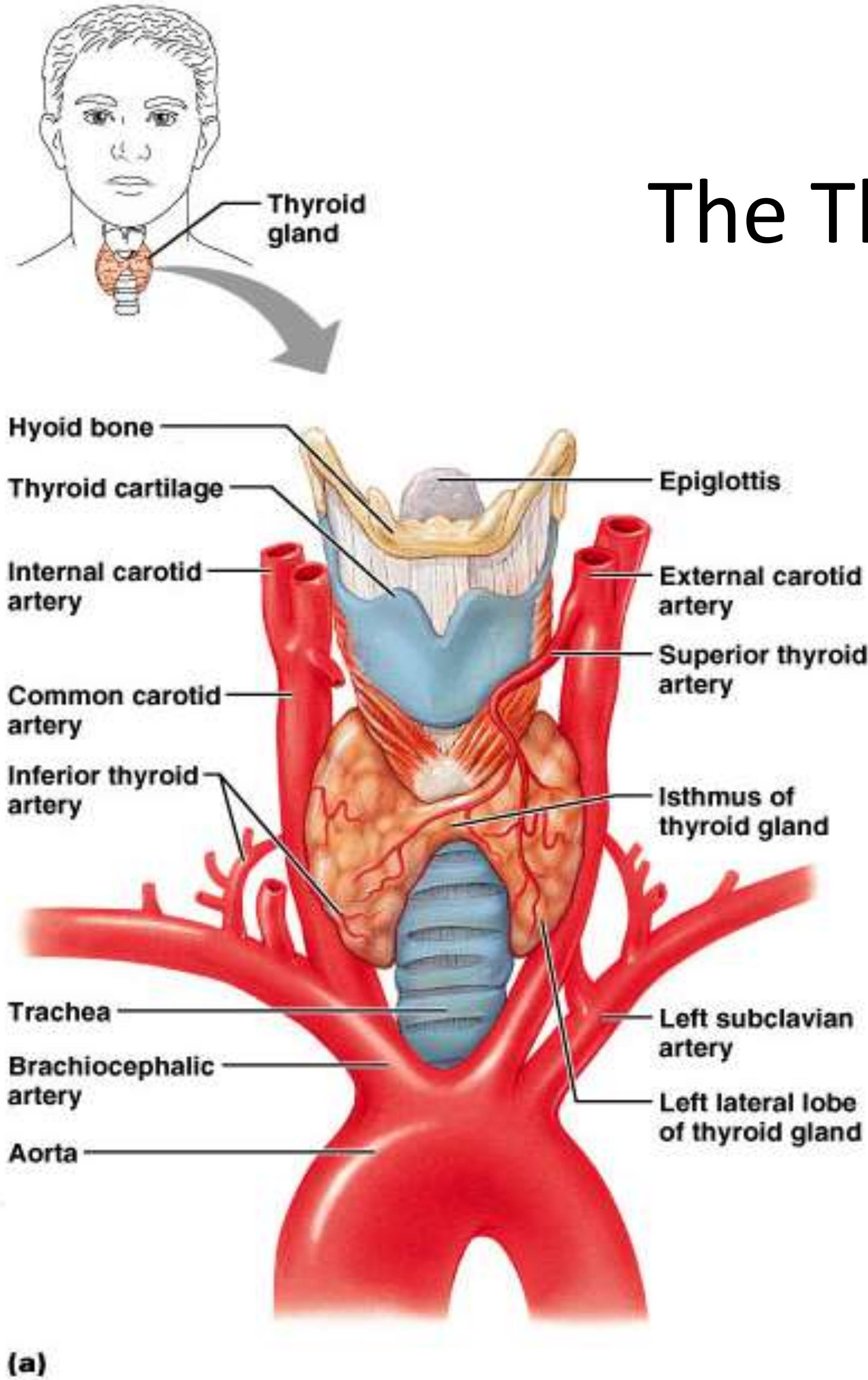
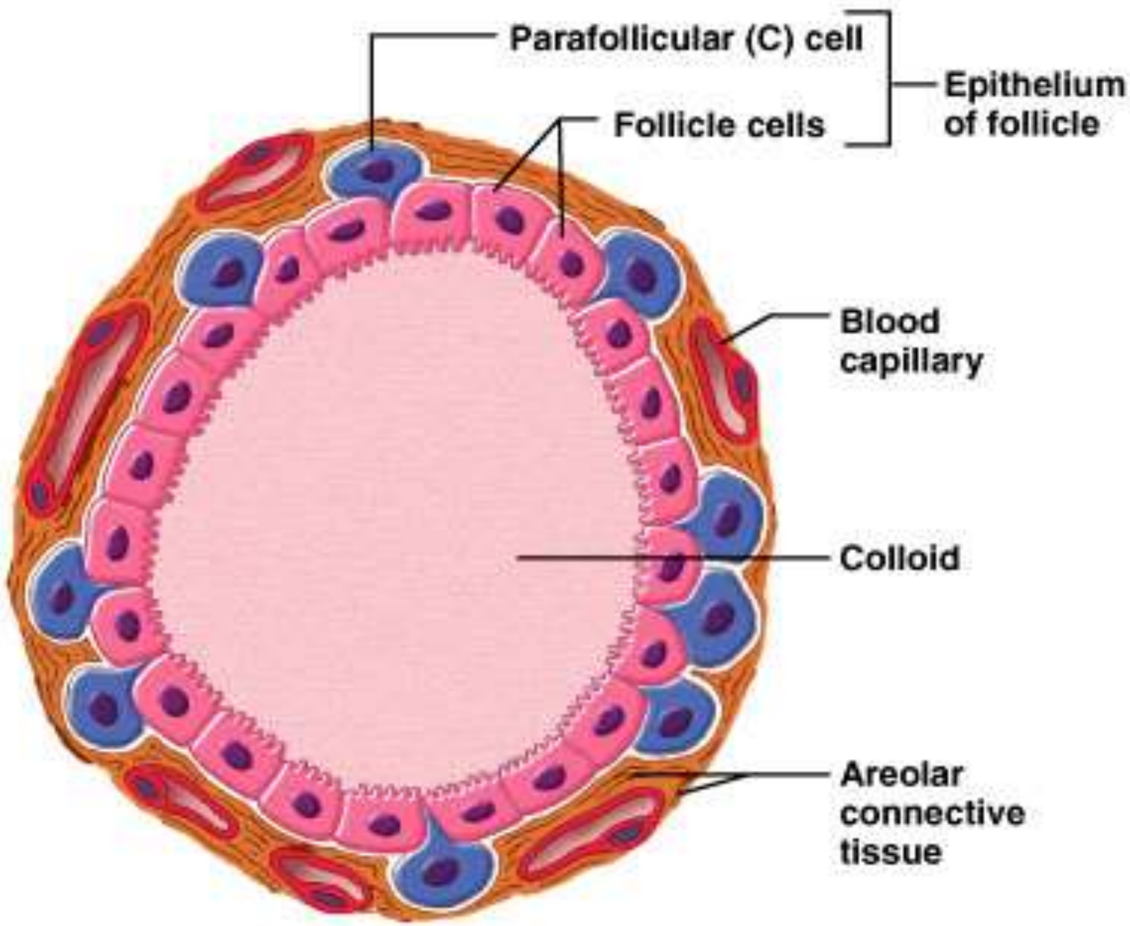


Figure 25.7a

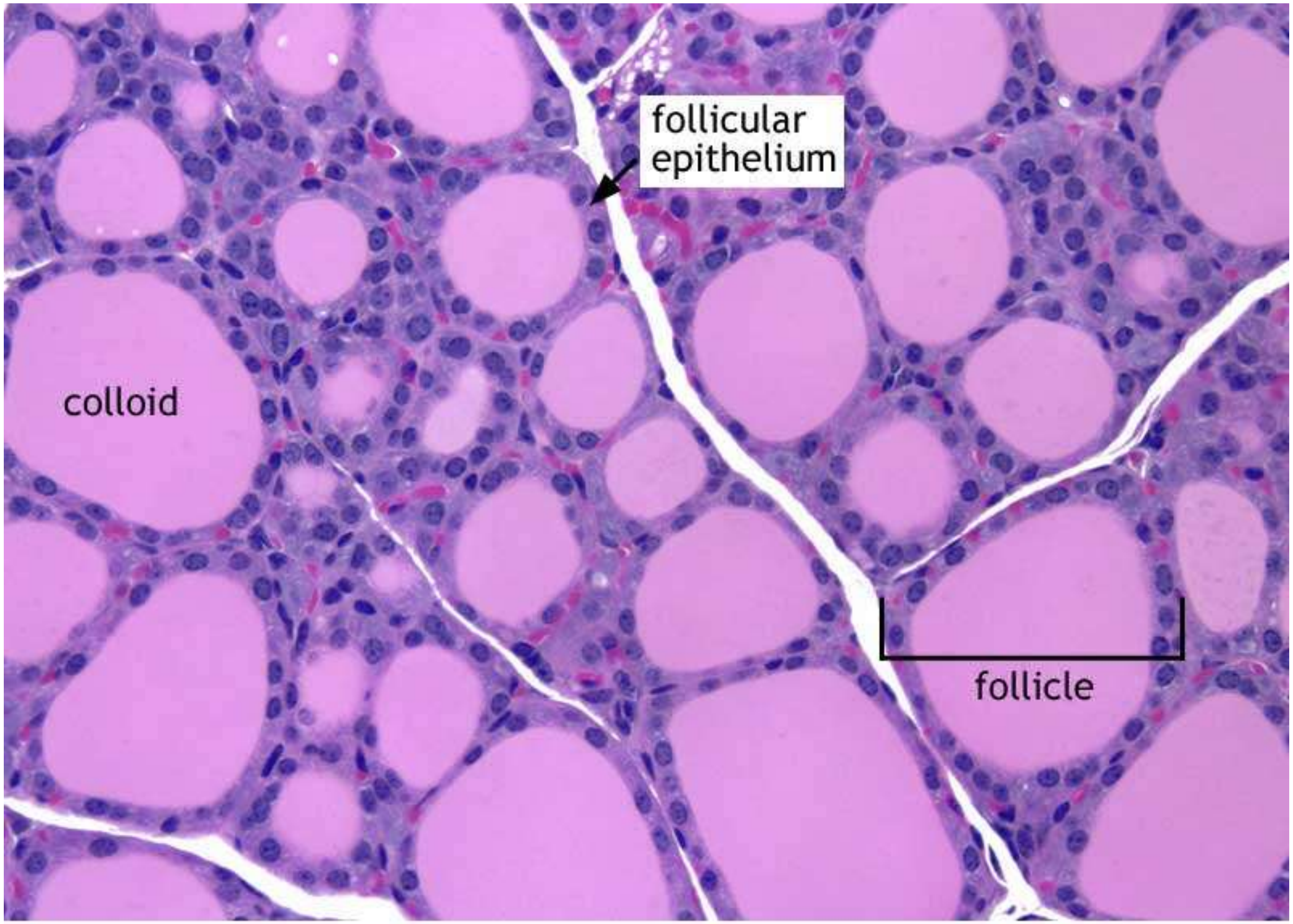
Thyroid Follicle with Thyroid Hormone



(c)

Figure 12.9c

Thyroid Gland



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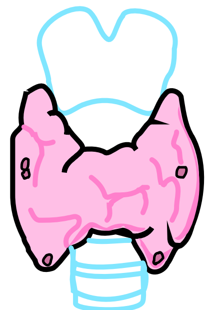
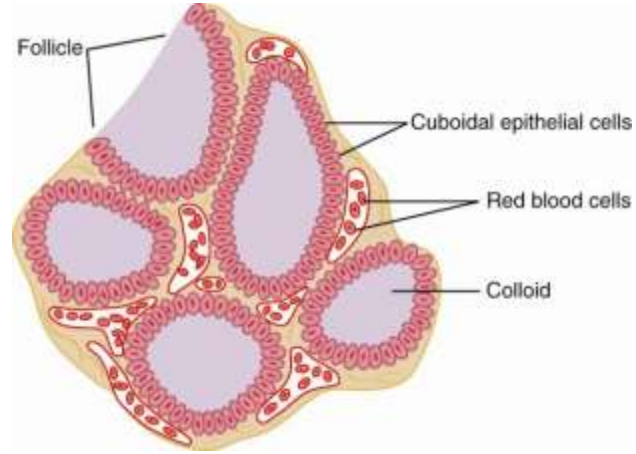
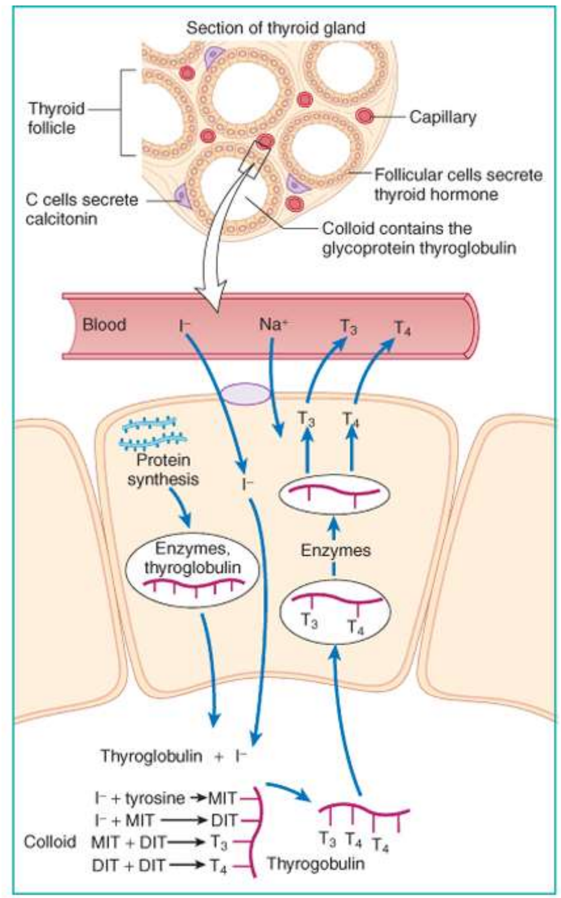
Thyroid Gland





- Thyroid has a follicles - hollow structures surrounded by follicular and parafollicular cells
- Follicular cells produce Thyroglobulin (TG)
- Building block of TH, chemically attaching I^- to tyrosine.
- In plasma, TH needs a “carrier molecule” or it will be cleared from body



Tyrosine: a bulky amino acid containing a large benzyl ring.

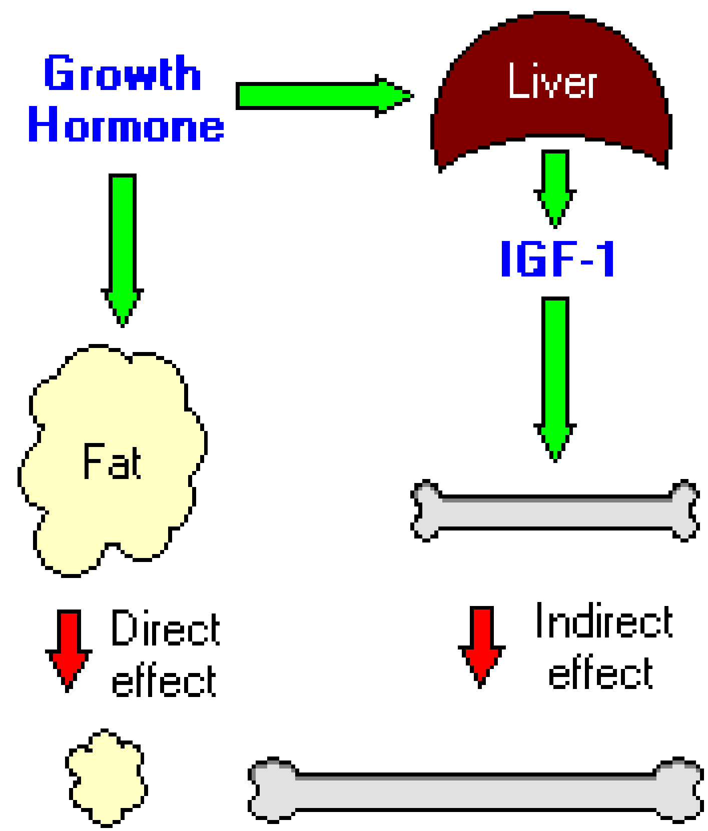
Role of Hypothalamus

- The papers then go out to every cell in the body.
- Some of the papers land on the desk of the boss. When his desk is covered with papers, he tells the manager to stop the orders for more papers.
- If not enough papers are on his desk, he tells the manager to keep sending out the order for more papers.

BONE

AND GENERAL GROWTH

Growth hormones have important effects on protein, lipid and carbohydrate metabolism. In some cases, a direct effect of growth hormone has been clearly demonstrated, in others, IGF-I is thought to be the critical mediator, and some cases it appears that both direct and indirect effects are at play.



SEX GLANDS

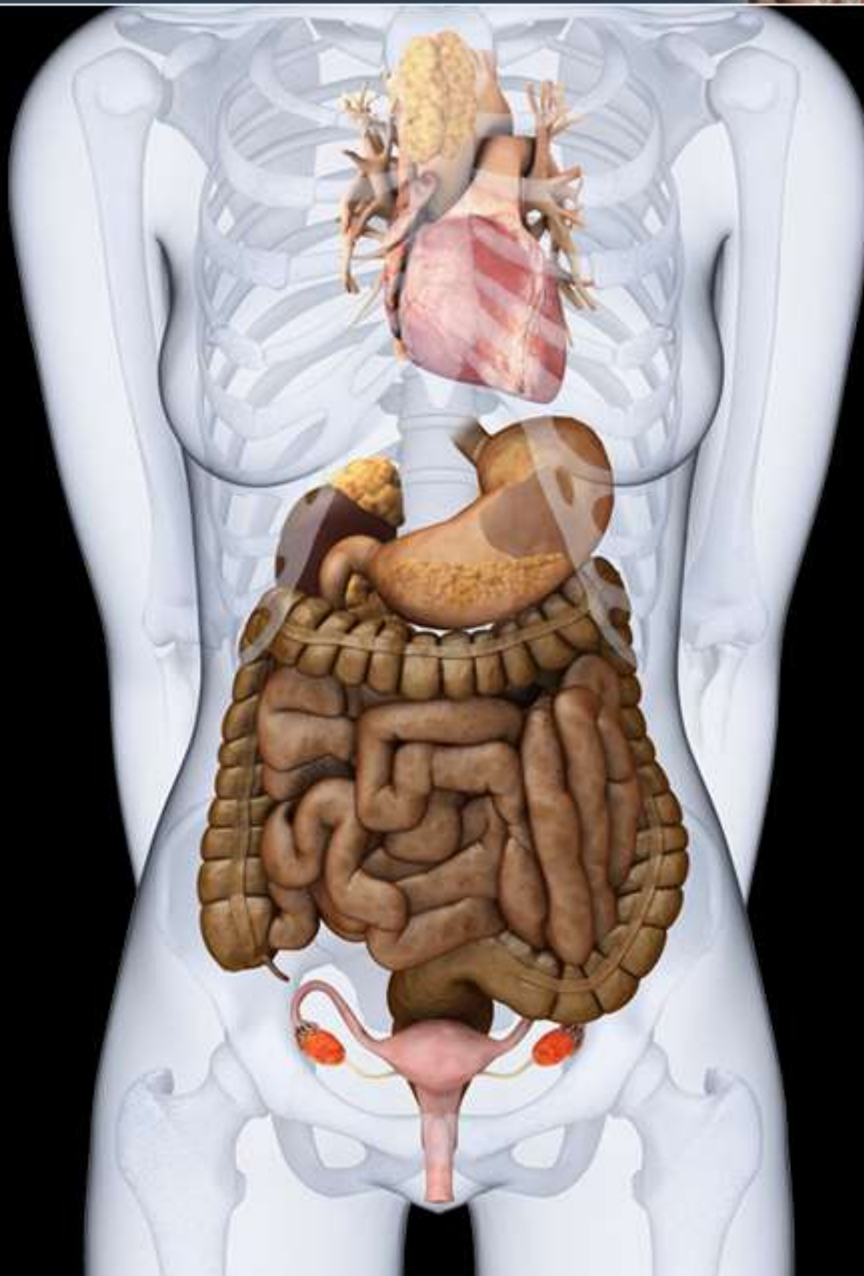
Luteinizing hormone (LH) and follicle-stimulating hormone (FSH) trigger the sex glands in both males and females to make their own hormones>and also to produce ripe egg cells in females and mature sperm cells in males

HUMAN
BODY
DVD-ROM

- CELLS AND DNA
- SKELETAL SYSTEM
- MUSCULAR SYSTEM
- NERVOUS SYSTEM
- ENDOCRINE SYSTEM
- CARDIOVASCULAR SYSTEM
- RESPIRATORY SYSTEM
- SKIN, HAIR, AND NAILS
- LYMPH AND IMMUNITY
- DIGESTIVE SYSTEM
- URINARY SYSTEM
- REPRODUCTIVE SYSTEM
- DEVELOPMENT
- ANIMATIONS

SYSTEM

HOME QUIT

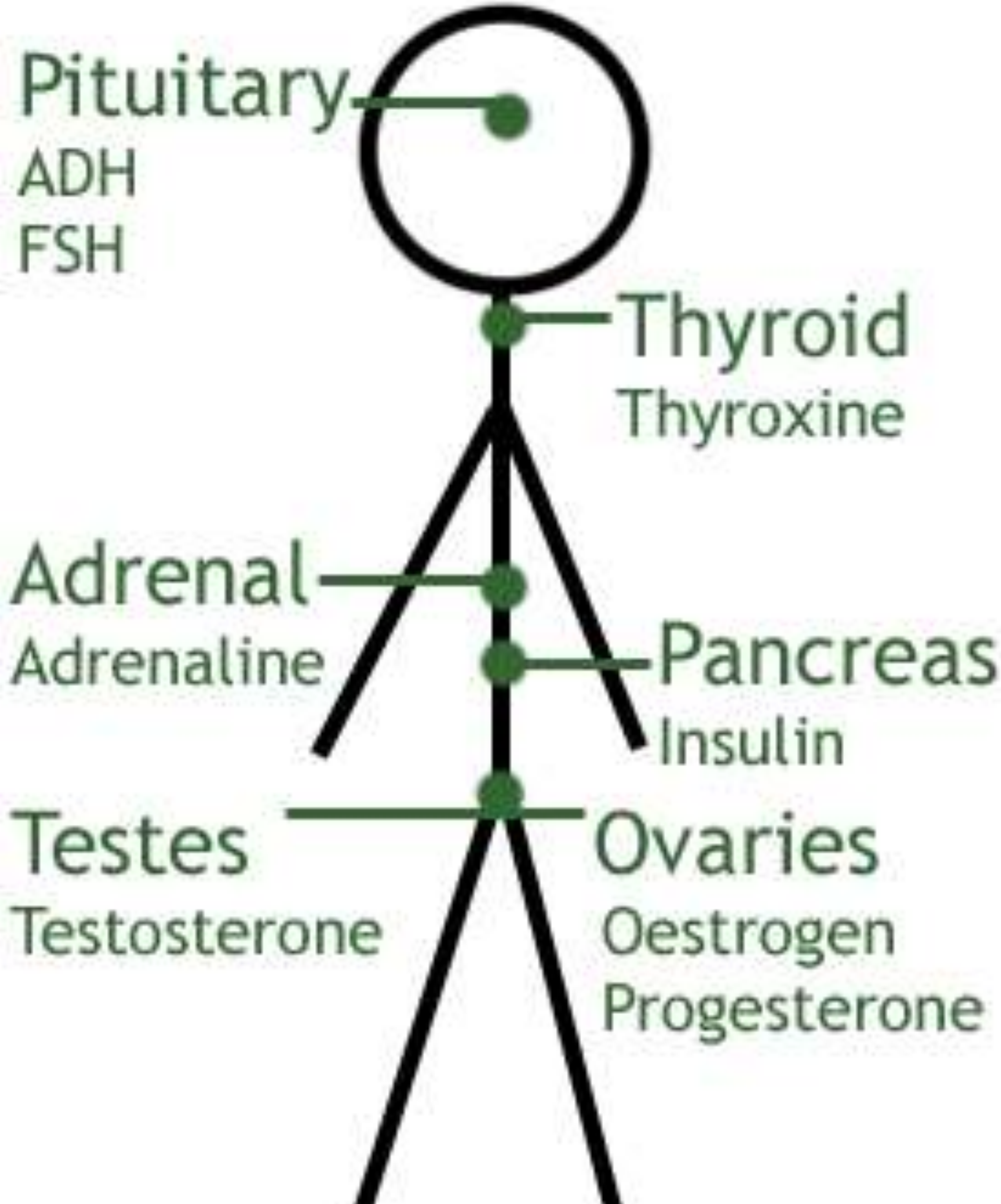


ENDOCRINE SYSTEM

The body's chemical messengers (hormones) are made by endocrine glands. These glands have no ducts but secrete their hormones directly into the blood, by which means they reach every cell in the body. Hormones affect certain target tissues or organs and regulate their activities. For more detail, see *The Human Body Book*, pp.122–23.

Ovaries

The two ovaries manufacture the female sex hormones oestrogen and progesterone, which stimulate egg ripening and thickening of the uterine wall respectively

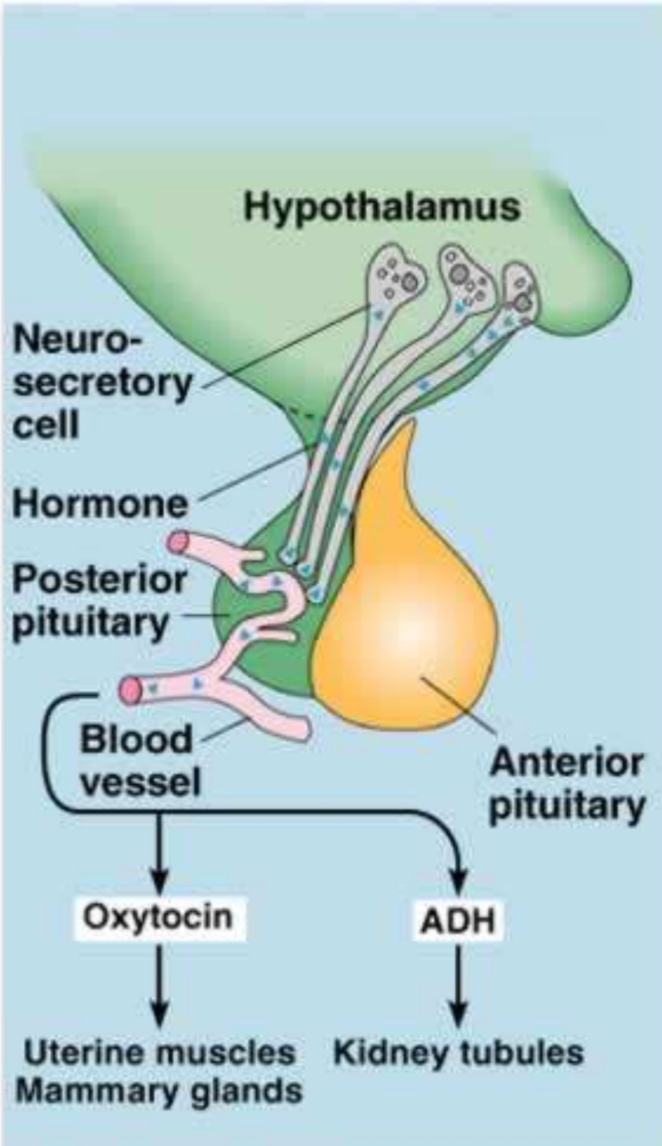

Pituitary
ADH
FSH
Thyroid
Thyroxine
Adrenal
Adrenaline
Pancreas
Insulin
Testes
Testosterone
Ovaries
Oestrogen
Progesterone

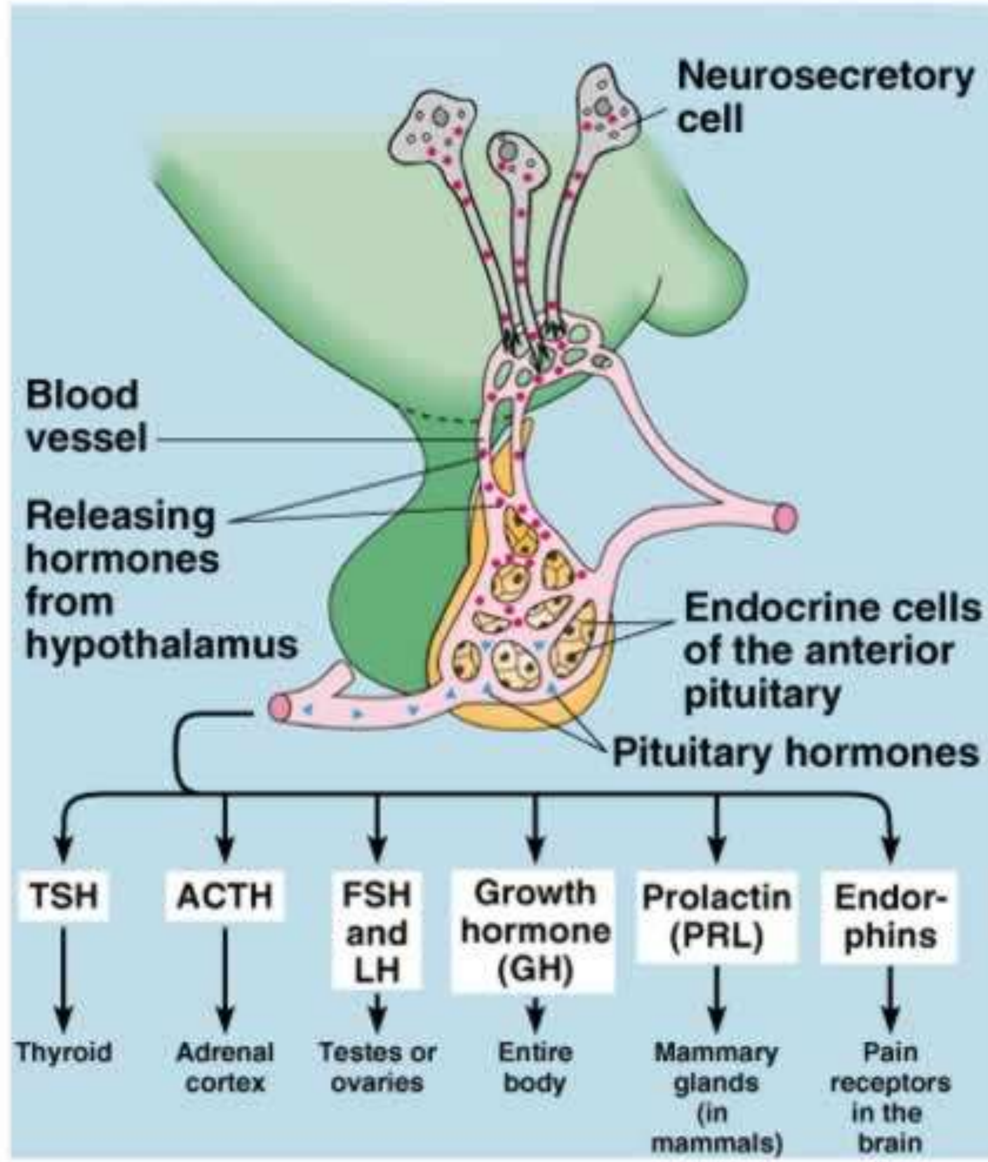
UTERINE MUSCLES AND MAMMARY GLANDS

Oxytocin is secreted by paraventricular nucleus and a small quantity is secreted by supraoptic nucleus in hypothalamus.

Oxytocin is secreted in both males and females.

- In female acts on mammary glands and uterus.
- In males facilitates release of sperm in to urethra by causing contraction of vas deferens.

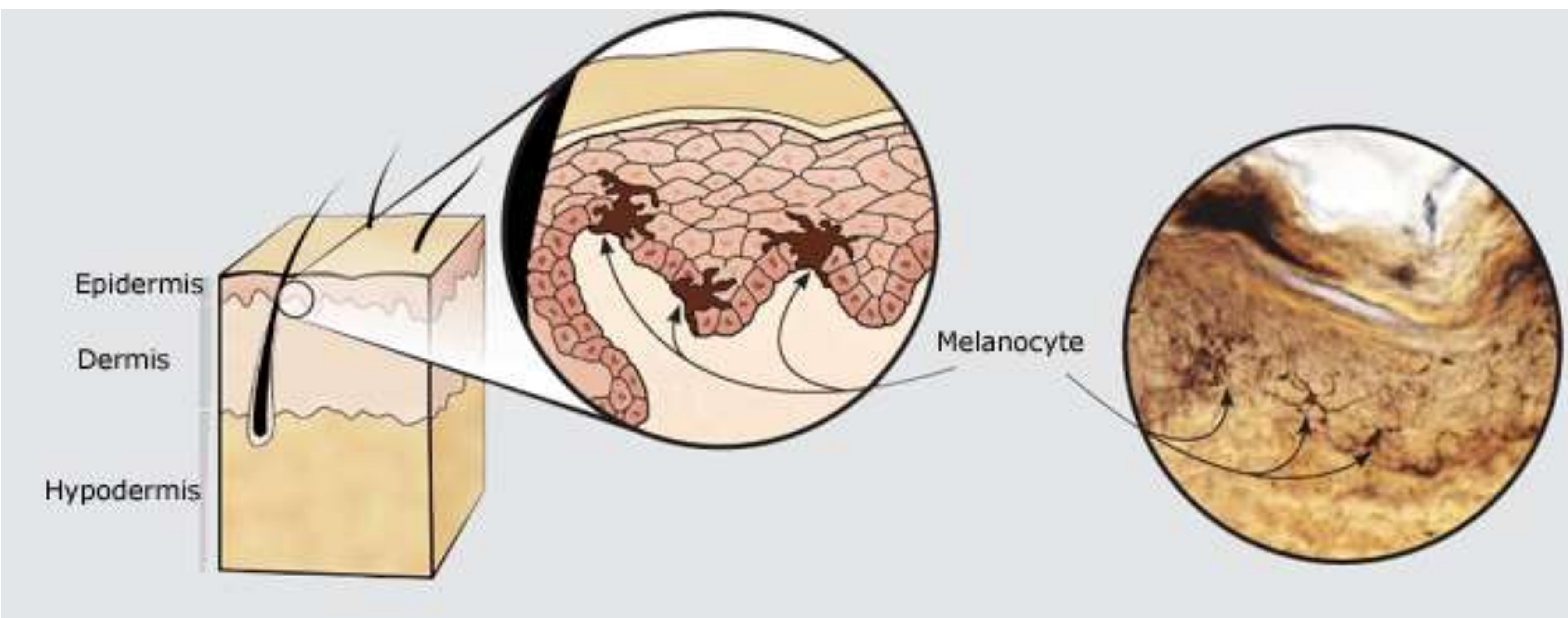

Hypothalamus
Neuro-
secretory
cell
Hormone
Posterior
pituitary
Blood
vessel
Anterior
pituitary
Oxytocin
ADH
Uterine muscles
Mammary glands
Kidney tubules

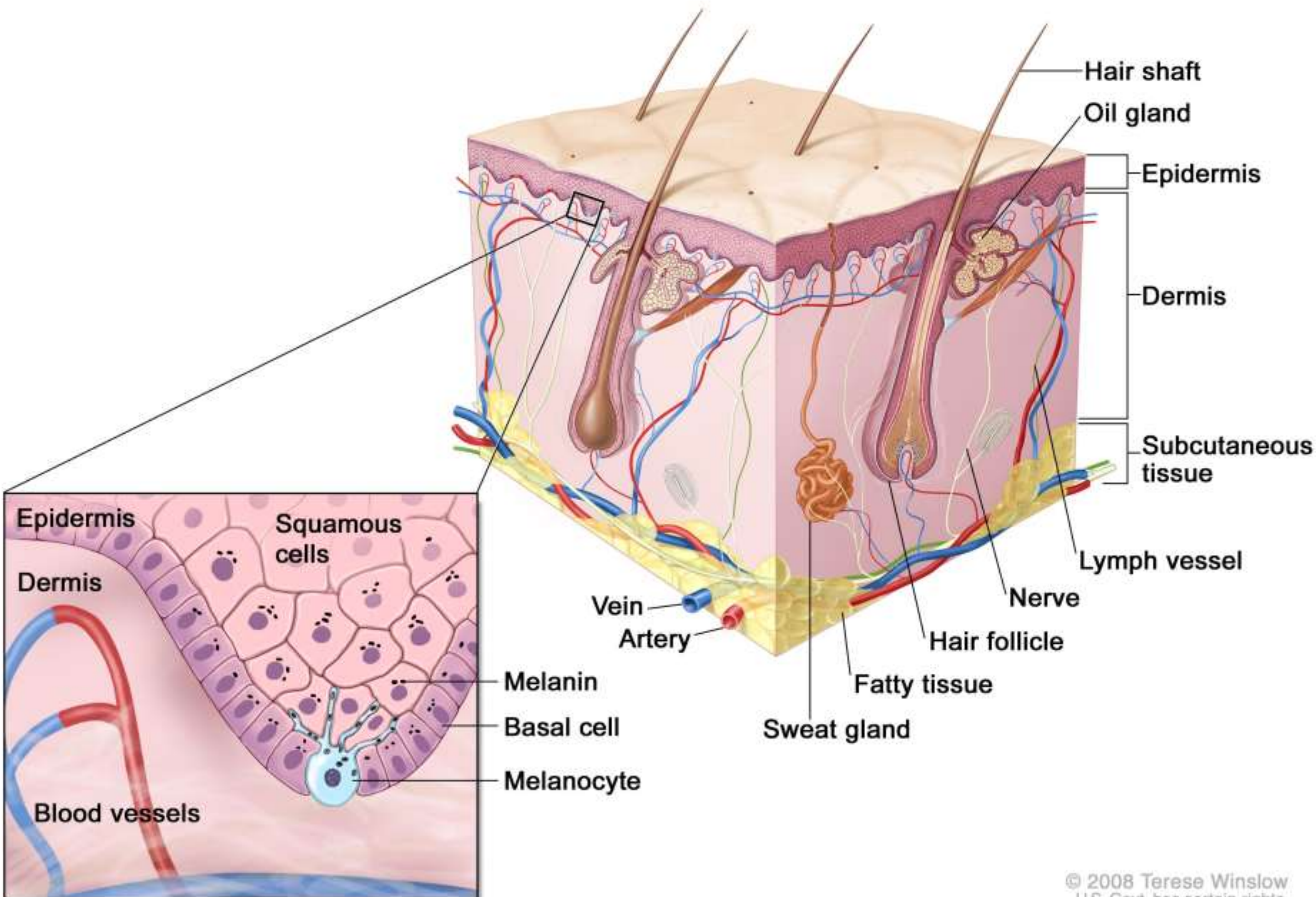

Neurosecretory
cell
Blood
vessel
Releasing
hormones
from
hypothalamus
Endocrine cells
of the anterior
pituitary
Pituitary hormones
TSH
ACTH
FSH
and
LH
Growth
hormone
(GH)
Prolactin
(PRL)
Endor-
phins
Thyroid
Adrenal
cortex
Testes or
ovaries
Entire
body
Mammary
glands
(in
mammals)
Pain
receptors
in the
brain

SKIN

Cutis

The skin darkens when melanocyte-stimulating(MSH)-produced in a thin layer between the two pituitary lobes – cause cells called melanocytes in skin tissue to produce more melanin pigment





Hair shaft
Oil gland
Epidermis
Dermis
Subcutaneous tissue
Lymph vessel
Nerve
Hair follicle
Fatty tissue
Sweat gland
Vein
Artery
Epidermis
Squamous cells
Dermis
Melanin
Basal cell
Melanocyte
Blood vessels
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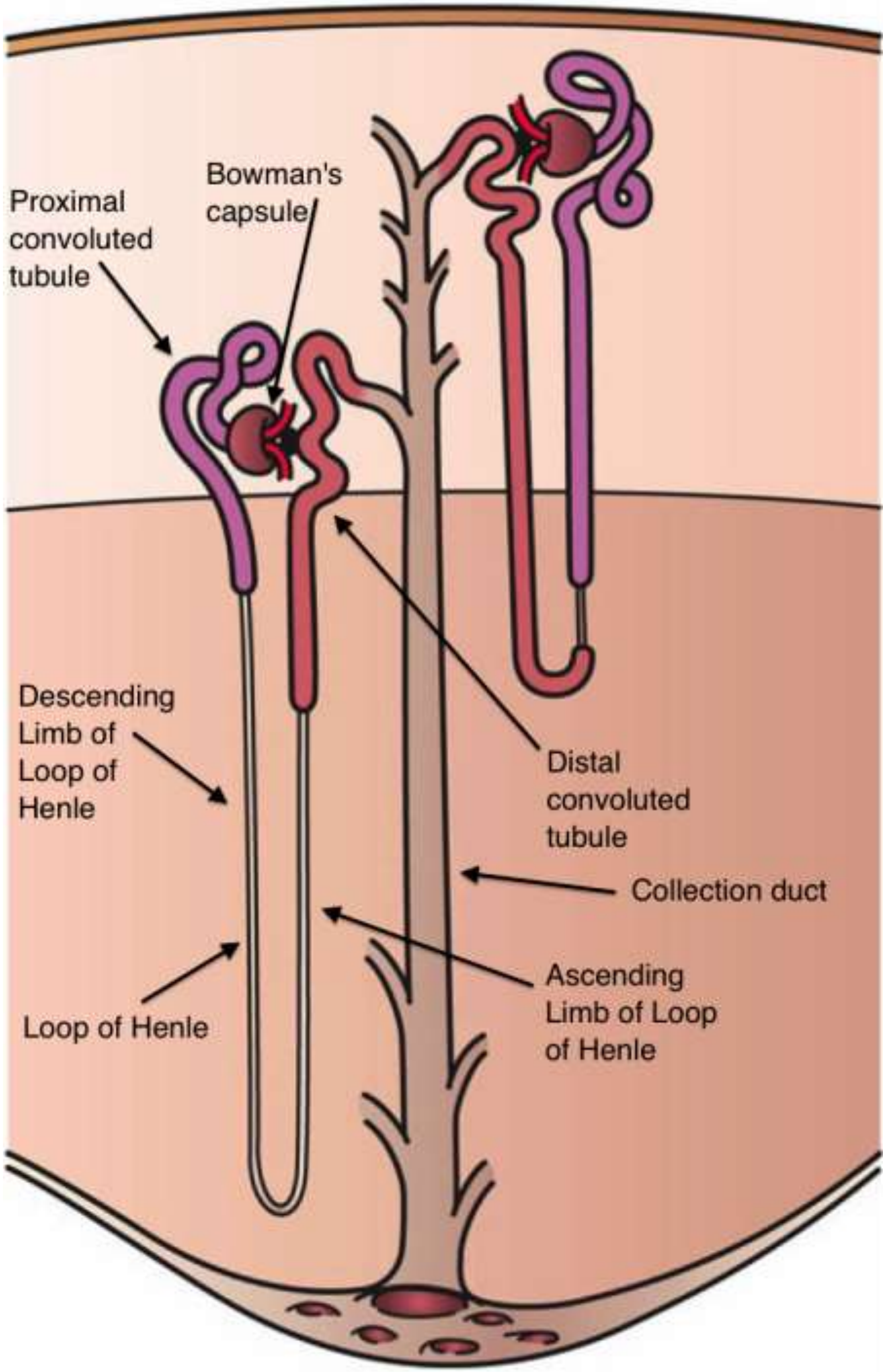
KIDNEY TUBULES

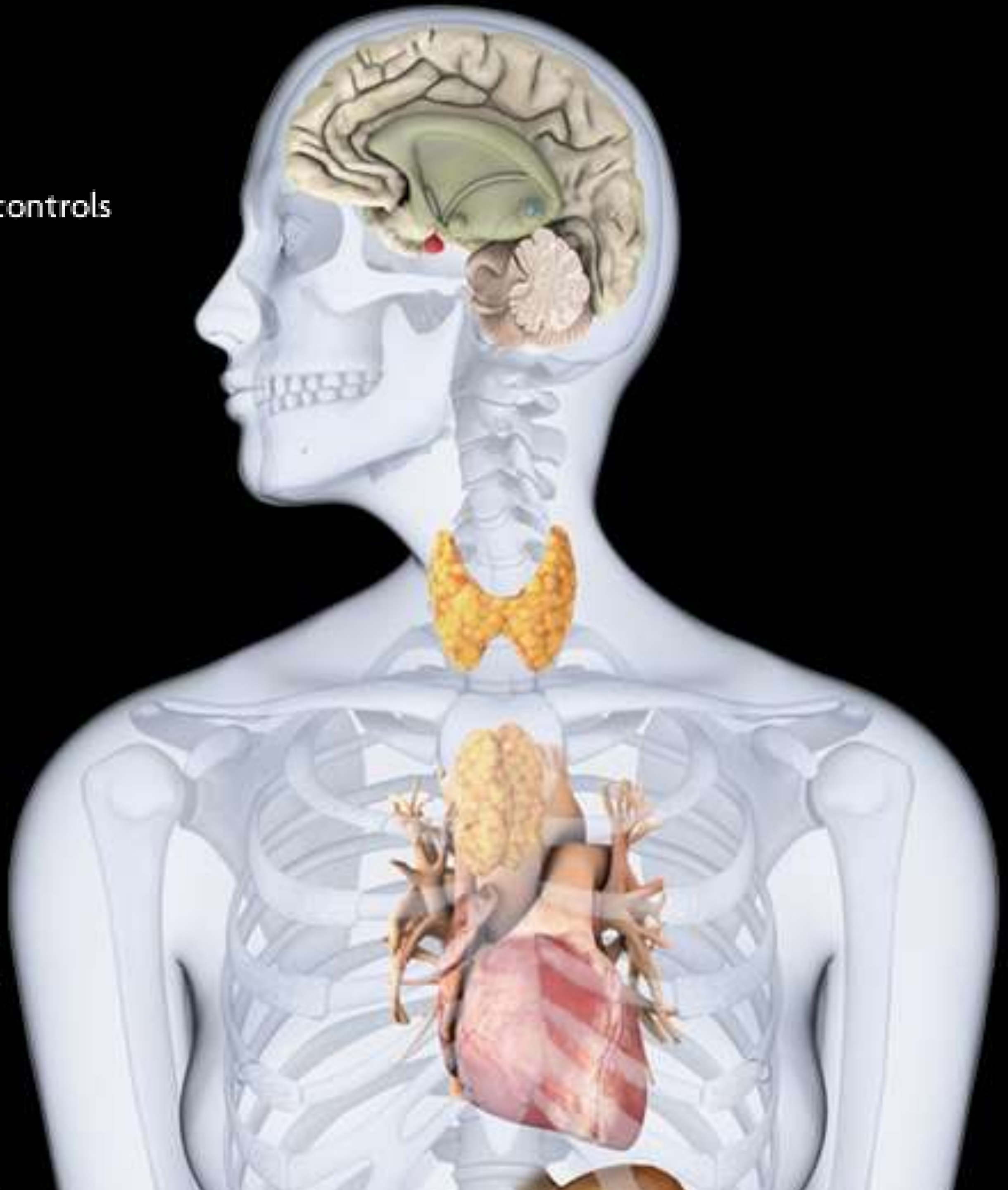
- **Antidiuretic hormone binds to receptors on cells in the collecting ducts of the kidney and promotes reabsorption of water back into the circulation. In the absense of antidiuretic hormone, the collecting ducts are virtually impermiable to water, and it flows out as urine.**
- **Antidiuretic hormone stimulates water reabsorbtion by stimulating insertion of "water channels" or aquaporins into the membranes of kidney tubules.**



PITUITARY VESSELS AND NERVES

Pituitary gland

Called the "master gland", this organ controls many other endocrine glands

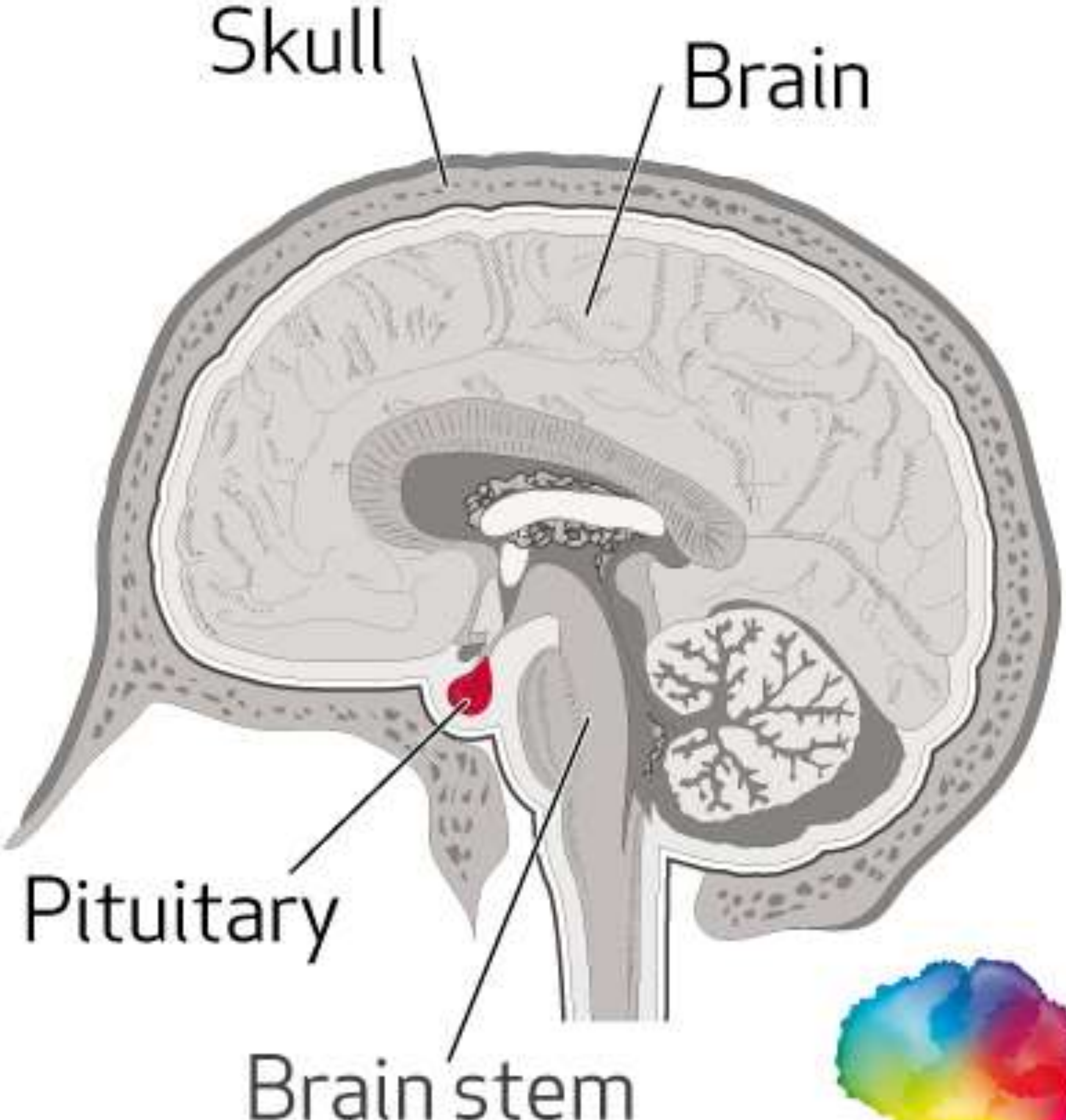


ENDOCRINE SYSTEM

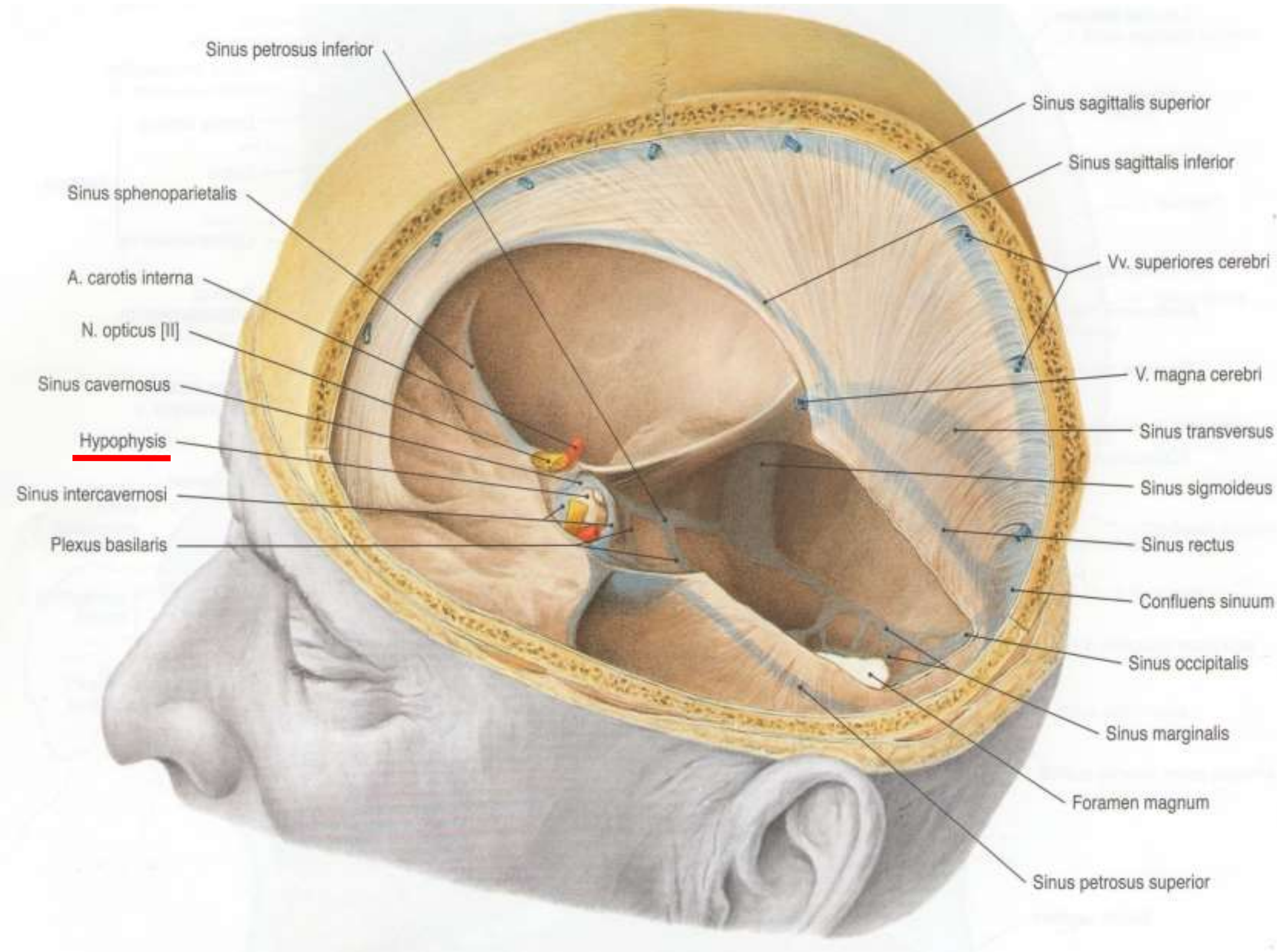
The body's chemical messengers (hormones) are made by endocrine glands. These glands have no ducts but secrete their hormones directly into the blood, by which means they reach every cell in the body. Hormones affect certain target tissues or organs and regulate their activities. For more detail, see *The Human Body Book*, pp.122–23.

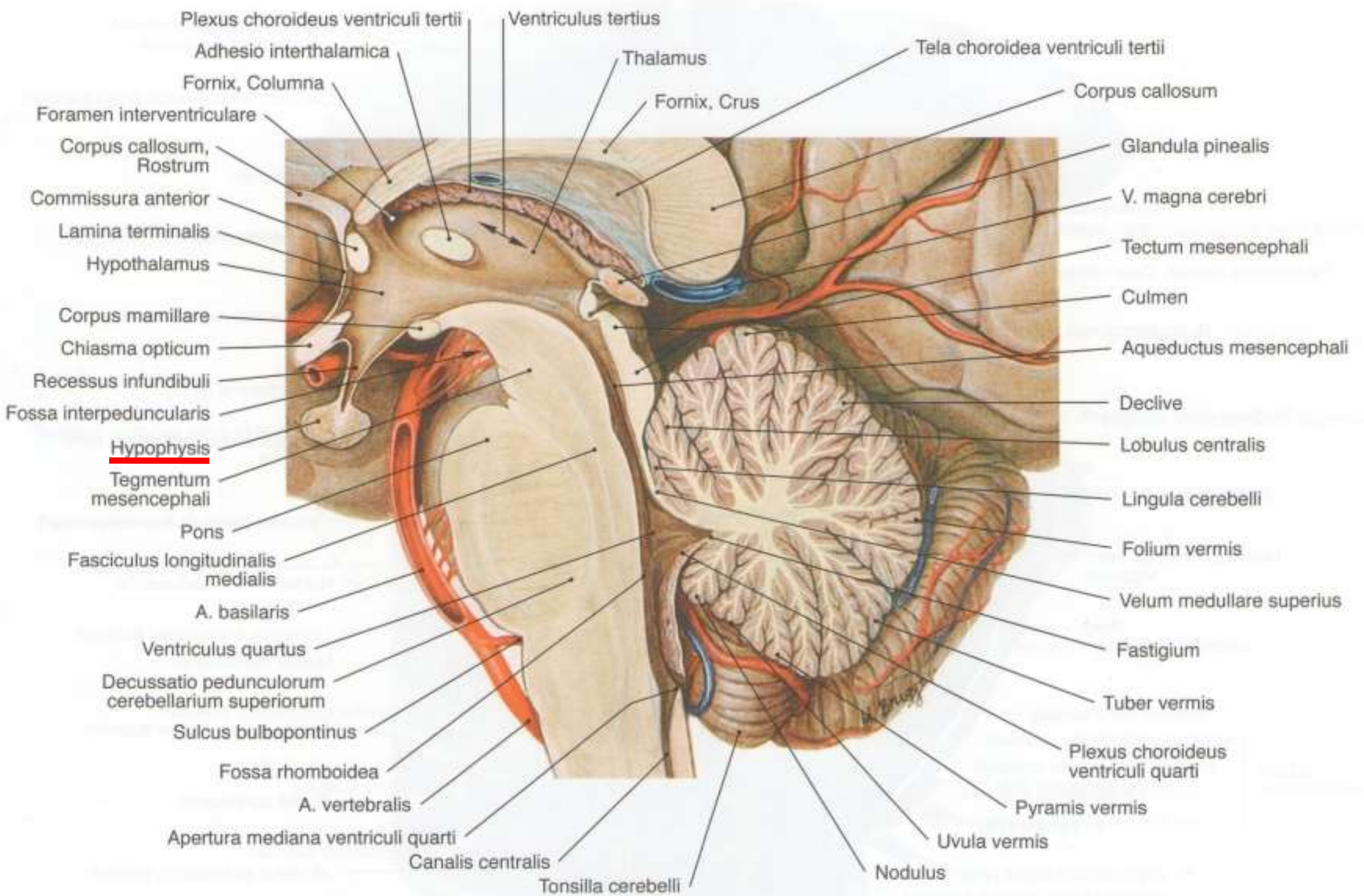
Pituitary gland

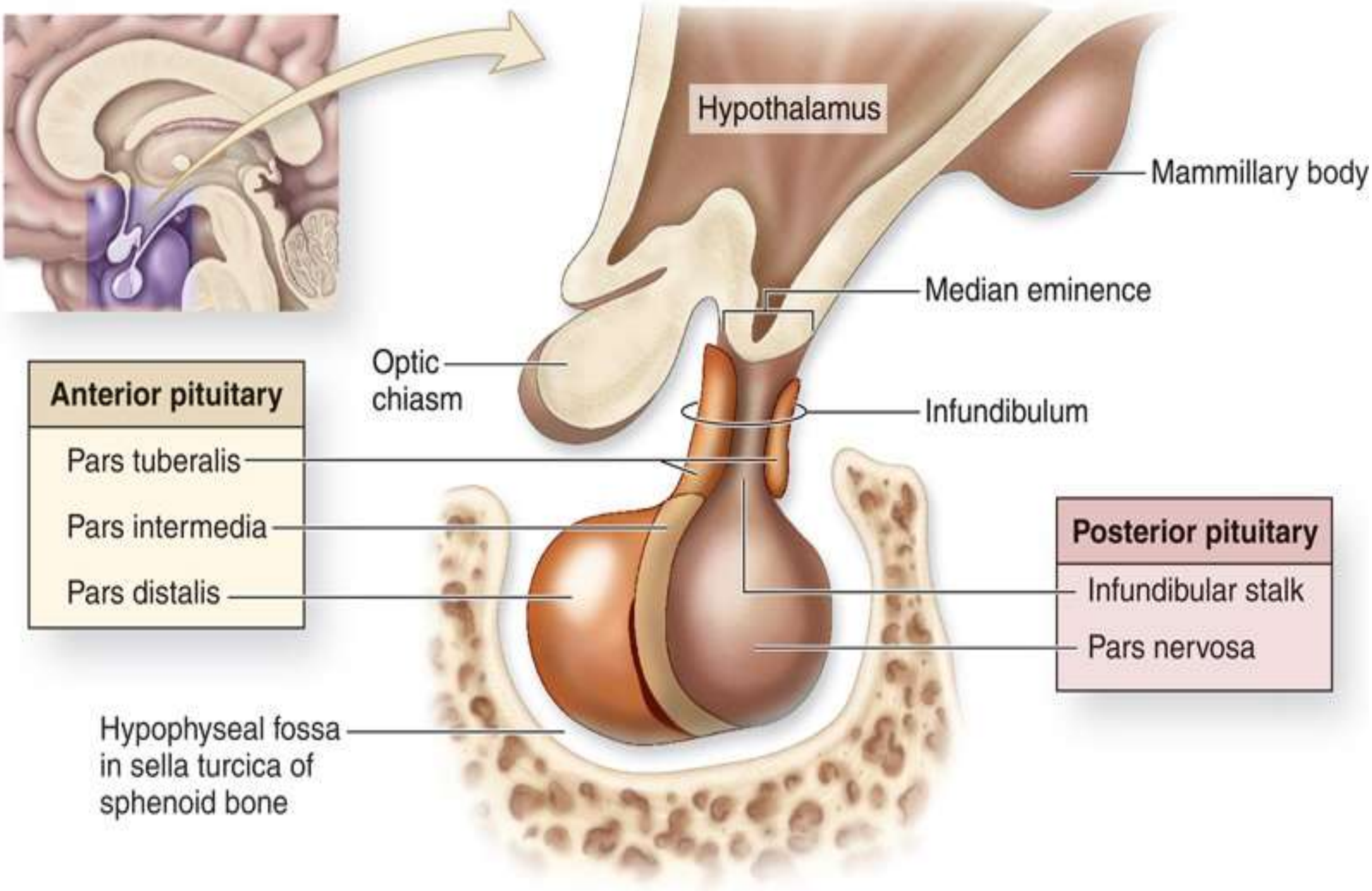
The pituitary gland, also known as the hypophysis, is connected to the hypothalamus of the brain by a tiny isthmus of nervous tissue called the infundibulum. It sits under the a small cavity inside the sphenoid bone of the skull known as the hypophyseal fossa. Thus the sphenoid bone surrounds and protects the delicate pituitary gland from damage by external forces.

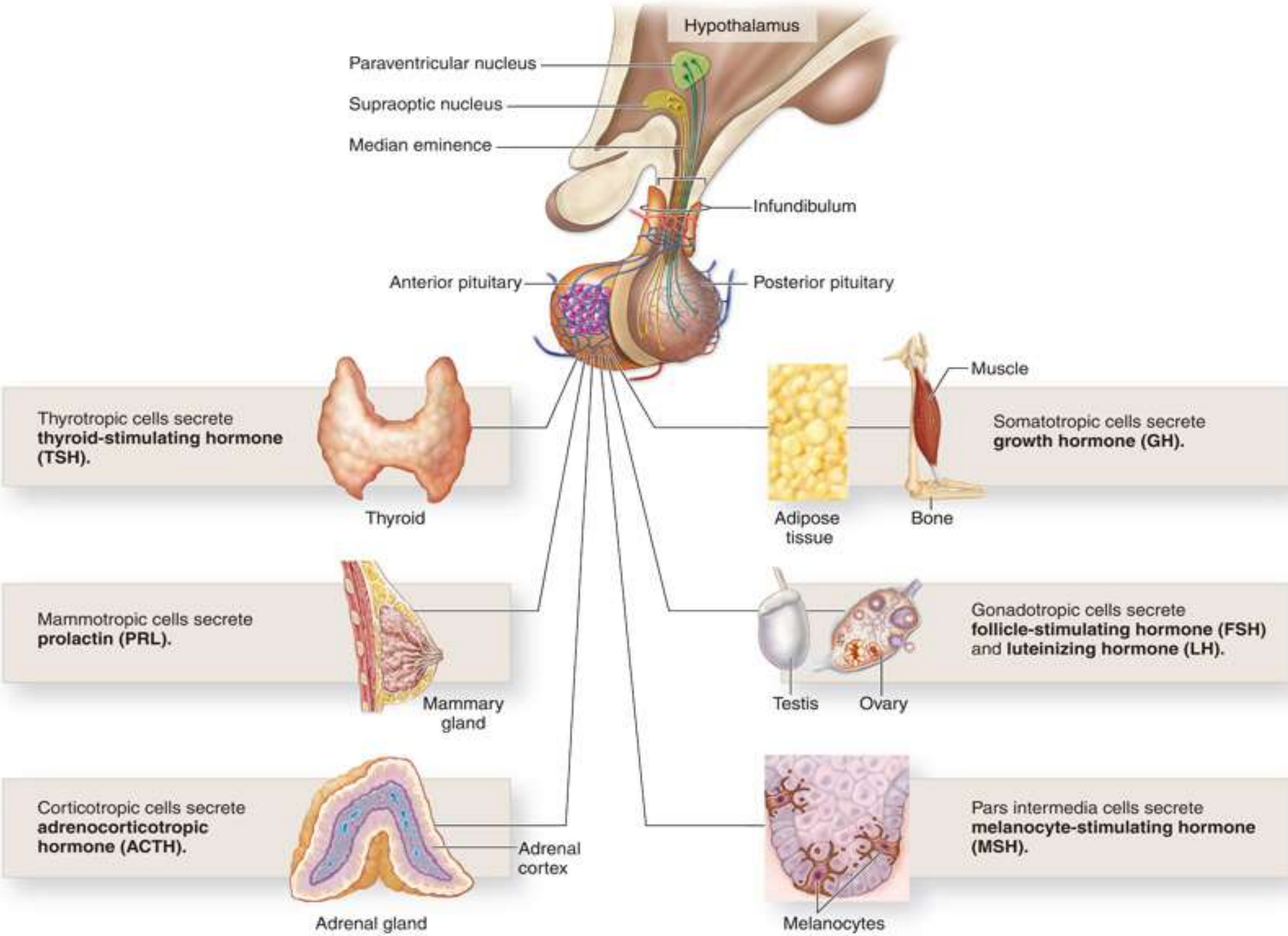


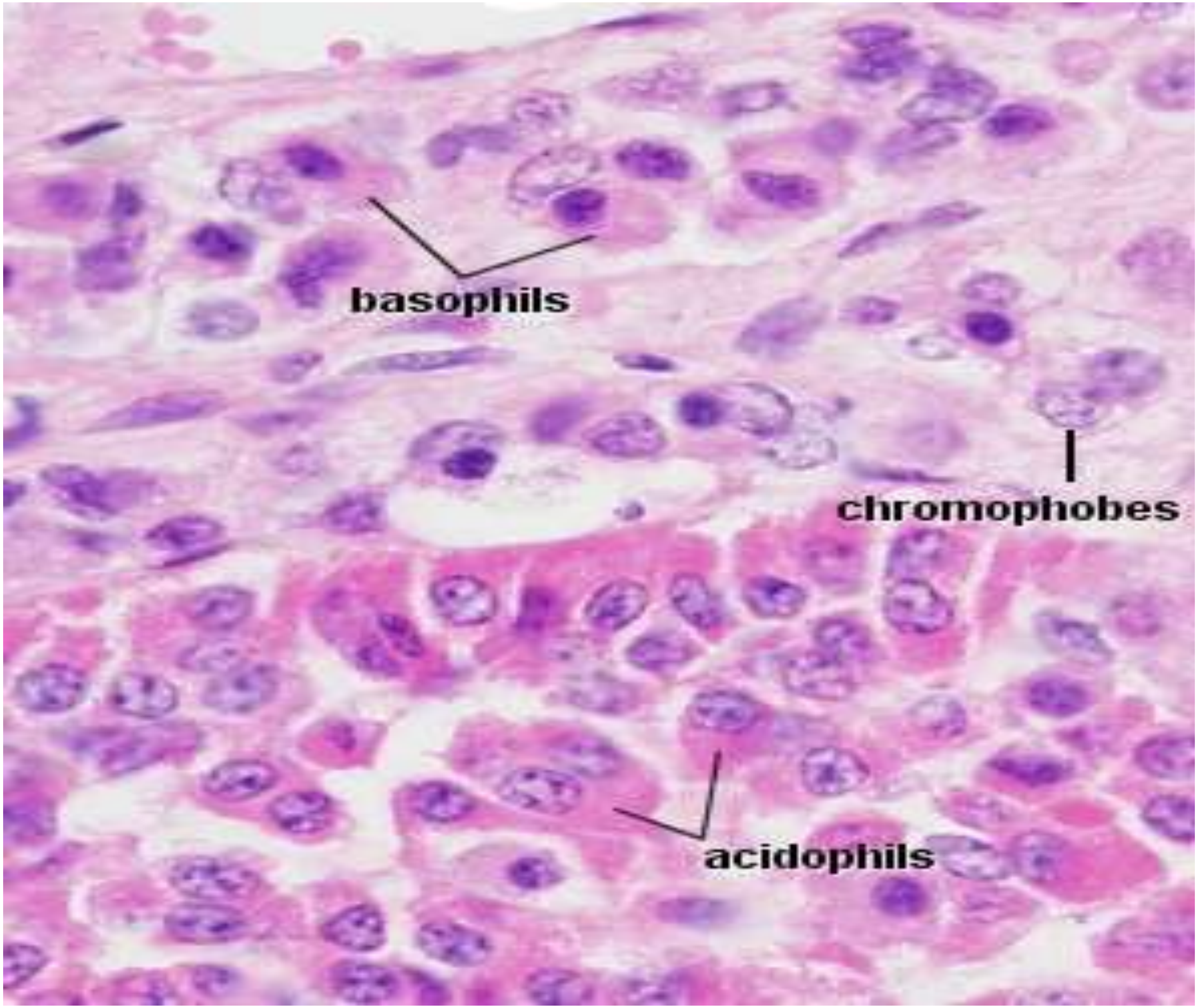
BRAIN
TUMOUR
UK

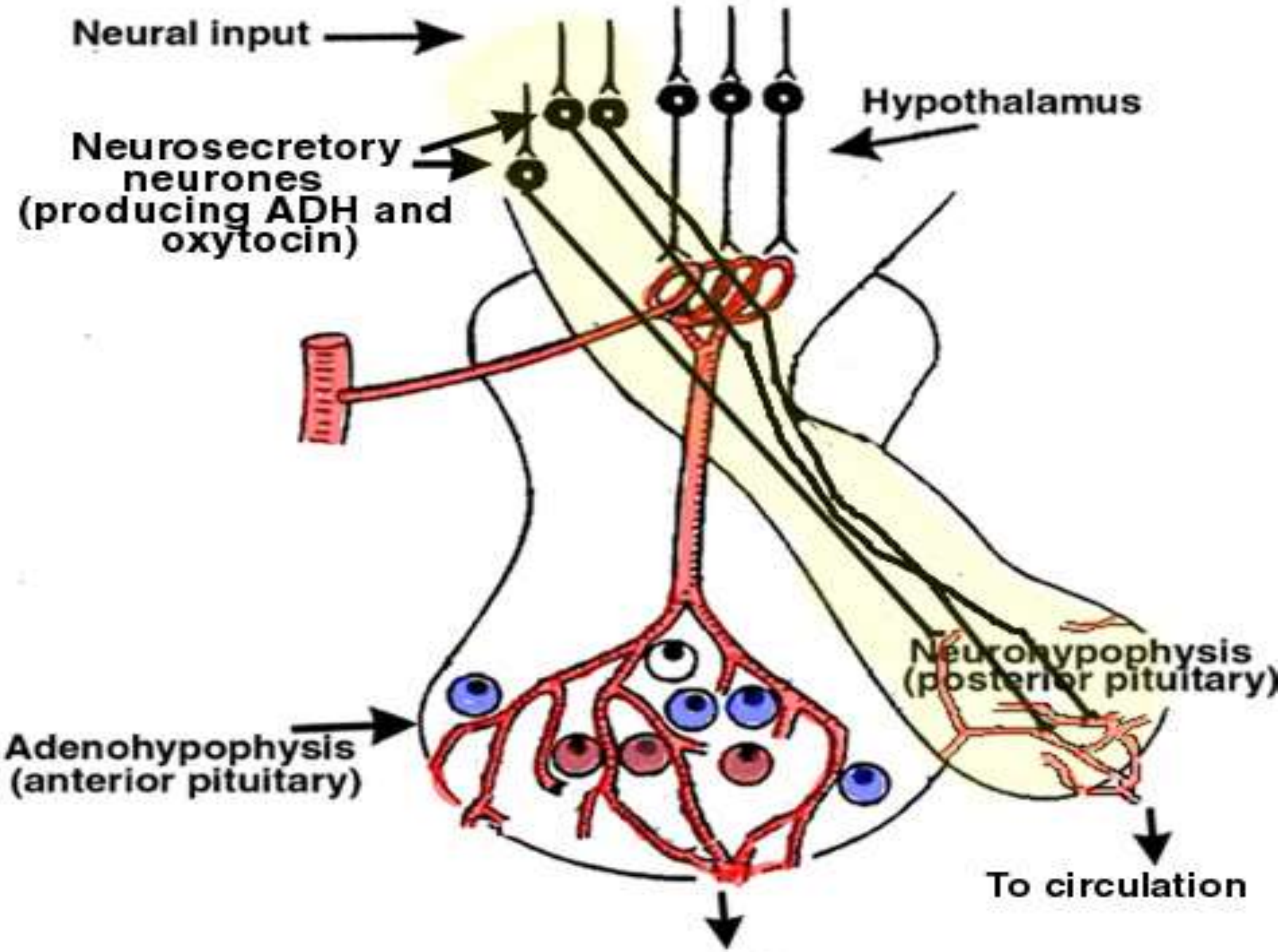

Sinus petrosus inferior
Sinus sphenoparietalis
A. carotis interna
N. opticus [II]
Sinus cavernosus
Hypophysis
Sinus intercavernosi
Plexus basilaris
Sinus sagittalis superior
Sinus sagittalis inferior
Vv. superiores cerebri
V. magna cerebri
Sinus transversus
Sinus sigmoideus
Sinus rectus
Confluens sinuum
Sinus occipitalis
Sinus marginalis
Foramen magnum
Sinus petrosus superior


Plexus choroideus ventriculi tertii
Ventriculus tertius
Adhesio interthalamica
Thalamus
Tela choroidea ventriculi tertii
Fornix, Columna
Fornix, Crus
Corpus callosum
Foramen interventriculare
Corpus callosum, Rostrum
Glandula pinealis
Commissura anterior
V. magna cerebri
Lamina terminalis
Tectum mesencephali
Hypothalamus
Culmen
Corpus mamillare
Aqueductus mesencephali
Chiasma opticum
Recessus infundibuli
Declive
Fossa interpeduncularis
Lobulus centralis
Hypophysis
Tegmentum mesencephali
Lingula cerebelli
Pons
Folium vermis
Fasciculus longitudinalis medialis
Velum medullare superius
A. basilaris
Ventriculus quartus
Fastigium
Decussatio pedunculorum cerebellarium superiorum
Tuber vermis
Sulcus bulbopontinus
Plexus choroideus ventriculi quarti
Fossa rhomboidea
Pyramis vermis
A. vertebralis
Apertura mediana ventriculi quarti
Uvula vermis
Canalis centralis
Nodulus
Tonsilla cerebelli

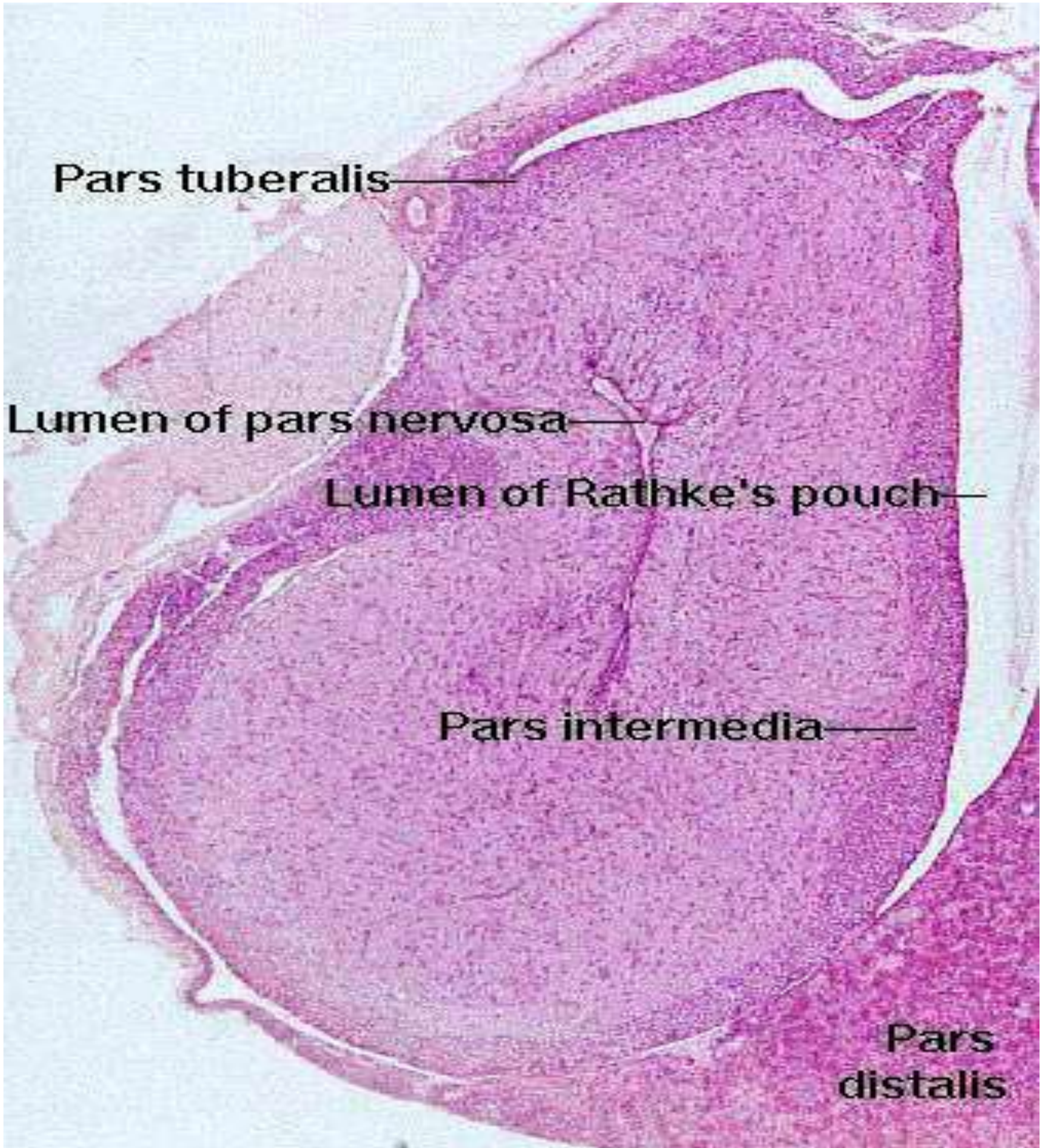

Hypothalamus
Mammillary body
Median eminence
Optic chiasm
Infundibulum
Anterior pituitary
Pars tuberalis
Pars intermedia
Pars distalis
Posterior pituitary
Infundibular stalk
Pars nervosa
Hypophyseal fossa in sella turcica of sphenoid bone

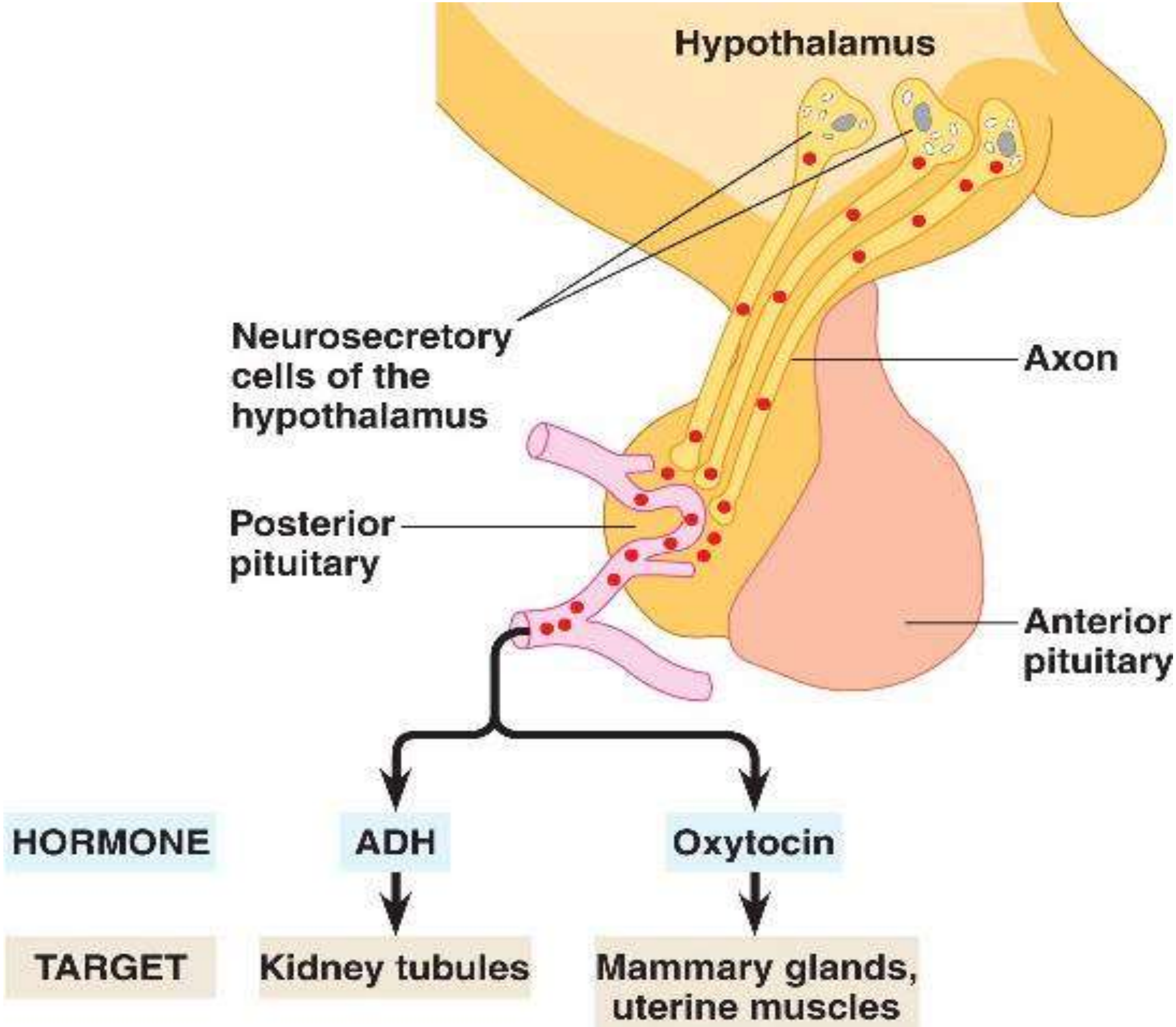

Hypothalamus
Paraventricular nucleus
Supraoptic nucleus
Median eminence
Infundibulum
Anterior pituitary
Posterior pituitary
Thyrotropic cells secrete **thyroid-stimulating hormone (TSH).**
Thyroid
Muscle
Somatotropic cells secrete **growth hormone (GH).**
Adipose tissue
Bone
Mammotropic cells secrete **prolactin (PRL).**
Mammary gland
Gonadotropic cells secrete **follicle-stimulating hormone (FSH)** and **luteinizing hormone (LH).**
Testis
Ovary
Corticotropic cells secrete **adrenocorticotropic hormone (ACTH).**
Adrenal cortex
Adrenal gland
Pars intermedia cells secrete **melanocyte-stimulating hormone (MSH).**
Melanocytes

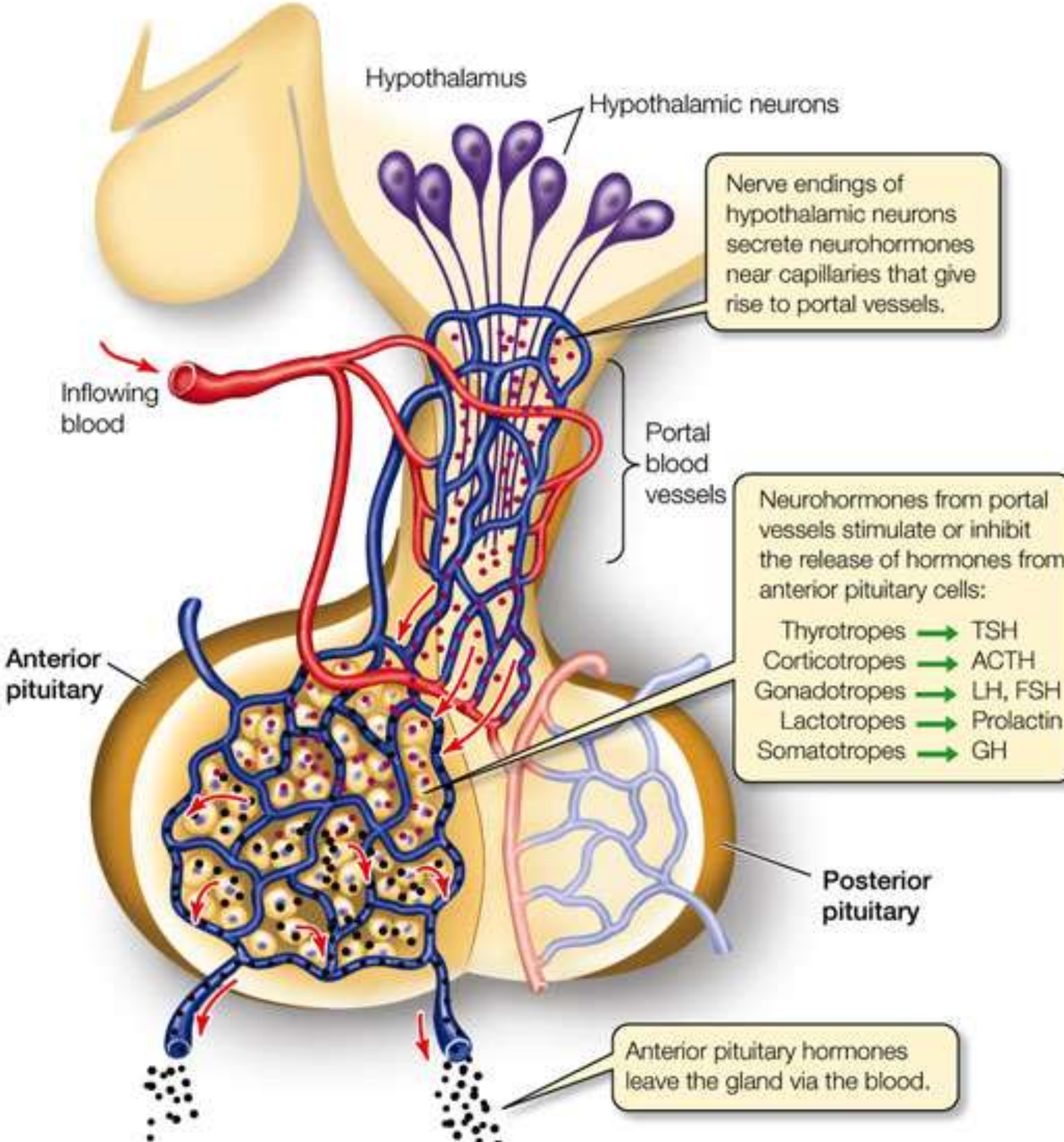

basophils
chromophobes
acidophils



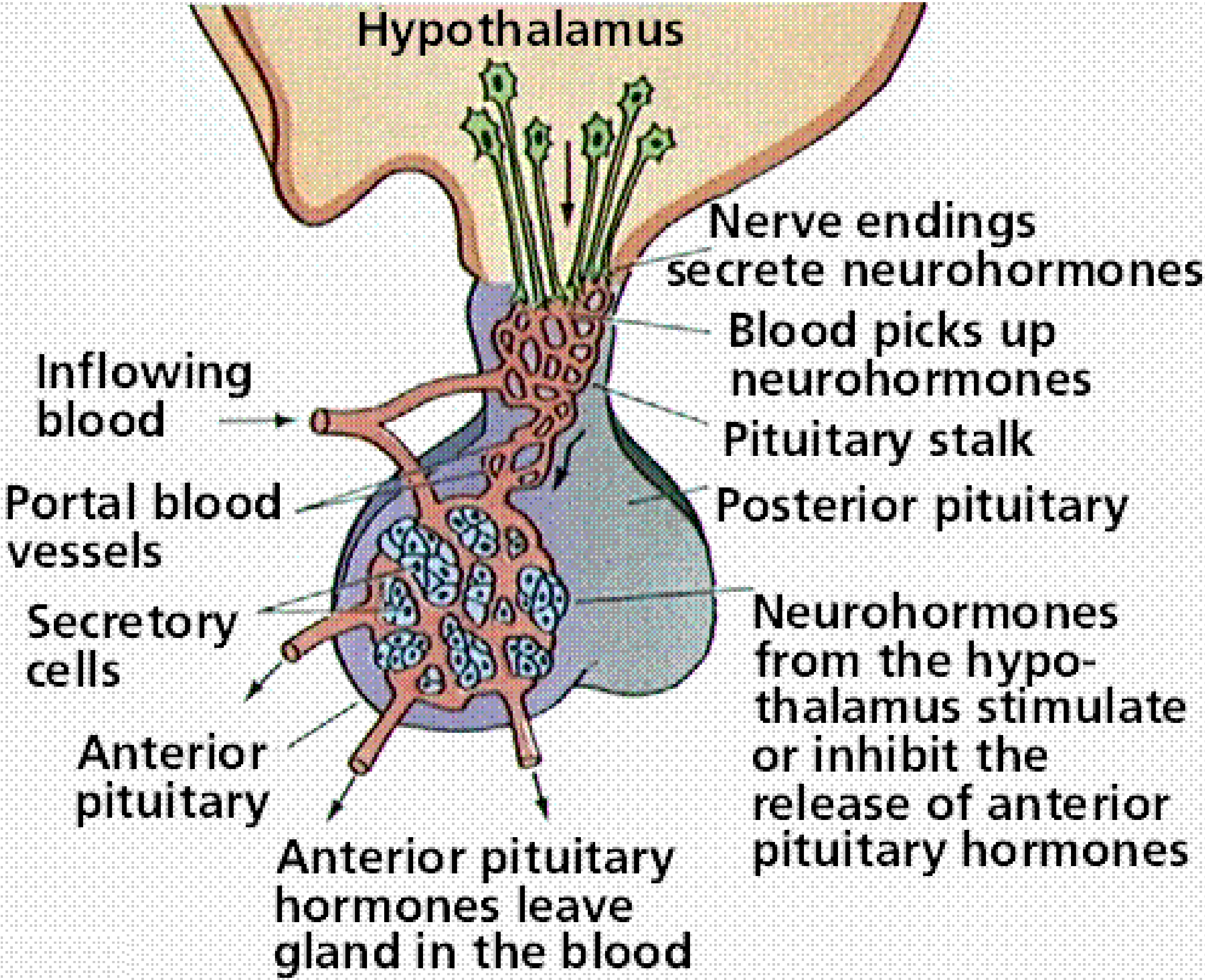
Neural input
Hypothalamus
Neurosecretory neurones (producing ADH and oxytocin)
Neurohypophysis (posterior pituitary)
Adenohypophysis (anterior pituitary)
To circulation


Pars tuberalis
Lumen of pars nervosa
Lumen of Rathke's pouch
Pars intermedia
Pars
distalis


Hypothalamus
Neurosecretory cells of the hypothalamus
Axon
Posterior pituitary
Anterior pituitary
HORMONE
ADH
Oxytocin
TARGET
Kidney tubules
Mammary glands, uterine muscles



Hypothalamus
Hypothalamic neurons
Nerve endings of hypothalamic neurons secrete neurohormones near capillaries that give rise to portal vessels.
Inflowing blood
Portal blood vessels
Neurohormones from portal vessels stimulate or inhibit the release of hormones from anterior pituitary cells:
Thyrotropes → TSH
Corticotropes → ACTH
Gonadotropes → LH, FSH
Lactotropes → Prolactin
Somatotropes → GH
Anterior pituitary
Posterior pituitary
Anterior pituitary hormones leave the gland via the blood.


Hypothalamus
Nerve endings secrete neurohormones
Blood picks up neurohormones
Pituitary stalk
Posterior pituitary
Inflowing blood
Portal blood vessels
Secretory cells
Anterior pituitary
Neurohormones from the hypo-thalamus stimulate or inhibit the release of anterior pituitary hormones
Anterior pituitary hormones leave gland in the blood

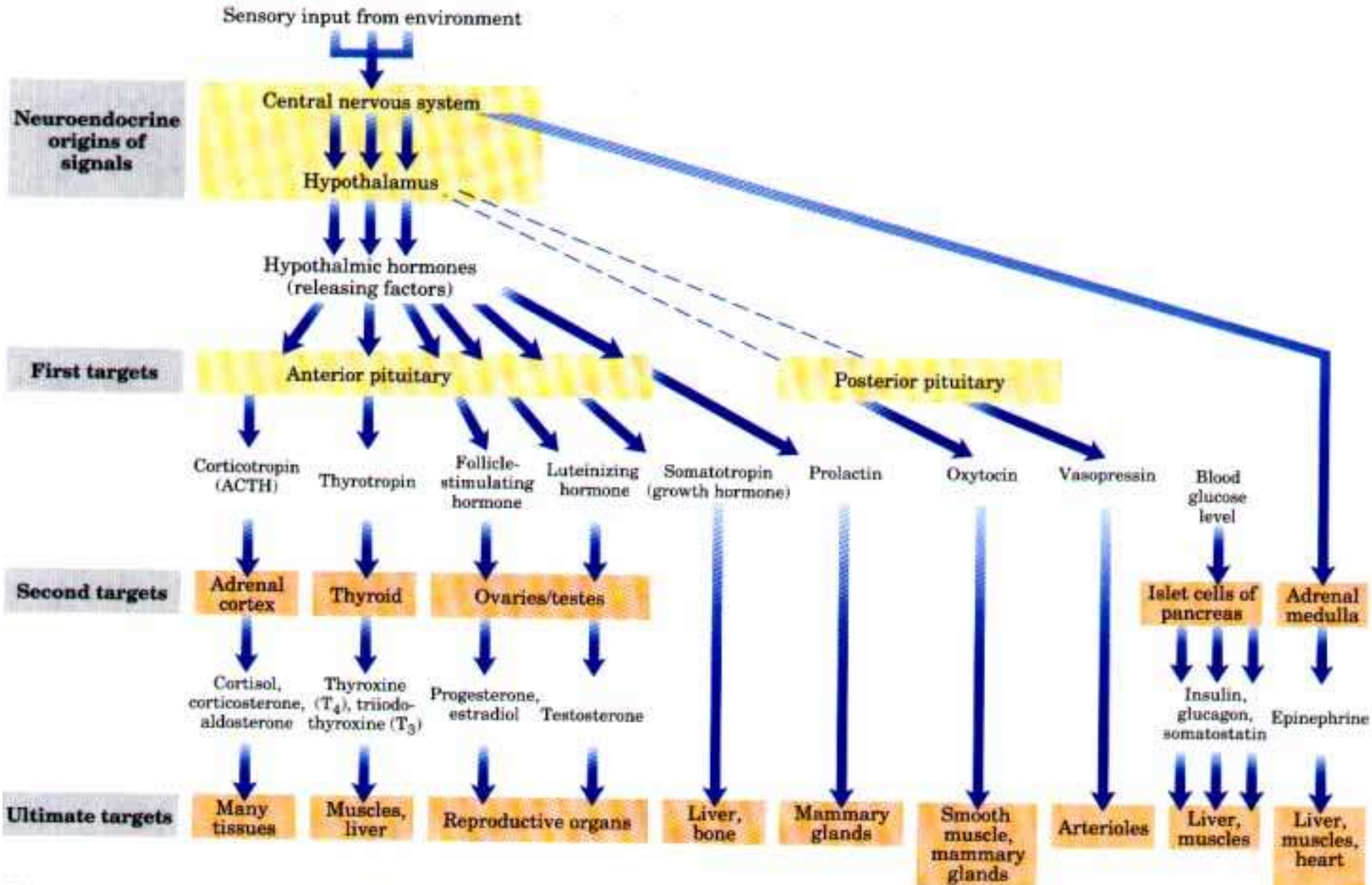
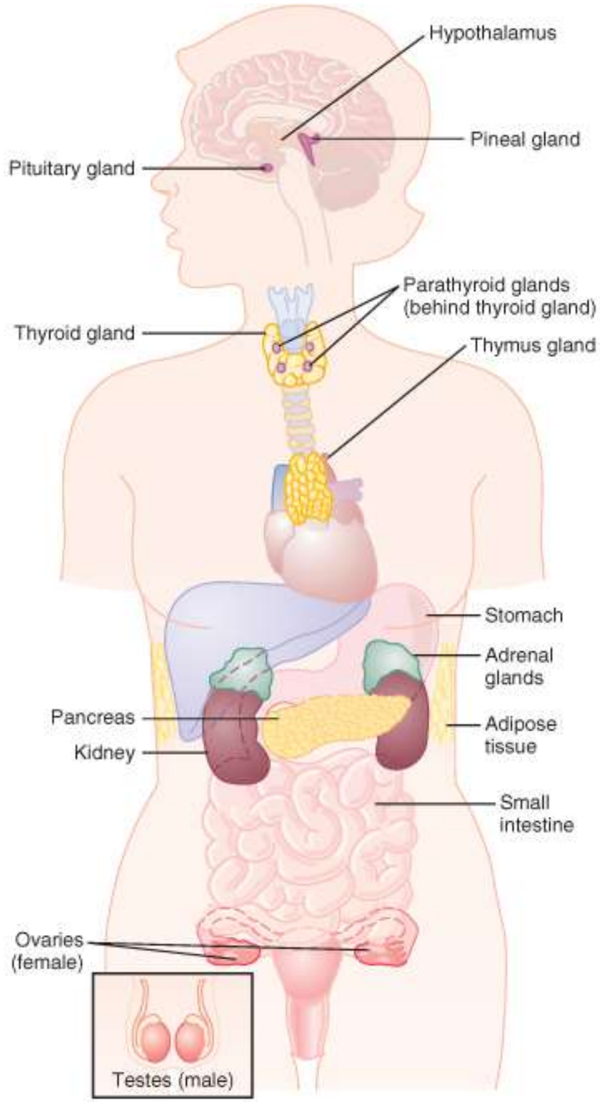


Figure 22–14 The major endocrine systems and



The Endocrine System

Endocrine System

- The endocrine system include all the organs of the body which called endocrine glands.
- An endocrine gland secretes hormones.
- Hormones are molecules that are secreted into the blood.
- Hormones are substances that are secreted by one group of cells that affects the physiology of another group of cells (organs). The endocrine system is controlled by the pituitary gland and the hypothalamus.
- Compared to most other organs in the body, endocrine organs are well vascularized.
- VIDEO http://www.youtube.com/watch?v=HrMi4GikWwQ

The Endocrine System

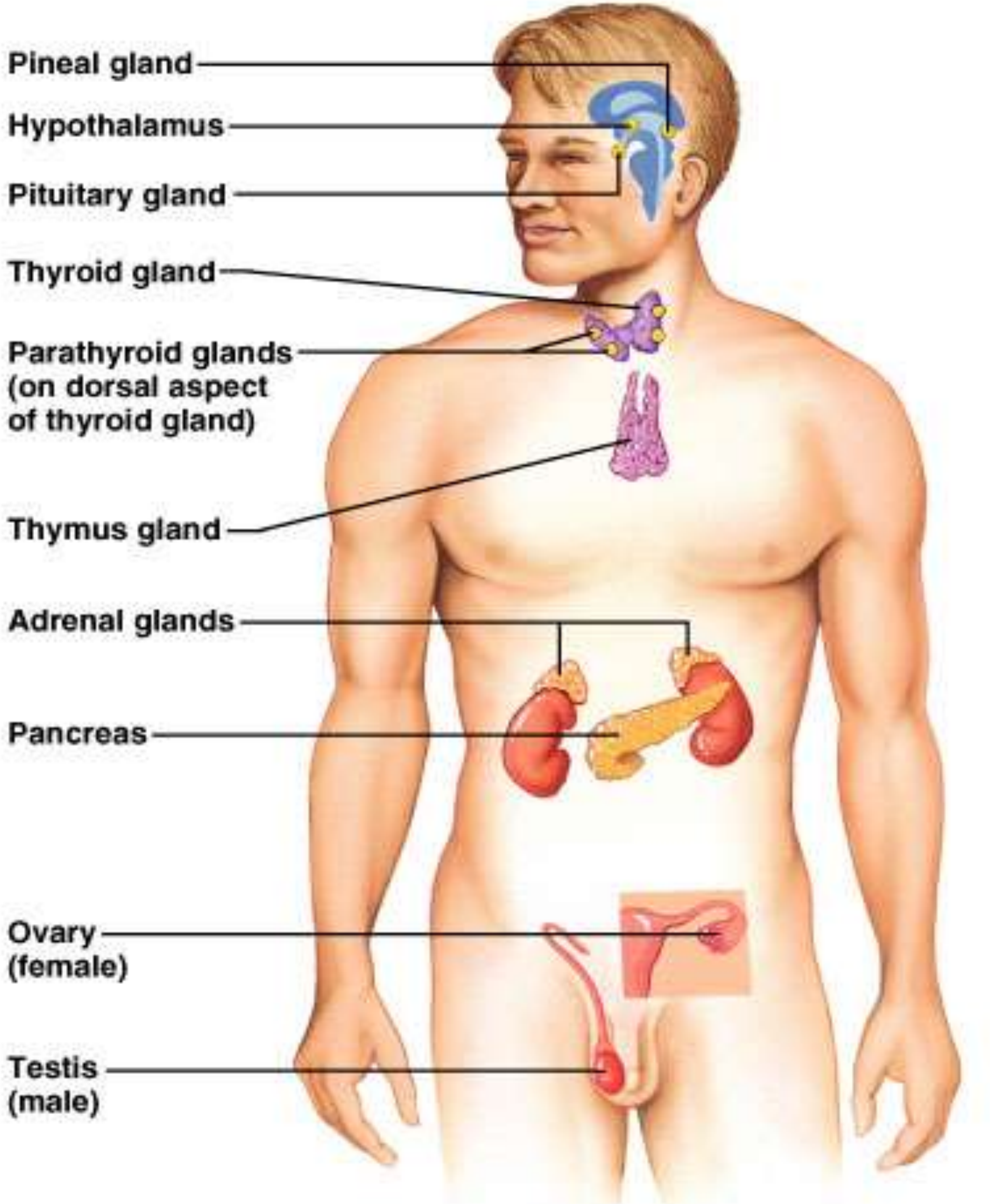
- A system of **ductless** glands
  - Secrete messenger molecules called **hormones**
- Interacts closely with the nervous system
- **Endocrinology** – study of hormones and endocrine glands

Major Endocrine Glands

- Hypothalamus
- Pituitary Gland
- Thyroid Gland
- Parathyroid Glands
- Thymus Gland
- Adrenal Glands
- Pancreas
- Ovaries
- Testes
- Pineal Gland

Endocrine Organs

- Scattered throughout the body
- Pure endocrine organs
  - Pituitary, pineal, thyroid, parathyroid, and adrenal glands
- Organs containing endocrine cells
  - Pancreas, thymus, gonads, and the hypothalamus
- Richly vascularized


Pineal gland
Hypothalamus
Pituitary gland
Thyroid gland
Parathyroid glands (on dorsal aspect of thyroid gland)
Thymus gland
Adrenal glands
Pancreas
Ovary (female)
Testis (male)

Endocrine System

- The endocrine system is a series of glands that release a hormone into the plasma, where it is dissolved and transported throughout entire body within 60 seconds.
- Every cell is exposed to the hormone, but not every cell responds to it. **For a cell to be able to respond to a hormone, the cell must have a functional hormone receptor.** A cell that responds will do so in various ways. The cells in the heart, pancreas, and brain respond to epinephrine differently. One thing that always happens is that **a cell will change its physiology in response to a hormone.**

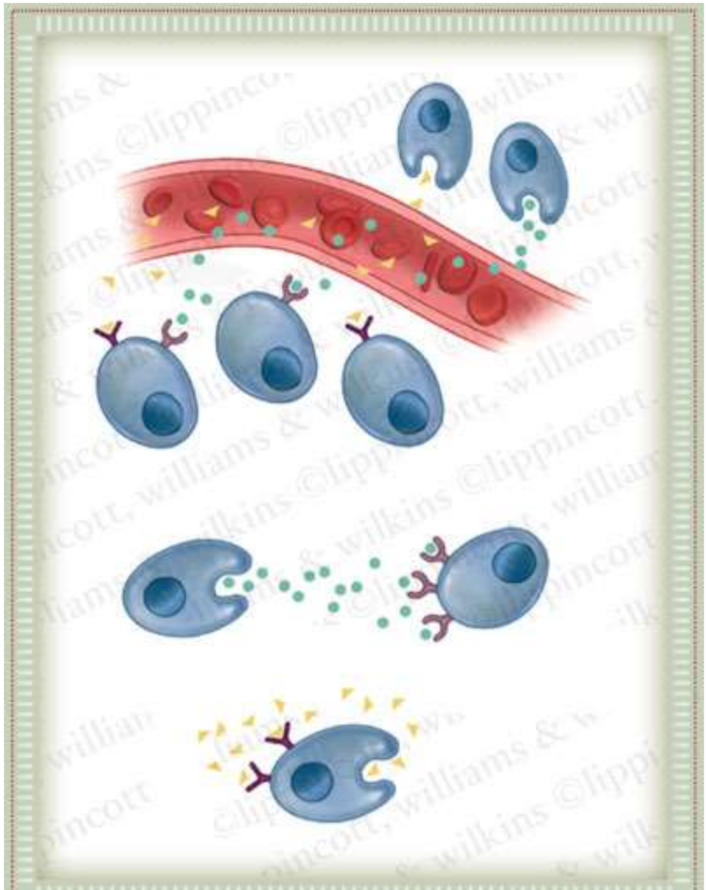
Hormones

- Hormones can be **synergistic;** aldosterone and antidiuretic hormone (ADH) both help increase volume of fluid in body to raise blood pressure.
- Some hormones are **antagonists**; Atrial natriuretic peptide (ANP, produced by heart cells) is released when you have high blood pressure. It causes the kidney to secrete more water, so blood pressure can decrease. That is the opposite of ADH, which makes you urinate less.
- Some hormones are **permissive;** you need one in order for a second to do its job well. **Thyroid hormone is permissive for growth hormone** (you need thyroid hormone in order for GH to work)**.** Not enough thyroid hormone can cause stunted growth, even if enough growth hormone is present.

Hormones

- Basic hormone action
  - Hormones are made by the gland's cells, possibly stored, then released
  - Circulate throughout the body vasculature, fluids
  - Influences only specific tissues: <u>target cells</u> that have a receptor for that particular hormone
  - A hormone can have different effects on different target cells: depends on the receptor
  - Some hormones are "permissive" for the actions of another (TH for GH)

Ultimate goal: alter cell activity by altering protein activity in the target cell.



What would happen if there was a defect in the hormone receptor on the target cell membrane? The hormone might be fine, but doesn't work.

http://www.megalo-media.com/art/ccolor3.html

Target Cell

- **A target cells have a functional receptor (a protein) for the hormone.** At home, you may watch TV with either a cable or satellite dish. Satellite waves are exposed to those homes with cable, but only those with dishes receive the signal. The target cell's receptor serves to convert the signal into a response.
- Receptors are proteins, which can be inside the cell or on its membrane. **What would happen if there were a gene defect in the DNA code for a receptor?** The receptor becomes faulty, and will not respond to the hormone. The receptor will also not function properly if the cell is exposed to excess salt, heat, or pH.

What is a “receptor”?

- It is a protein made by the target cell (protein synthesis after gene expression)
- The protein is made, then inserted into plasma membrane, or found in cytoplasm or nucleoplasm
- The active site on the protein “fits” the hormone
- Acts to convert the signal into a response

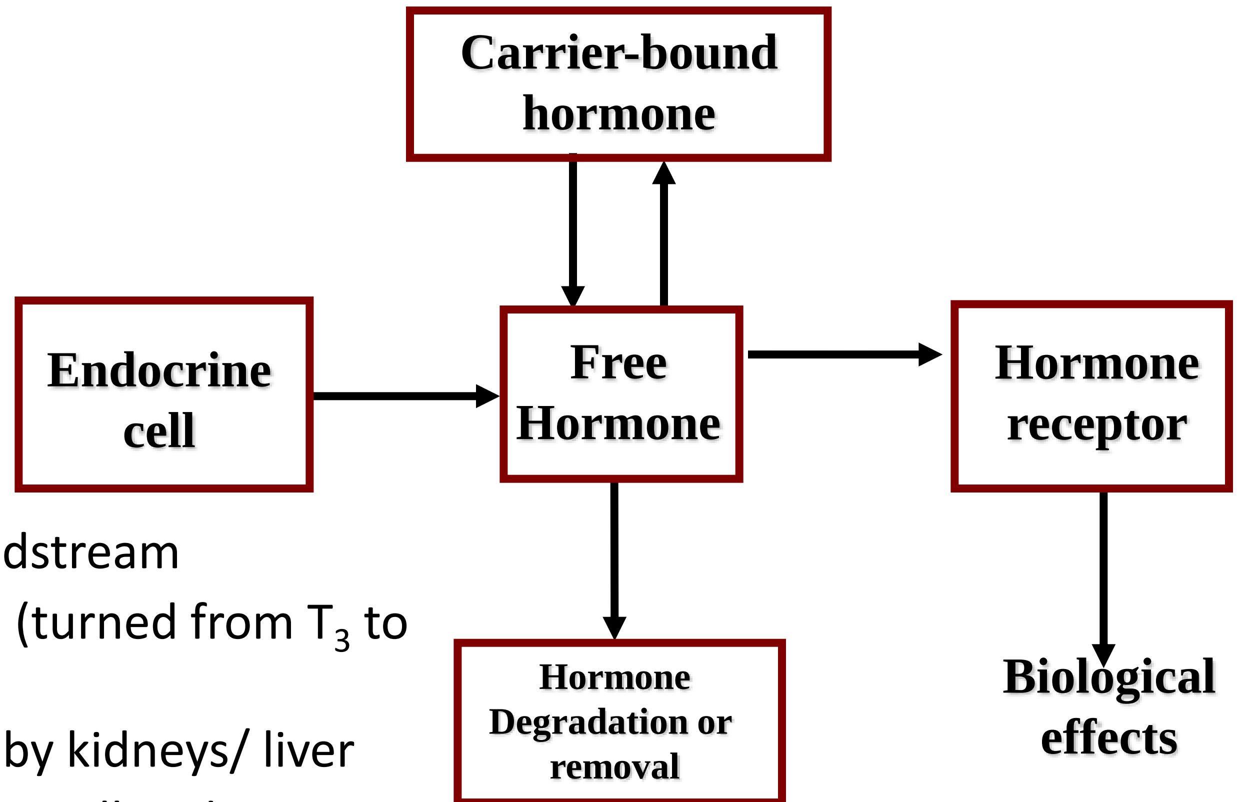
What would happen if there were a gene defect in the DNA code for a receptor?

What would happen if the receptor protein was denatured?

Hormones

- Endocrine glands secrete hormones into the plasma. Then, several different events could occur.
- It could bind to its receptor on the target cell, causing a change.
- Or, it could be destroyed by enzymes in the plasma.
- It could land in the kidneys and be filtered out before reaching its target.

What happens with hormones once it's secreted?



- Degraded in bloodstream
- May be activated (turned from T_3 to T_4)
- May be excreted by kidneys/ liver
- May reach a target cell and cause a cell response
- May need carrier to reach target cell

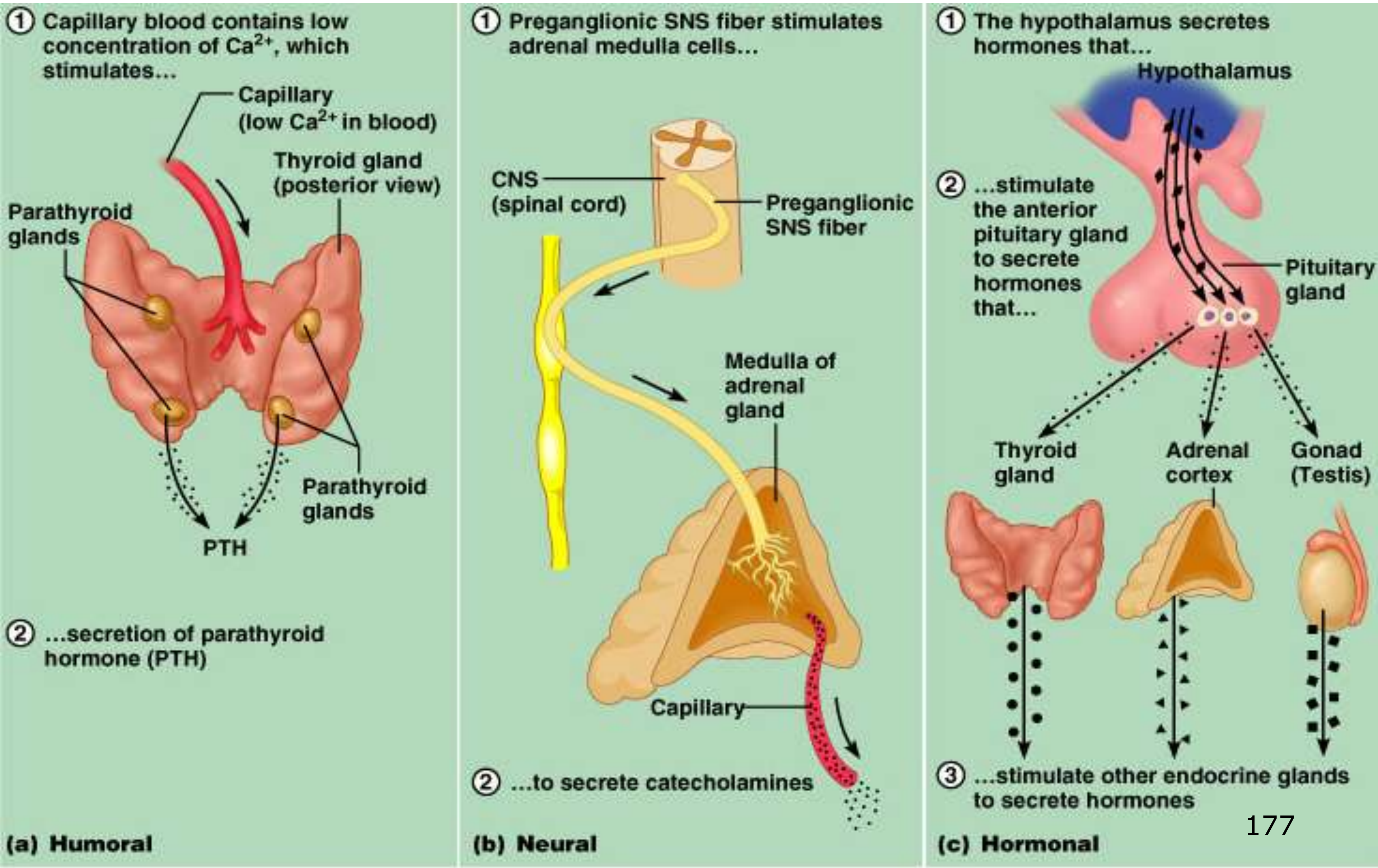
Control of Hormone Secretion

- The endocrine system is controlled by the pituitary gland and the hypothalamus.
- Always controlled by feedback loops
- Concentration declines below a minimum: more hormone is secreted
- Concentration exceeds maximum: Hormone production is halted

MECHANISMS OF HORMONE SECRETION

- **Humoral Trigger**
- Something in the blood is being monitored. When the level of that substance is too high or low, it stimulates the release of the hormone.
- **Neuronal Trigger**
- A neuron directly stimulates the gland to cause secretion of the hormone.
- **Hormonal Trigger**
- One endocrine gland releases a hormone that stimulates another endocrine gland to release its hormone.
- This is how thyroid hormone is secreted. The hypothalamus releases a hormone that causes the pituitary gland to release TSH, which causes the thyroid gland to release thyroid hormone.

Control of Hormones Release: Three Mechanisms



Humoral Trigger

- Something in the blood is being monitored. When the level of that substance is too high or low, it stimulates the release of the hormone or stop its production.
- Examples are **insulin, glucagon, parathyroid** hormone, and **aldosterone**.
- When you eat, glucose gets high, releases insulin, which tells cells to take in the sugar. Excess sugar is then converted to glycogen, which is the storage form.
- When glucose is low, glycogen is broken back down to glucose and released into the blood.
- When blood calcium is low, parathyroid gland hormone tells the intestinal cells to absorb more calcium, and kidneys to reabsorb more Ca^{++}, and stimulates osteoclasts to degrade bone matrix so calcium goes into blood.

Neuronal Trigger

- Examples are **oxytocin, ADH** (neurohypophysis hormones) **and Epinephrine** (adrenal medulla hormone)

Hormonal Trigger

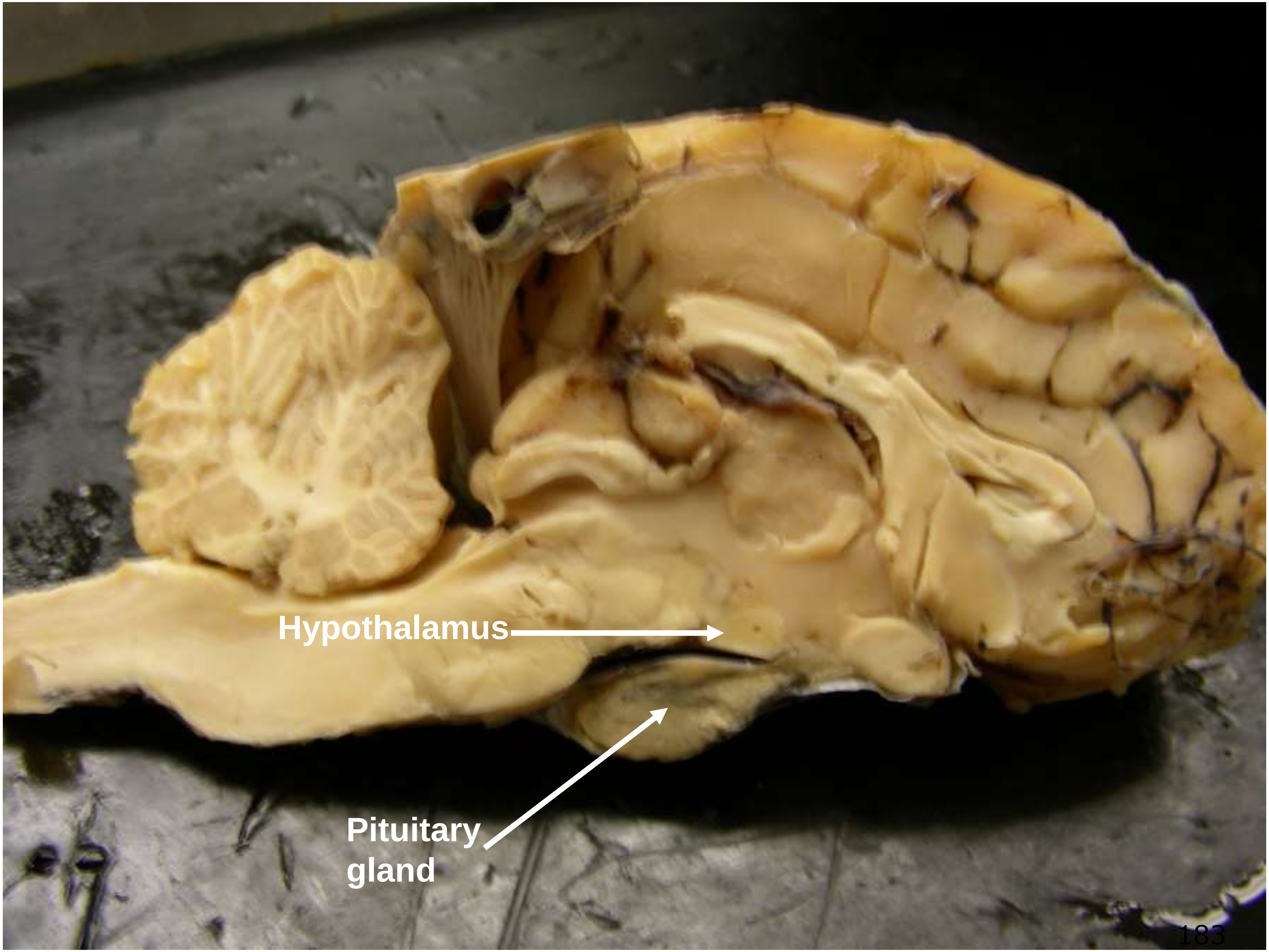
- This is when one endocrine gland releases a hormone that stimulates another endocrine gland to release its hormone.
- Examples are any of the hypothalamus or anterior pituitary hormones, and also the adrenal cortex (steroid) hormones (except aldosterone) and thyroid hormone.

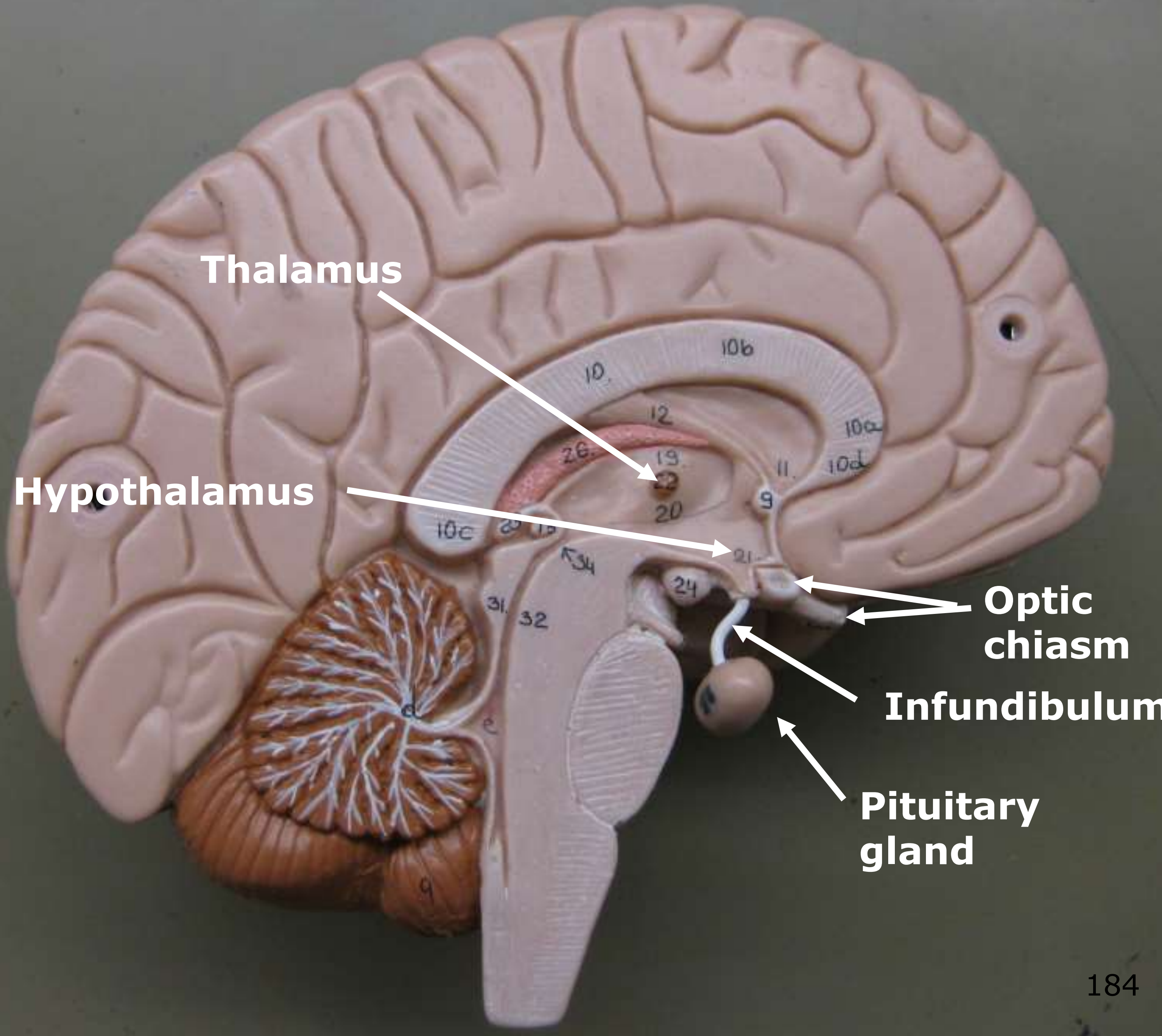
Hypothalamus

- This is located at the base of the brain. It is part of the limbic system, which controls the autonomic nervous system and the endocrine systems.
- The hypothalamus controls the endocrine system by controlling the pituitary gland.
  - Secretes **releasing hormones** to cause the pituitary to release hormones
  - Secretes **inhibiting hormones** to turn off secretion of pituitary hormones

The Pituitary Gland

- This is located in the sella tursica (totally encased in bone), which gives you a clue as to how important this gland is.
- The adenohypophysis portion of the pituitary gland (anterior lobe) actually develops from an embryonic pouch that grows upward from the ectoderm of the pharynx!
- One type of diabetes (insipidus) can be caused by trauma to the pituitary gland.
- A tumor of the pituitary gland can lead to blindness because it is so close to the optic chiasma.


Hypothalamus
Pituitary
gland



Thalamus
Hypothalamus
Optic chiasm
Infundibulum
Pituitary gland

The Pituitary Gland

- Secretes nine major hormones
- Attached to the hypothalamus by the infundibulum (stalk)
- Two basic divisions of the pituitary gland
  - Adenohypophysis (anterior lobe)
  - Neurohypophysis (posterior lobe)

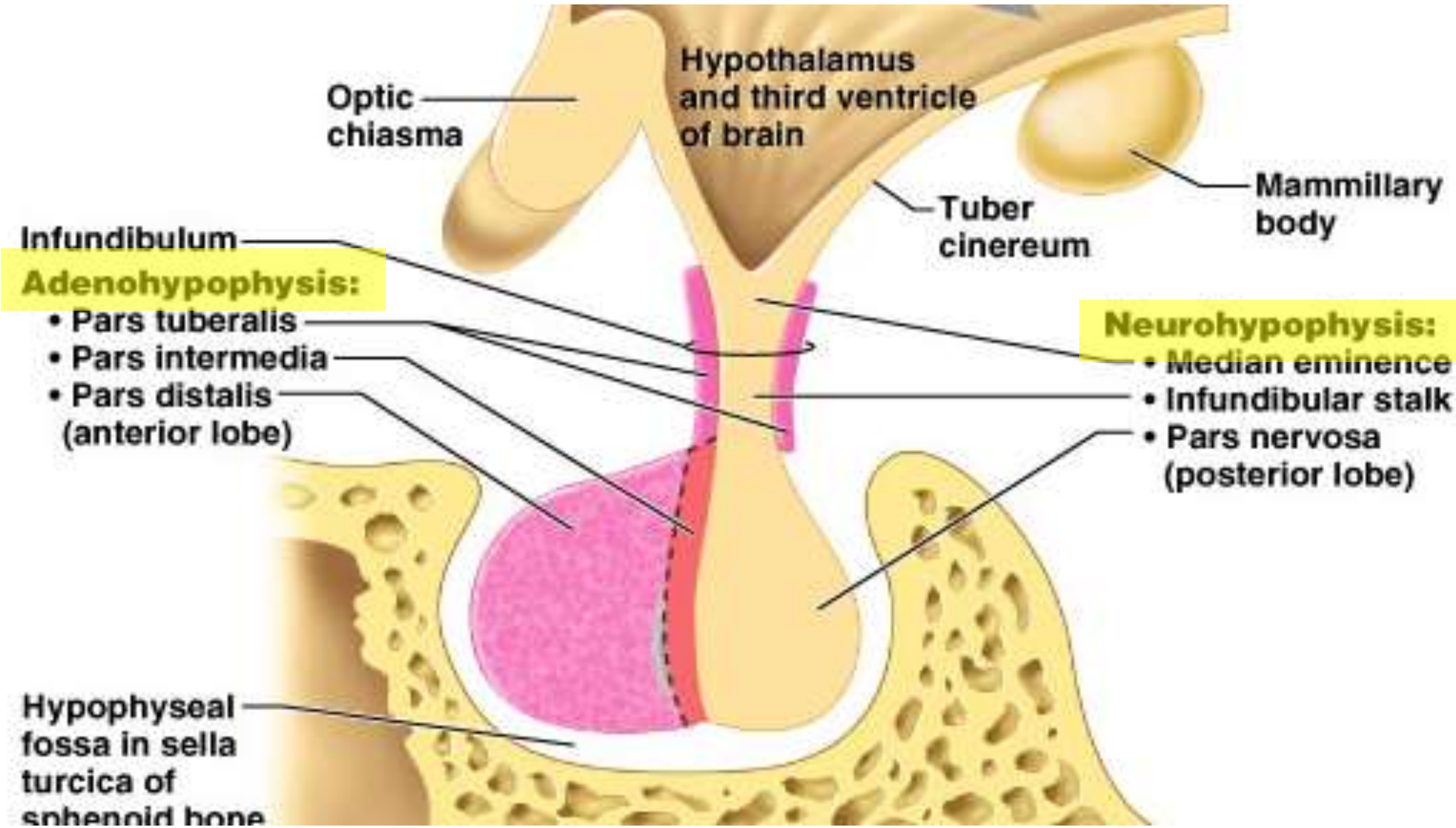


Figure 25.3a-c

The Adenohypophysis

- Growth hormone (GH)
  - Causes the body to grow
- Prolactin (PRL)
  - Stimulates lactation (milk production) in females
  - Stimulates lacrimation (desire to cry)
  - Decreased in adolescent males so it decreases desire to cry
- Thyroid Stimulating Hormone (TSH)
  - Causes the thyroid gland to release thyroid hormone

The Adenhypophysis

- Adrenocorticotropic hormone (ACTH)
  - Acts on adrenal cortex to stimulate the release of cortisol
  - Helps people cope with stress
- Melanocyte-stimulating hormone (MSH)
  - Darkens skin pigmentation
  - Increases during pregnancy
- Follicle-stimulating hormone (FSH)
  - Present in males and females, affects both
  - Stimulates maturation of sex cells
- Luteinizing hormone (LH)
  - Induces ovulation in females
  - Induces testosterone in males

Study Tip to remember the hormones secreted by the anterior pituitary gland

- “Melons grow and produce through late fall” stands for the hormones made in the anterior pituitary.

- **Melanocyte stimulating hormone (MSH)**
- **Growth Hormone (GH)**
- **Adrenal corticotropic Hormone (ACTH)**
- **Prolactin (PRL)**
- **Thyroid stimulating hormone (TSH)**
- **Luteinizing Hormone (LH)**
- **Follicle stimulating Hormone (FSH)**

The Neurohypophysis

- This is a continuation of the brain; cell bodies of special neurons in the hypothalamus have axons which go to the neurohypophysis and synapse on capillaries there. Instead of releasing neurotransmitter, they release hormones.
- Oxytocin
  - Childbirth contractions
- Antidiuretic hormone (ADH)
  - Signals kidneys to increase water reabsorption

Figure 25.6

The Neurohypophysis

The neurohypophysis is a continuation of the brain

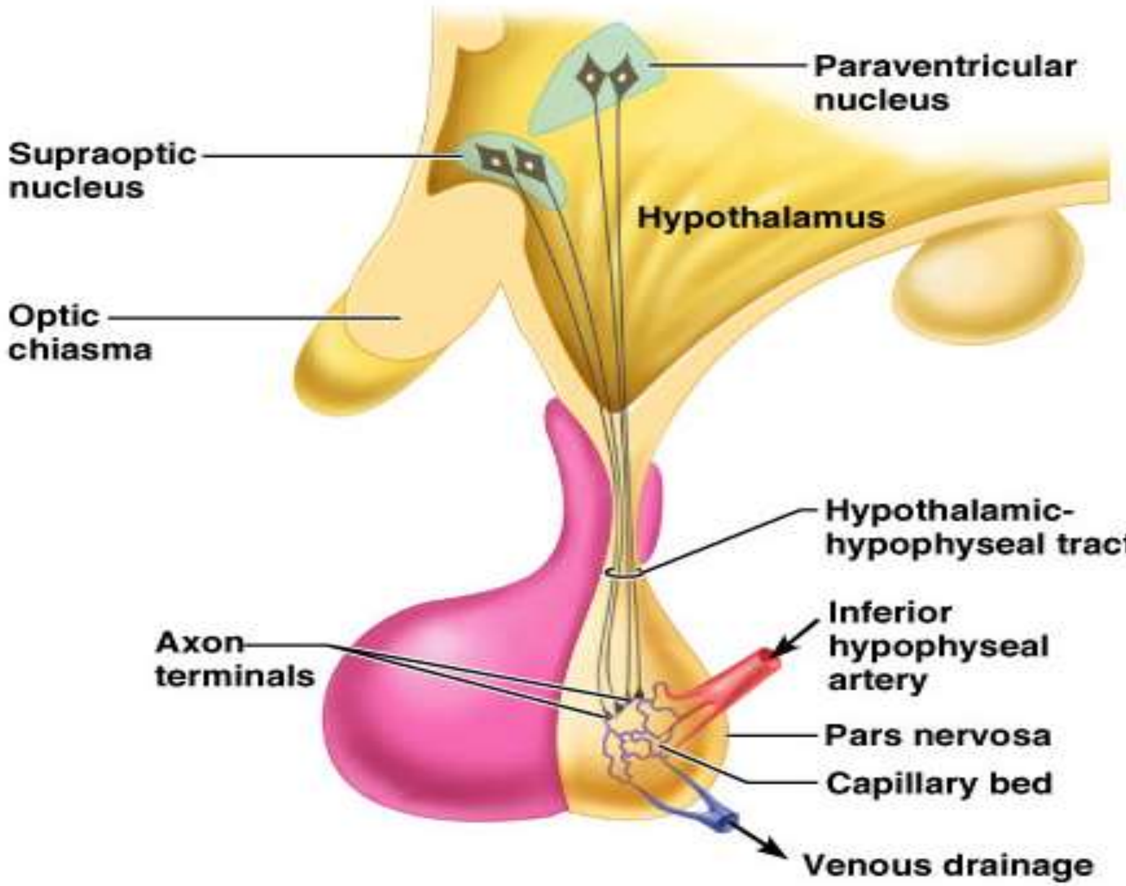
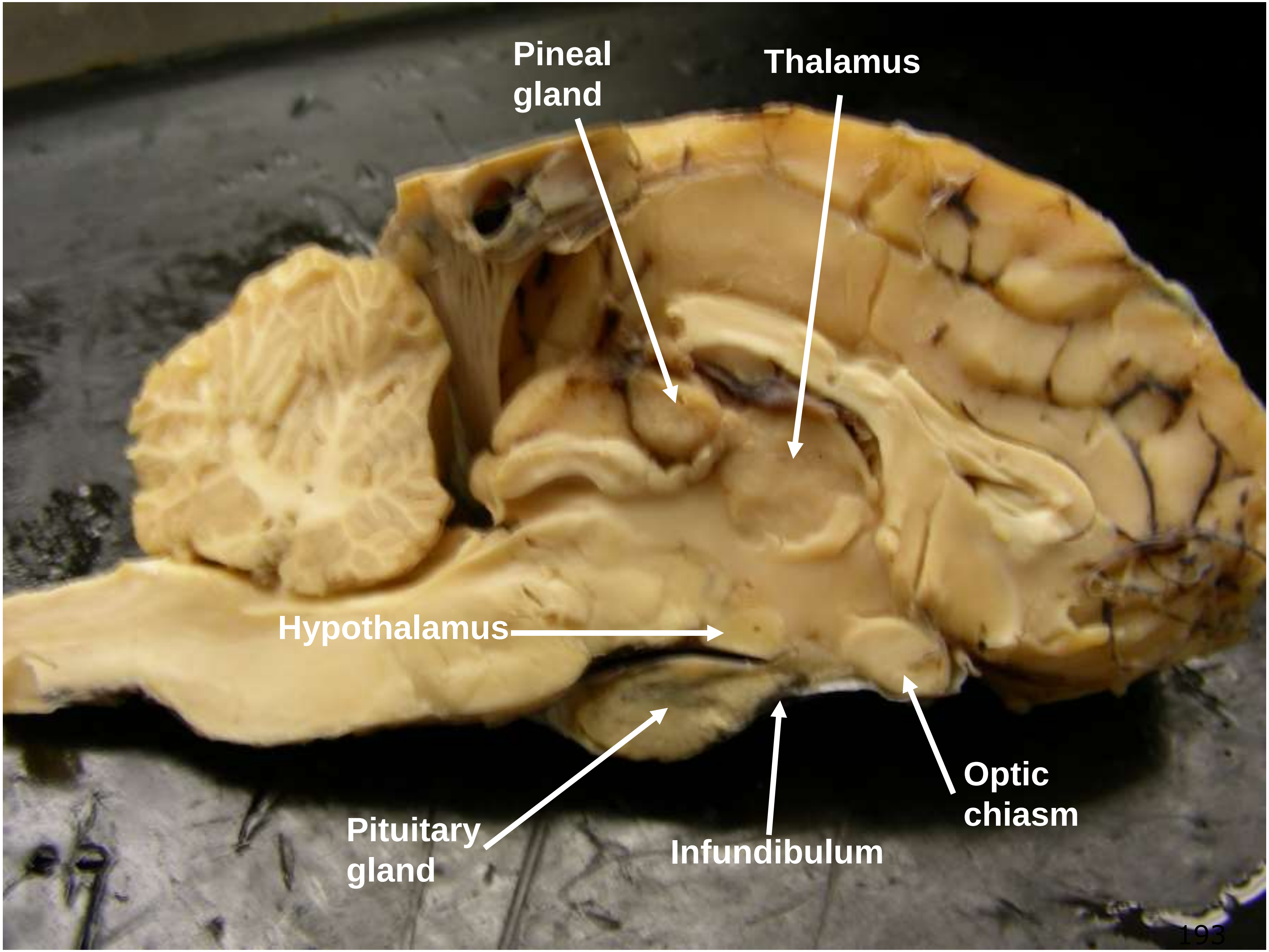


Figure 25.6

HYPOTHALAMUS

- Some people say the pituitary gland is the master gland because it controls the many other endocrine glands, but the hypothalamus controls the pituitary gland, so really, maybe the hypothalamus is the Master Gland.


Pineal gland
Thalamus
Hypothalamus
Pituitary gland
Infundibulum
Optic chiasm

Hypothalamus Regulation

- The hypothalamus produces hormones which affect the pituitary, for example:
- Thyroid Stimulating Hormone Releasing Hormone (TSH-RH)
  - Causes adenohypophysis to secrete TSH
  - TSH affects thyroid gland to secrete TH
- Thyroid Stimulating Hormone Inhibiting Hormone (TSH-IH)
  - Causes adenohypophysis to stop secreting TSH so thyroid gland stops secreting thyroid hormone
- The hypothalamus affects the pituitary gland, and that's about it.

Some Hypothalamus Hormones

- Growth Hormone Releasing Hormone (GH-RH)
- Prolactin Releasing Hormone (PRL-RH)
- Thyroid Stimulating Hormone Releasing Hormone (TSH-RH)
- Adrenocorticotropic Hormone Releasing Hormone (ACTH-RH)
- Melanocyte Stimulating Hormone Releasing Hormone (MSH-RH)
- Follicle Stimulating Hormone Releasing Hormone (FSH-RH)
- Luteinizing Hormone Releasing Hormone (LH-RH)

More Hypothalamus Hormones

- Growth Hormone Inhibiting Hormone (GH-IH)
- Prolactin Inhibiting Hormone (PRL-IH)
- Thyroid Stimulating Hormone Inhibiting Hormone (TSH-IH)
- Adrenocorticotropic Hormone Inhibiting Hormone (ACTH-IH)
- Melanocyte Stimulating Hormone Inhibiting Hormone (MSH-IH)
- Follicle Stimulating Hormone Inhibiting Hormone (FSH-IH)
- Luteinizing Hormone Inhibiting Hormone (LH-IH)

Pituitary Disorders

- Diabetes insipidus
  - Not enough ADH (anti-diuretic hormone; a diuretic takes out excess fluid from the body)
  - Lack ADH make a person urinates frequently (polyuria), so they are thirsty and drink a lot of water (polydipsia). But the level of glucose in the blood is normal.
  - The synthetic form of ADH is vasopressin (a medicine)

Pituitary Disorders

- Hypersecretion of GH in children
  - Gigantism (overall growth)
- Hypersecretion of GH in adults
  - Acromegaly: enlarged hands and feet, and big chin, nose, and forehead
- Hyposecretion of GH
  - Pituitary dwarfism
  - Proportions are normal, overall size is small

GROWTH HORMONE

- GH needs for thyroid hormone (TH) to be present. GH stimulates all cells to increase protein synthesis, fat utilization, and gluconeogenesis.
- **Gigantism** is the result of excess GH during pre-puberty and **acromegaly** is the result of excess GH after growth plates closed.
- The genetic determination of a person's height has multiple genes involved, so parents might be tall and have smaller children. There are no rules to predict it. A child may also be small due to a defect in the placenta, blocking nutrients during development.

Gigantism

Robert Wadlow 8′11″

Sandy Allen 7′7″

Acromegaly


3 4 5 6 7 8 9 10 1 2 3 4 5 6 7 8 9 20 1 2 3 4 5 6


Supreme


SHAG
33
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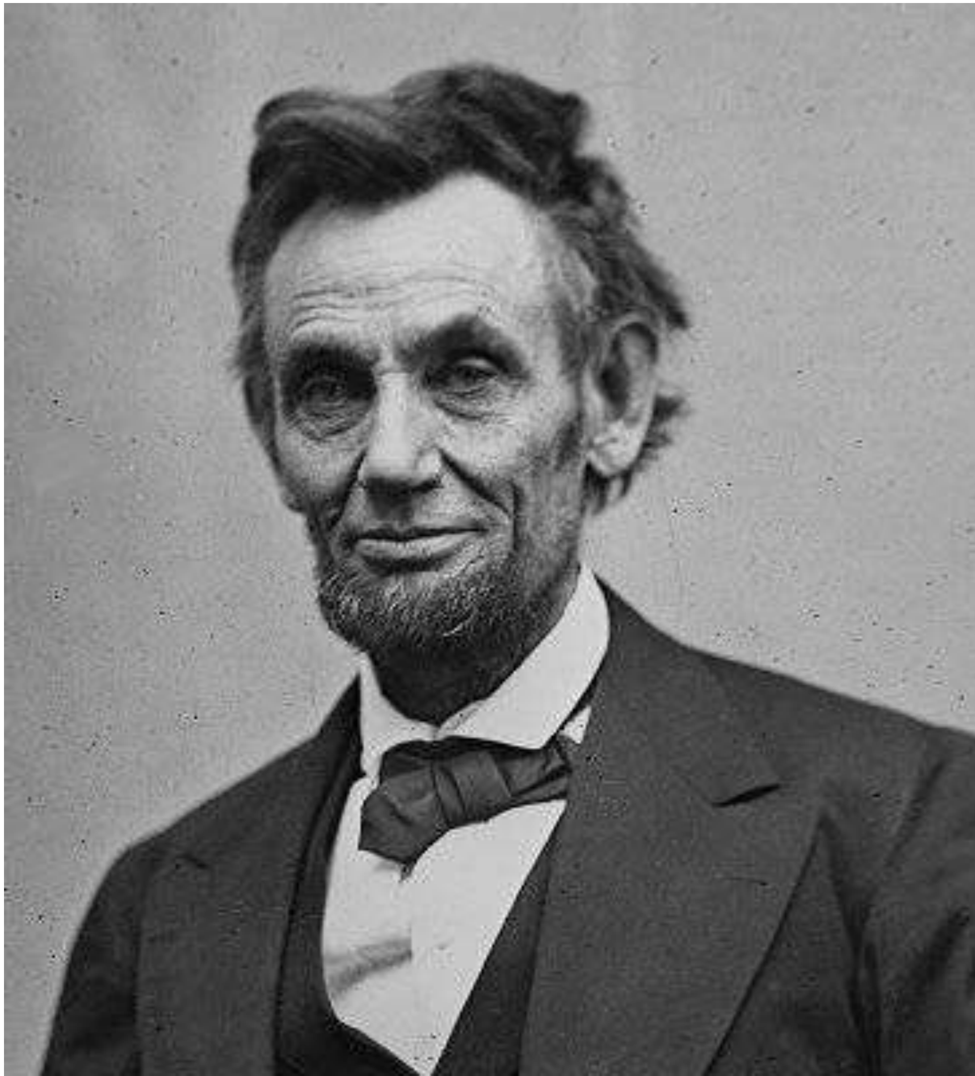
- 7 Feet 7 and 360 Pounds, With Bigger Feet Than Shaq's. Kenny George leads the nation in blocked shots per game.

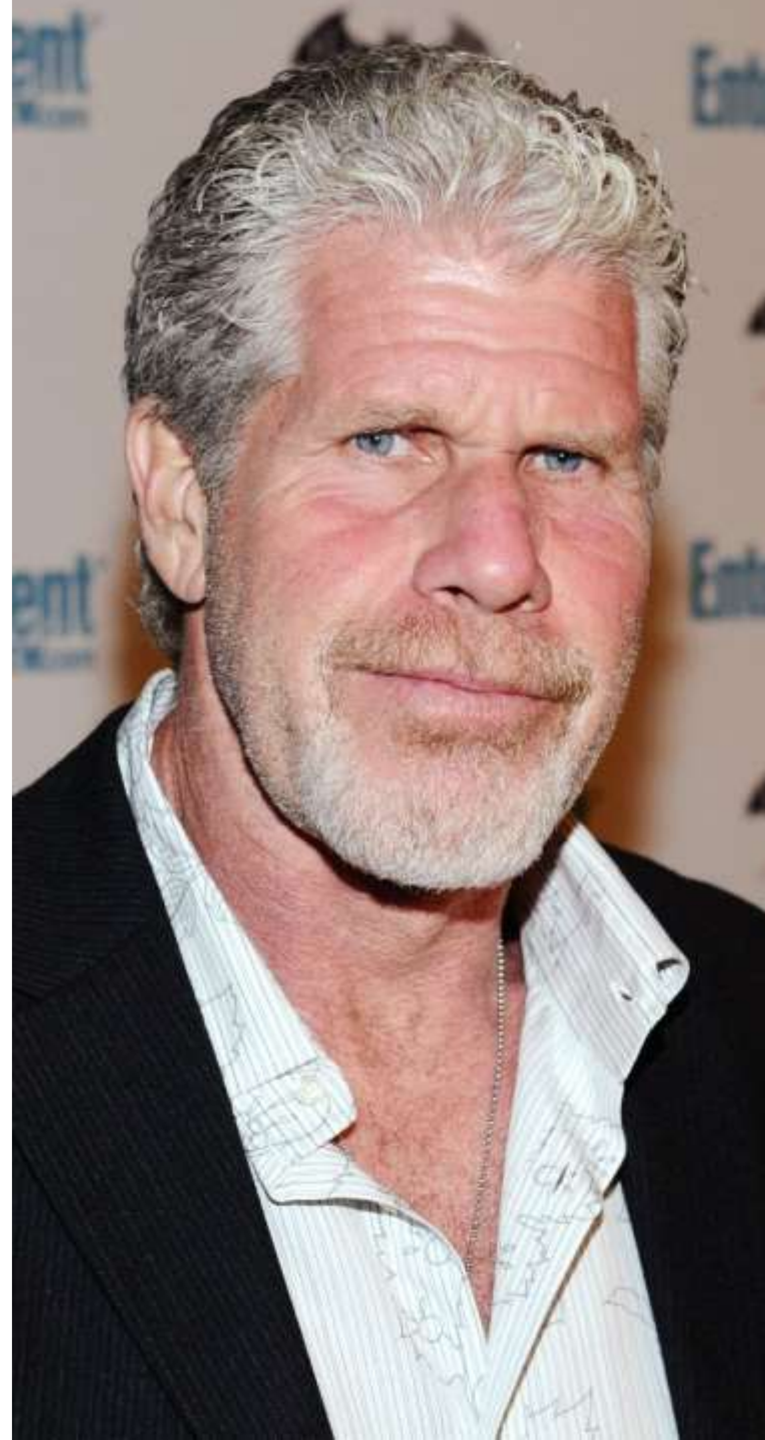
- Andre the Giant

- Lurch, Addams Family

- Abraham Lincoln

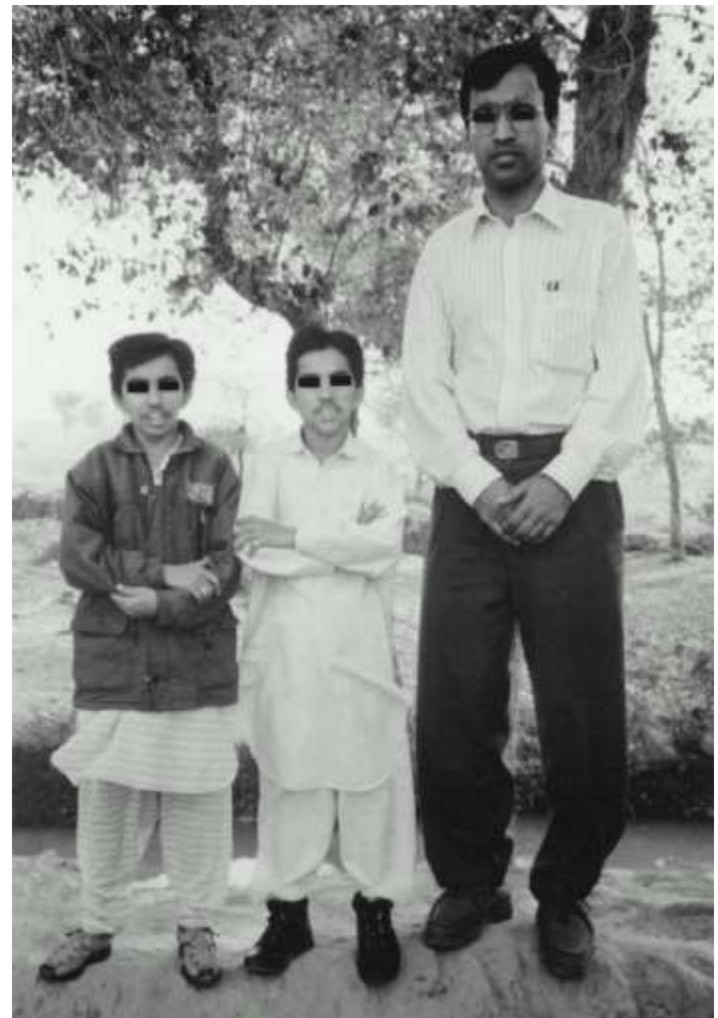


- Yao Ming



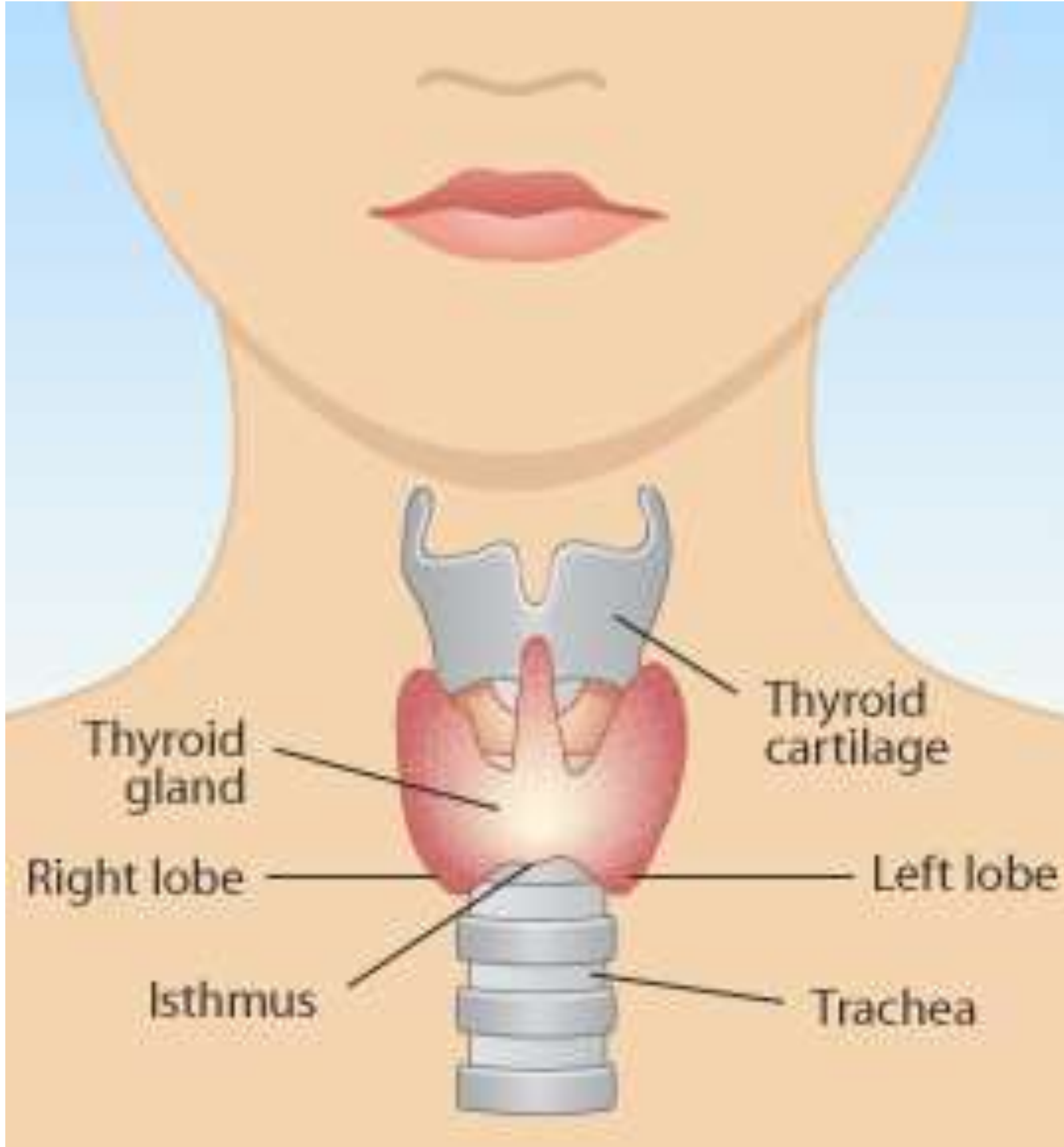
- Ron Perlman

Pituitary Dwarfism

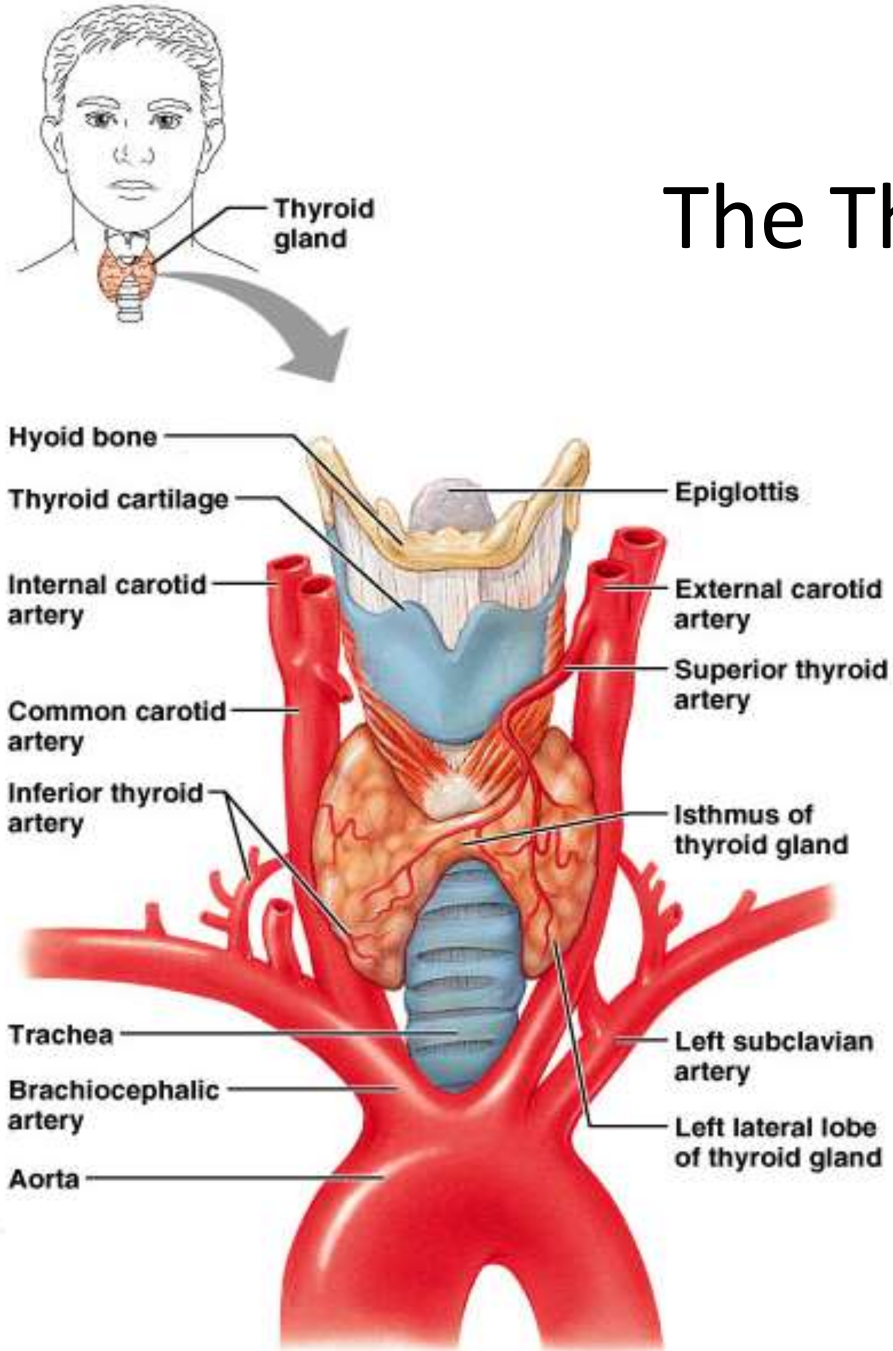


The Thyroid Gland

- Located in the anterior side of the neck, inferior to thyroid cartilage
- Largest pure endocrine gland
- Produces two hormones
  – Thyroid hormone (TH)
  – Calcitonin


Thyroid gland
Right lobe
Isthmus
Thyroid cartilage
Left lobe
Trachea

The Thyroid Gland



(a)

Figure 25.7a

The Thyroid Gland

- Thyroid hormone (TH)
  - Acted most cells of the body
  - Increases metabolic rate
  - Controlled by hormonal mechanism
  - Iodine is needed to make TH
- Calcitonin
  - Lowers blood calcium levels; secreted in children
  - Slows osteoclasts to allow osteoblasts to deposit bone in the skeleton.

(Vitamin D is synthesized and secreted by the dermis)

Thyroid Gland

- **The functional unit of the thyroid gland is the thyroid follicle**. The cells making up the perimeter of the follicle are called **follicular cells**. They make and secrete the light purple liquid within the follicle, called **colloid**. Colloid is water, filled with a lot of protein called **thyroglobulin**, which is made by the follicular cells. Since thyroglobulin is a protein, there is a gene that codes for it, so there can be genetic mutations that affect its production.
- **TSH is what stimulates the follicular cells to make thyroglobulin**. TSH also increases the size of the follicle to accommodate all this protein.

Thyroid Follicle with Thyroid Hormone

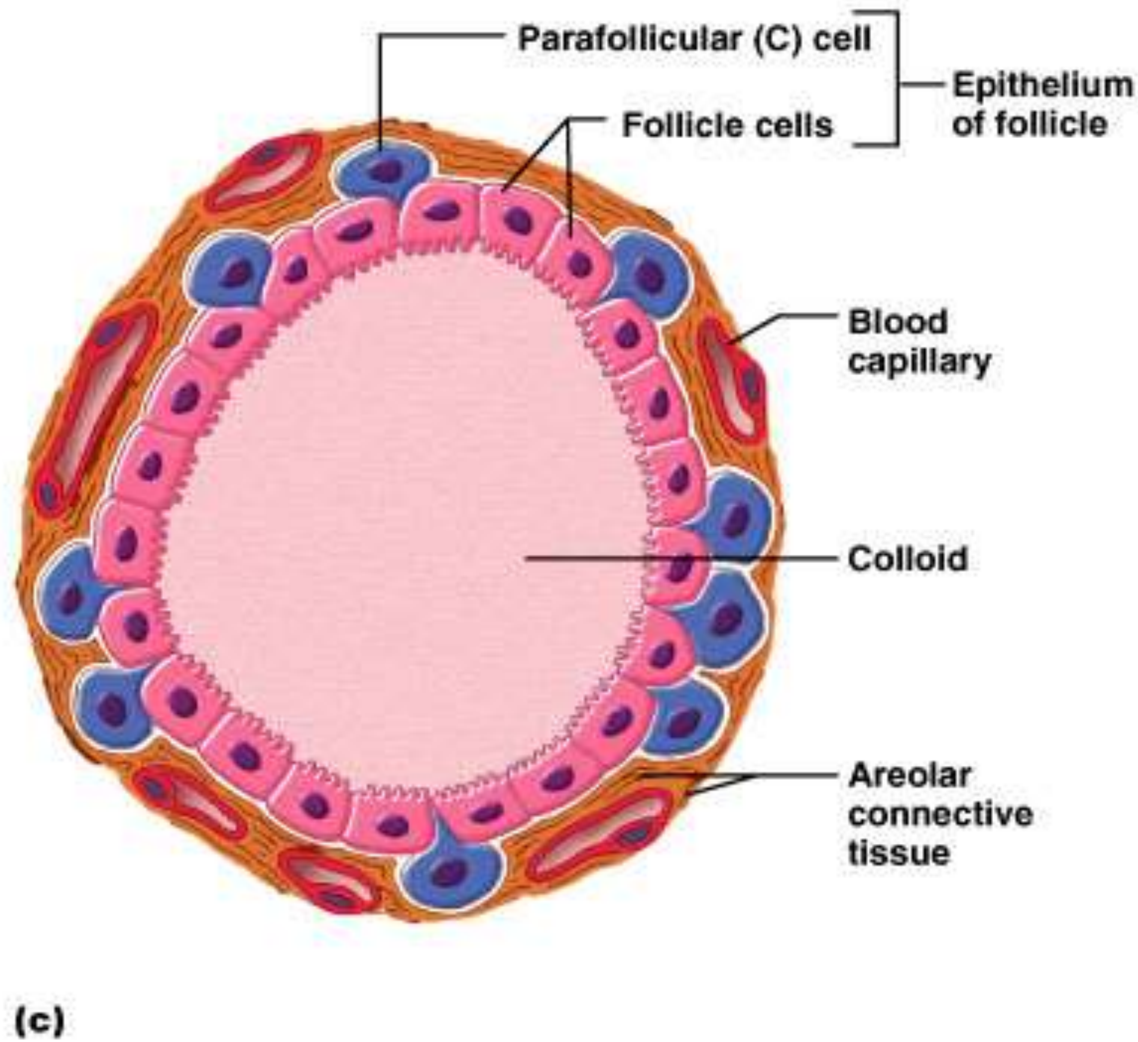
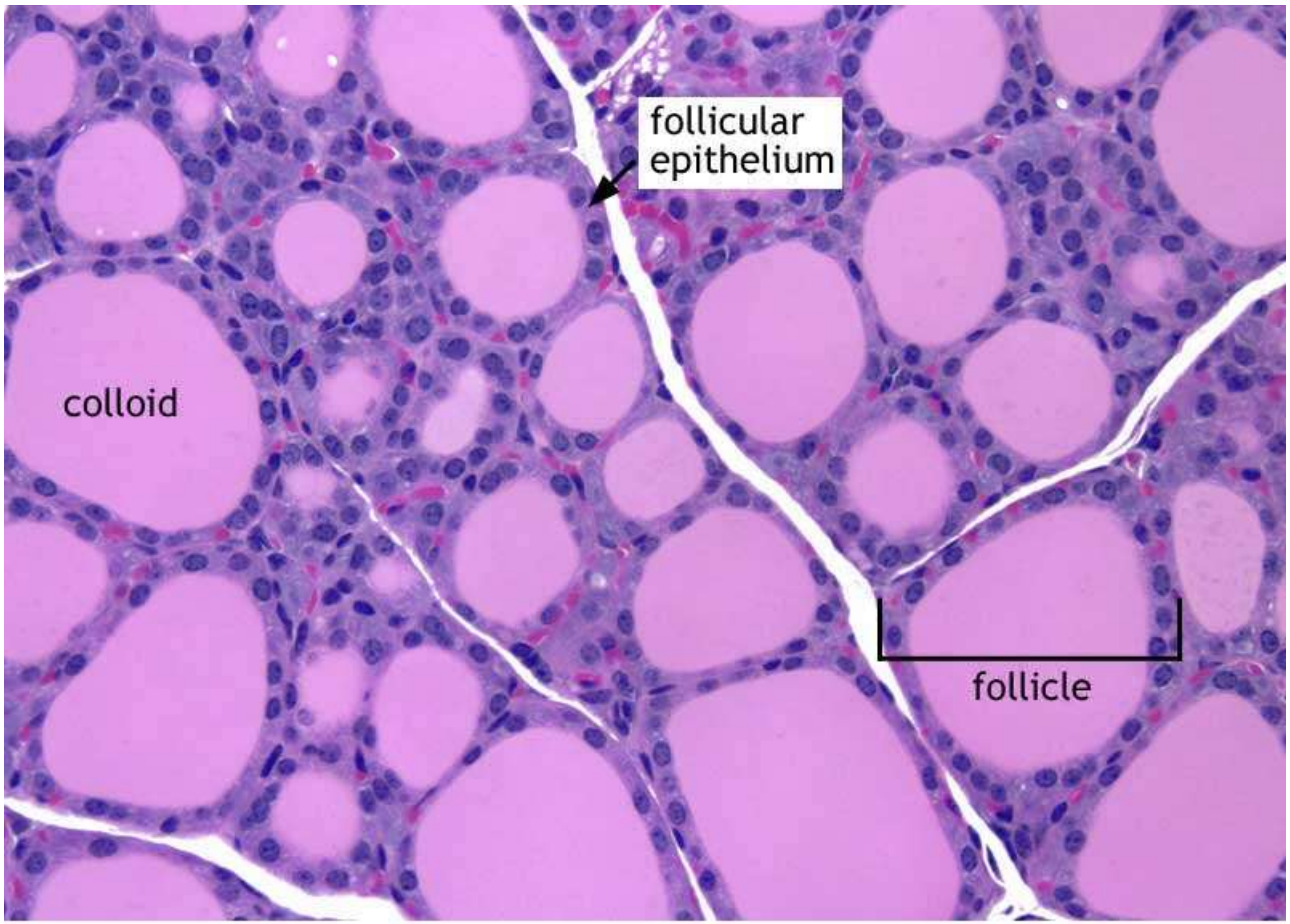


Figure 25.7c 21.8

Thyroid Gland



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Thyroglobulin

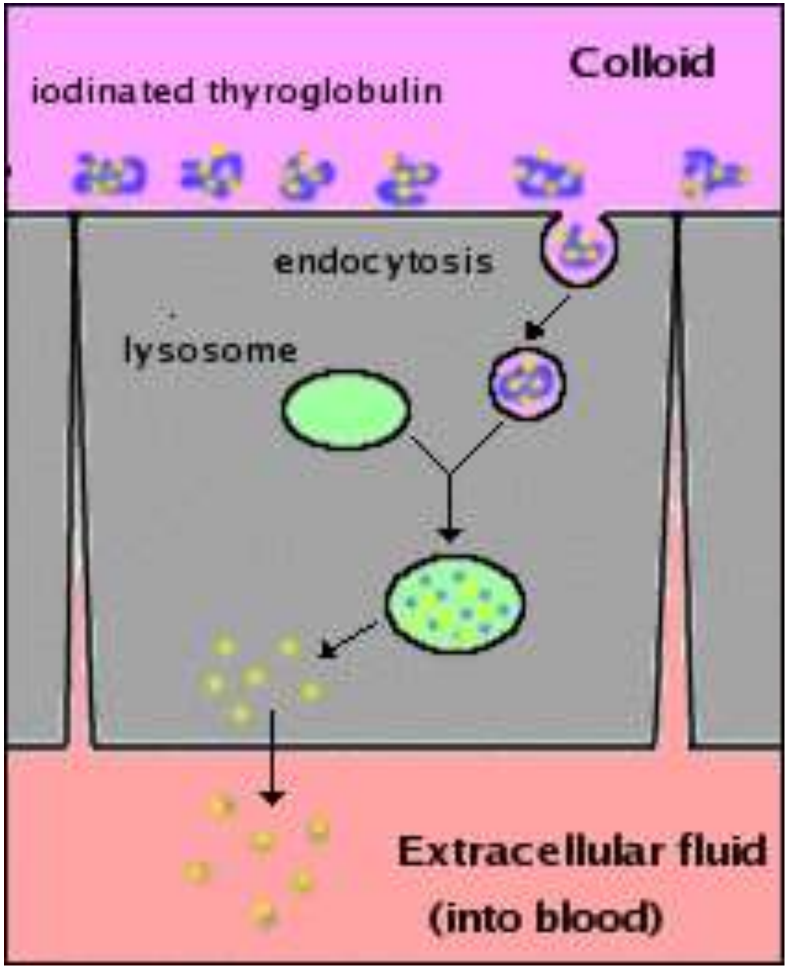
- When thyroglobulin is made, it is exocytosed (pushed out of the follicular cell) and stored outside of the cell, in the colloid of the follicle (like kids leaving the house through the patio door and jumping into the swimming pool in the back yard).
- As it moves across the cell membrane, a **peroxidase enzyme attaches iodine to the tyrosine** (an amino acid) portions of the thyroglobulin. This process is **iodination**.

Thyroglobulin

- The peroxidase enzyme is like a lifeguard putting safety floaters on the arms and legs on each kid as they leave the house, before the kids jump into the pool.
- The safety floaters are the iodine.

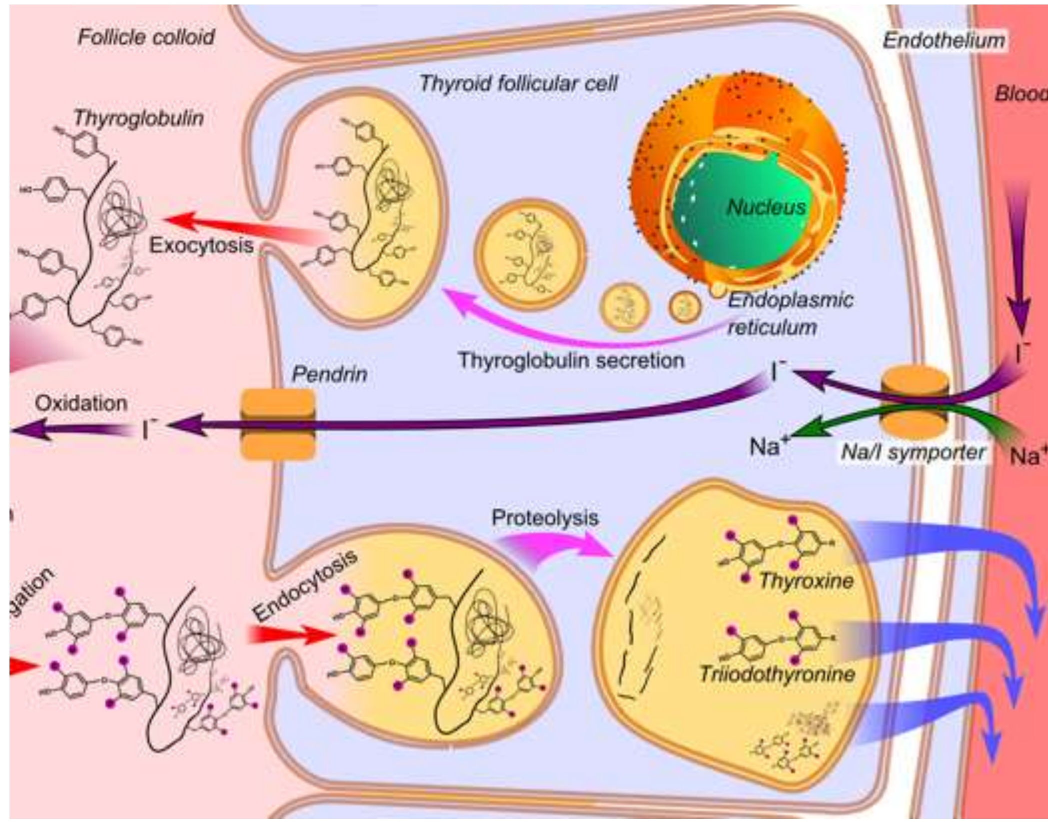
Thyroglobulin

- After TSH stimulation, the follicular cells endocytose the thyroglobulin (pulls it back into the follicular cells). Everyone get out of the pool and come inside! Here is where the story gets dark:
- Another enzyme comes along and chops up the long thyroglobulin protein into smaller pieces, each with some iodine on them.



Thyroglobin

- If a segment has two iodines, it is called **T2**.
- If there are 3 iodines attached, it is called **T3 (Triiodothyronine)**.
- If it has 4 iodines it is **T4 (thyroxine**).
- The T3 and T4 are then released into the bloodstream.
- Those thyroglobulin segments that have only 1-2 iodines are recycled for parts and are not released.

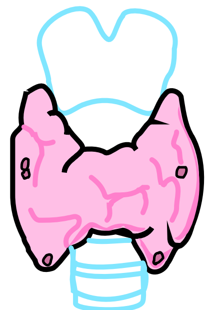


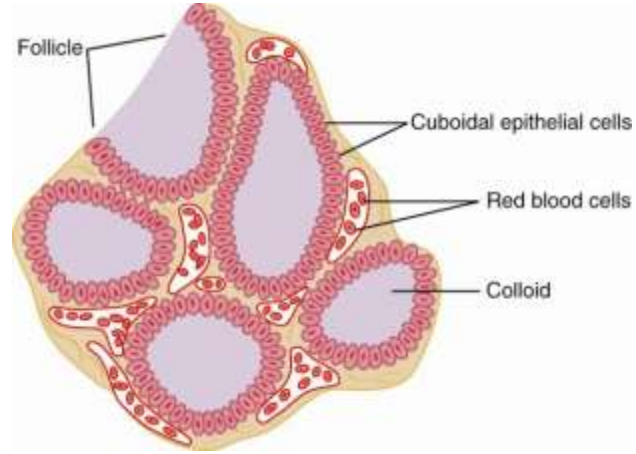
Thyroglobin

- **T4 is the most abundant form, but it is inert (inactive). T3 has robust activity in the cell**. So, T3 gets used first by the body cells. T4 takes longer to be ready; one iodine has to drop off. As T3 is used up, T4 is being converted to more T3.

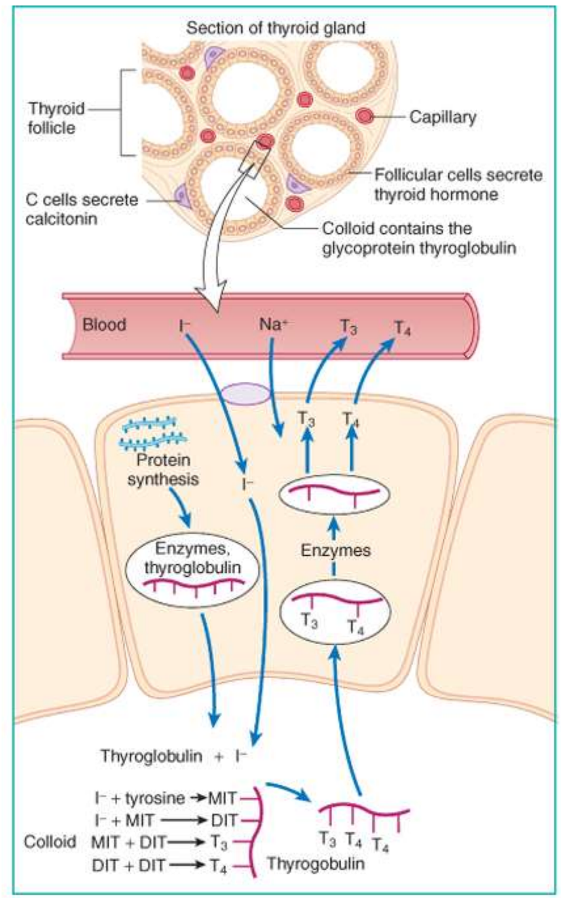
- To make thyroid hormone, you need iodine in your body. Iodized salt has enough to satisfy this need. Iodine is brought into the follicular cells, gene expression occurs, thyroglobulin is made. Without enough iodine's diet, thyroid hormone cannot be made, no matter how much TSH is present.

Thyroid Gland





- Thyroid follicles- hollow structures surrounded by follicular and parafollicular cells
- Follicular cells produce Thyroglobulin (TG)
- Building block of TH, chemically attaching I^- to tyrosine.
- In plasma, TH needs a "carrier molecule" or it will be cleared from body



Tyrosine: a bulky amino acid containing a large benzyl ring.

Thyroid Hormone Synthesis & Secretion

- Link two tyrosine aa's together and add iodine
- Thyroid hormone (TH) controls metabolic rate and protein synthesis
  - Thyroxine – T4 : (93%)
  - T3: triiodothyronine (7%); 4x as potent
    - Active form

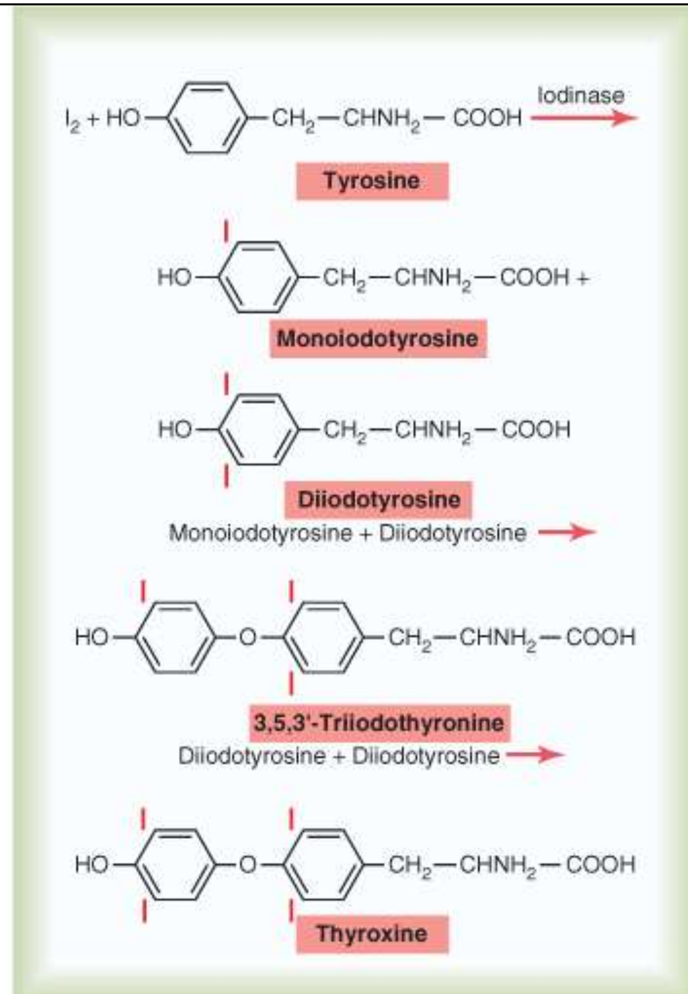


Figure 76-3 Chemistry of thyroxine and triiodothyronine formation.

TS Ratio

- The **TS ratio** is the amount of iodine in thyroid /iodine in serum.
- There are 30x more iodine ions in the thyroid gland than in the plasma.
- ATP is used to bring iodine into cells against its electrical gradient.

PTU

- People with hyperthyroidism can take a drug called PTU (Propylthiouracil), which inhibits TH production by blocking the peroxidase enzyme that joins the iodine to the tyrosine. It results in lower thyroid hormone levels.

•TG is booted out of the cell (exocytosis) and stored inside the hollow chamber of the follicle. “Colloid”

•When follicular cells receive signal to secrete (from TSH made in the pituitary), they take up TG (endocytosis), cleave off the TH from TG, and secrete it into blood (exocytosis.)

PTU is an anti-thyroid drug which blocks the peroxidase process.

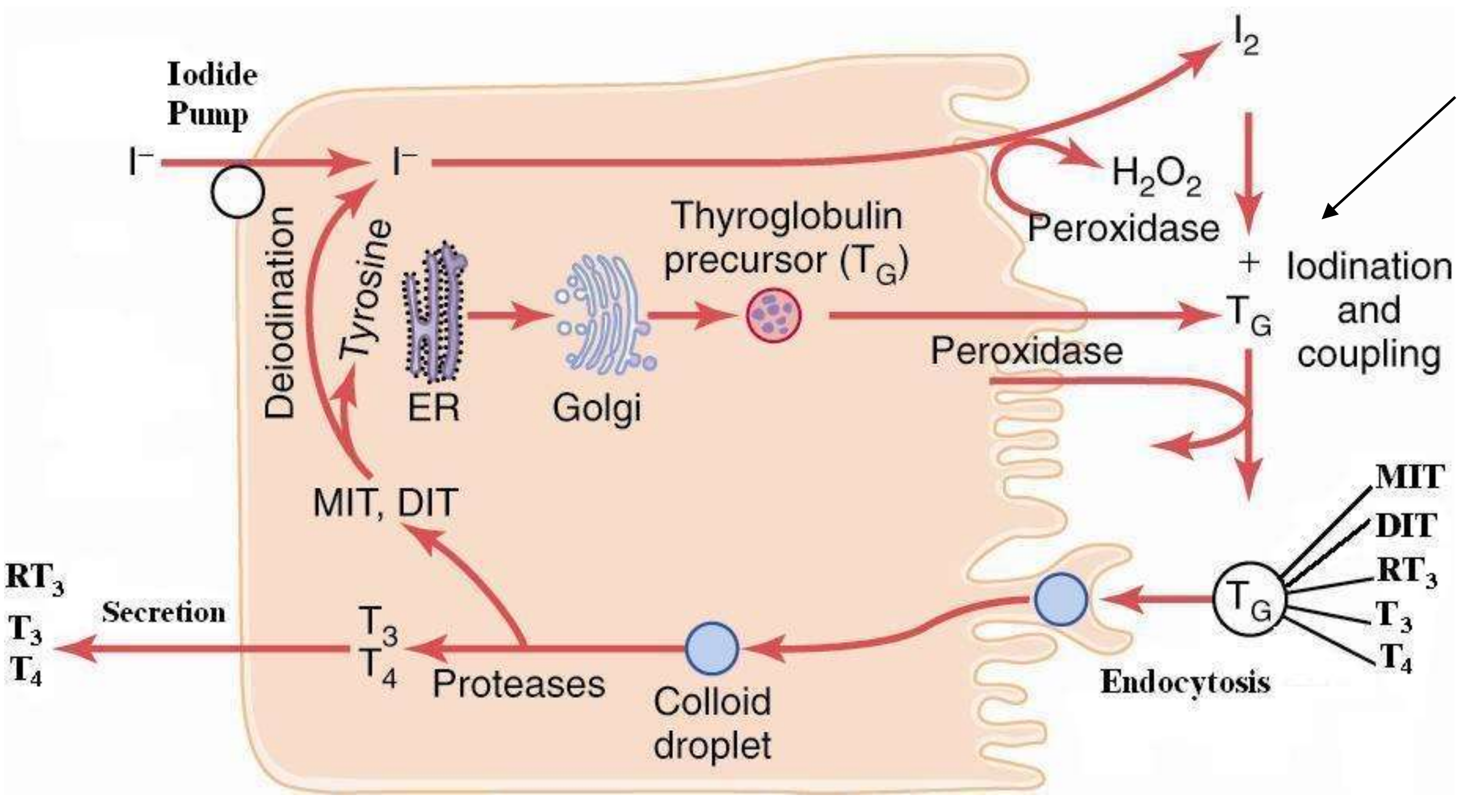


Figure 76-2; Guyton & Hall

What are the "actions" of TH?

Increases GI motility

Increases mental activity

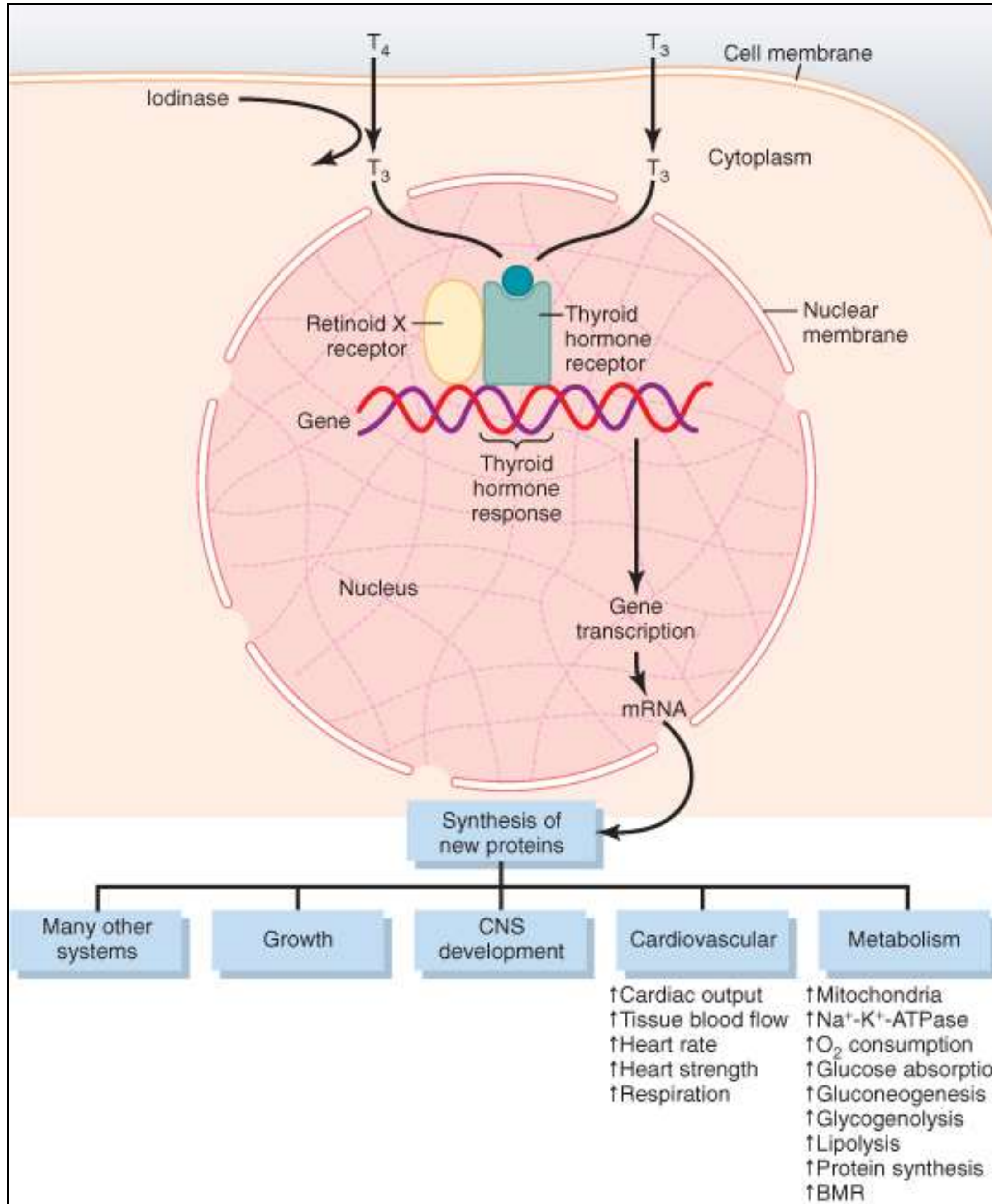
Increases endocrine activity

Promotes growth and brain development in the fetus and young children

Stimulates fat metabolism

Excites CNS

Causes sleep difficulty



Thyroid Hormone Effects

- All cells respond to thyroid hormone, increasing their metabolic rate (heart speeds up, beats with greater force, more nutrients are used, etc).
- Too much thyroid hormone is **hyperthyroidism**; these people are thin and active.
- When levels of TH are too low, it is called **hypothyroidism**; these people are overweight, move slowly, have no energy.

Effects of Thyroid Hormone

- TH also stimulates neurons; the person feels more alert, observing their environment with more interest. When there is too much TH, they get muscles tremors and increased blood glucose levels (hyperglycemia).
- With not enough TH, they lose interest, become sluggish, they get low blood glucose levels (hypoglycemia).

Thyroid Hormone

- The major stimulus for the release of thyroid hormone is hormonal (TSH from the pituitary tells the thyroid gland that it needs to make more thyroid hormone).
- What happens when TSH is released? Every step in the process of making TH is increased: Follicular cells become larger, metabolism increases: increase in O2 use (especially in mitochondria), heat is generated.
- TSH causes stimulation of sympathetic (beta) receptors in the heart, causing increased force of contraction and increased heart rate.

Thyroid Hormone

- Thyroid hormone is partly made of iodine. Iodine is essential for the formation of thyroxin (T3). If a person doesn’t eat enough iodine, they can’t make thyroid hormone.
- The hypothalamus responds by putting out more TSH-RH.
- The pituitary will respond by releasing TSH.
- But the thyroid can’t respond by releasing TH if it does not have the iodine to make the hormone, so it the size of the follicle grows → gland grows → **GOITER**.

GOITER

- This is usually caused by shortage of iodine in the diet.
- That’s why salt is iodized.
- Iodine is only found in seafood, so if salt wasn’t iodized, a lot of people wouldn’t get enough iodine, and there would be a lot of goiters.
- There are more problems with the thyroid gland than any other organ.

Goiter

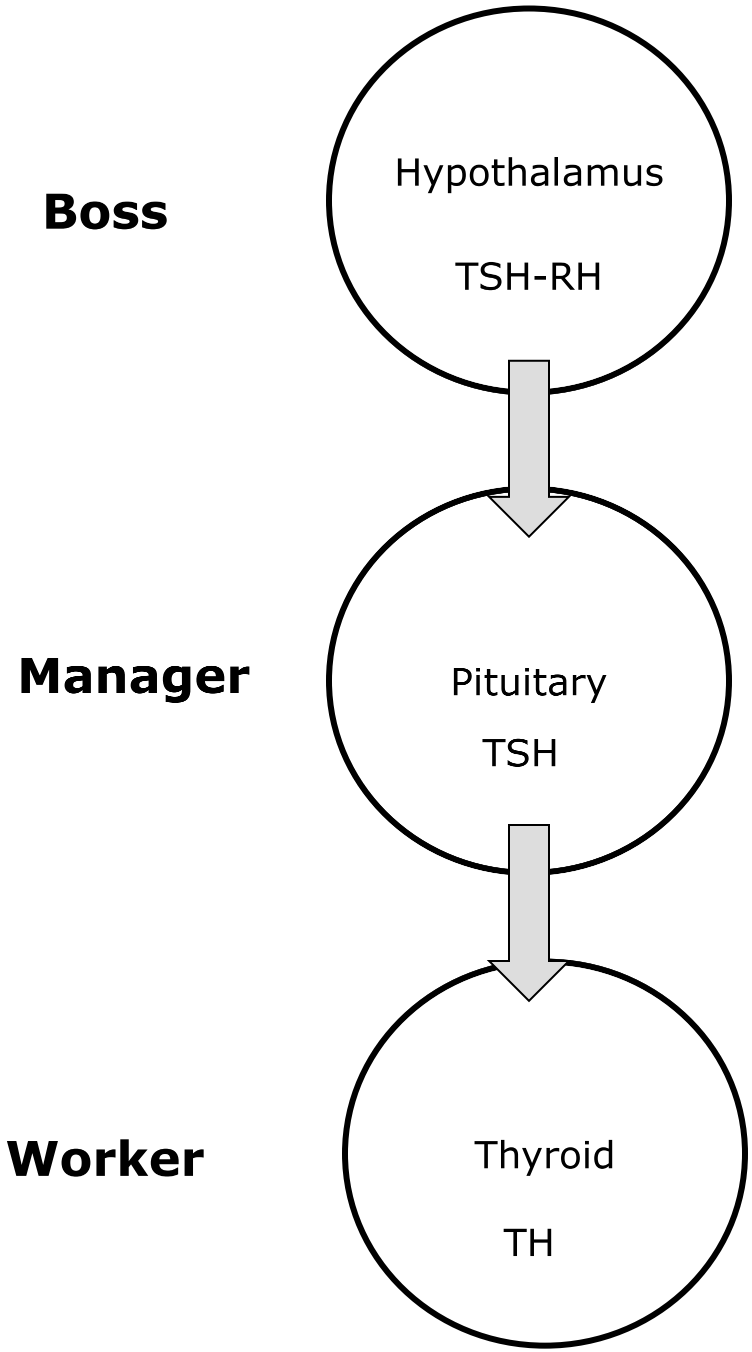
All you need is a pinch per day of salt. If you can't have salt, you can take iodine drops.

Role of Hypothalamus

- The hypothalamus is like the boss of a company; the pituitary gland is like the boss' manager, and the thyroid gland is the worker.
- The boss tells the manager to tell the worker to copy more papers.
- The manager tells the worker to copy more papers.
- The worker copies more papers.

Role of Hypothalamus

- The papers then go out to every cell in the body.
- Some of the papers land on the desk of the boss. When his desk is covered with papers, he tells the manager to stop the orders for more papers.
- If not enough papers are on his desk, he tells the manager to keep sending out the order for more papers.



This is what happens in the body:

Hypothalamus (the boss) makes TSH-RH (thyroid stimulating hormone releasing hormone)

Pituitary (the manager) makes TSH (thyroid stimulating hormone)

Thyroid gland (the worker) makes TH (thyroid hormone)

Thyroid Hormone

- When thyroid hormone is released, it will circulate throughout the body, causing an increase in metabolism in all of those cells.

- Some of the TH will bind to receptors in the hypothalamus, and then the hypothalamus knows there is enough TH, and it will stop releasing TSH-RH.
- Until the receptors in the hypothalamus are bound with the resulting thyroid hormone, the hypothalamus is not satisfied that there is enough thyroid hormone present.

What if the hypothalamus released its signal and the thyroid released too much hormone?

- The hypothalamus will stop secreting its releasing hormone. This is a negative feedback signal.
- When very few TH receptors are bound on the hypothalamus, it will keep releasing its hormone. When its thyroid receptors are saturated, will stop. This is still a hormonal mechanism of release, not humoral.

Positive and Negative Feedback

- The presence of thyroid hormone is what will stop the hypothalamus from wanting more.
- This is **negative feedback**, which is what most hormones have.
- The one hormone that uses **positive feedback** is luteinizing hormone (LH) in females.
- When LH is released, it stimulates the release of more LH, and more LH, until it reaches a maximum level, then negative feedback kicks in. LH is the hormone that causes fluid to rush into the follicle surrounding a woman's egg, and when enough fluid rushes in, the follicle pops like a balloon, releasing the egg during her monthly ovulation.
- Two other examples of positive feedback are blood clotting and oxytocin (childbirth contractions).

Problems with Thyroid

- Goiter
  - Deficit of iodine in the diet
- Hyperthyroidism
  - Graves' Disease - hyperthyroidism is caused by an autoimmune disorder.
  - Leads to nervousness, weight loss, sweating, and rapid heart rate.
- Hypothyroidism
  - Decreases metabolism, causes obesity

HYPERTHYROIDISM
(Most commonly caused by Graves Disease, which is an autoimmune disease)

- Signs include thinness, eyes that stick out like a bug (exophthalmoses).

There are two ways to treat Hyperthyroidism

- The patient can has the thyroid oblated (killed off) by drinking radioactive iodine; it kills just thyroid tissue. As metabolic rate slows, gains weight again. They can't be around people for 5 days, and they set off Geiger counters for months afterwards. Then start on artificial thyroxin, need to figure out what their set point is for normal.

- The other way (not so good) is to have the thyroid gland surgically removed. However, the parathyroid glands are often damaged or removed during this surgery. They often intentionally leave some thyroid tissue behind, in hopes of leaving enough parathyroid glands there. If too many of the parathyroid glands are removed, calcium levels go down, can go into cardiac arrest. Now the patient has to have two hormones replaced.

Hypothyroidism

This can be caused by

- Hashimoto’s thyroiditis (autoimmune)
- Iodine deficiency
- Tumor
- Defective enzyme in thyroid.

Hypothyroidism

- – Hashimoto's Thyroiditis - adult hypothyroidism
  - Antibodies attack and destroy thyroid tissue
  - Low metabolic rate and weight gain are common symptoms
  - Myxedema: non-pitting edema associated with hypothyroidism
- Cretinism – hypothyroidism in children
  - Short, disproportionate body, thick tongue and mental retardation

Hypothyroidism
Before and After Treatment

Cretinism (diminished mental ability)

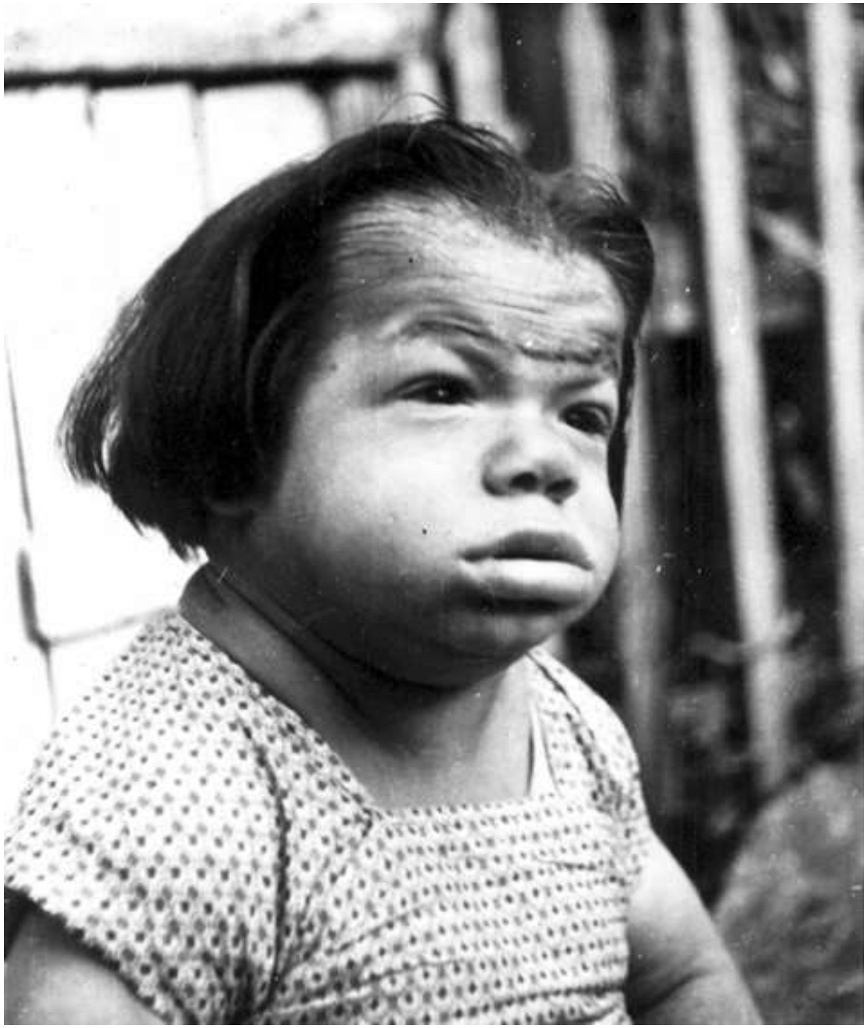
- This term describes babies whose MOTHER had the lack of iodine.
- Baby now cannot get iodine, and the baby will have reduced growth and intellectual ability.
- Once it is born and gets a healthy diet, it still won't go back to normal because TH is necessary for proper myelination and synaptic formation.

Congenital Hypothyroidism

- **Congenital hypothyroidism** is the term for a baby whose thyroid gland is not working correctly (not secreting enough TH). The problem is only with baby, not with the mom.
- Congenital hypothyroidism and cretin babies have similar symptoms. Child will stay tiny because GH does not work without TH.

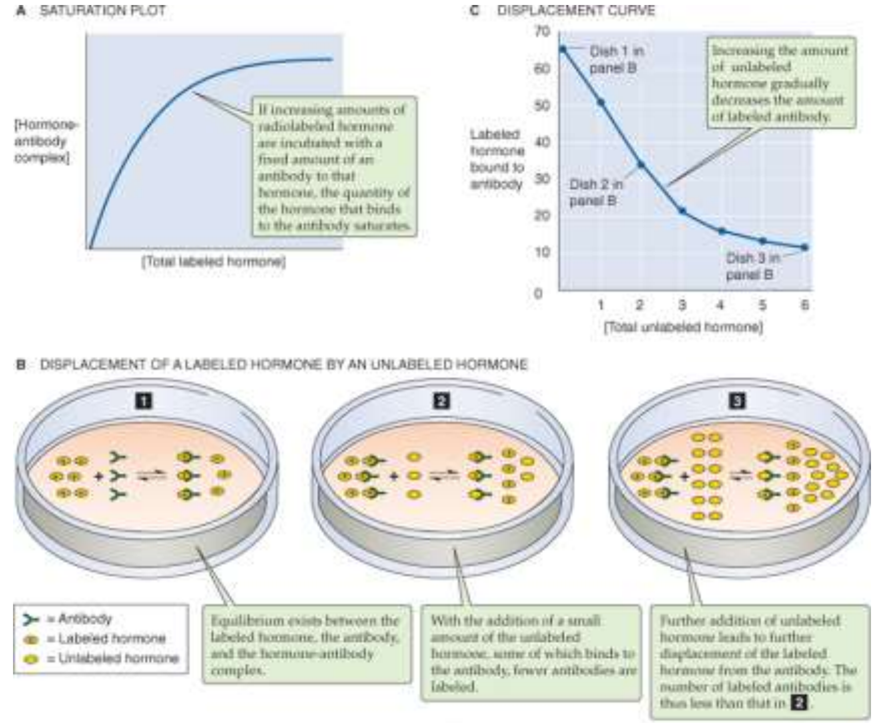
- Know the difference between cretinism and congenital hypothyroidism.

Cretinism

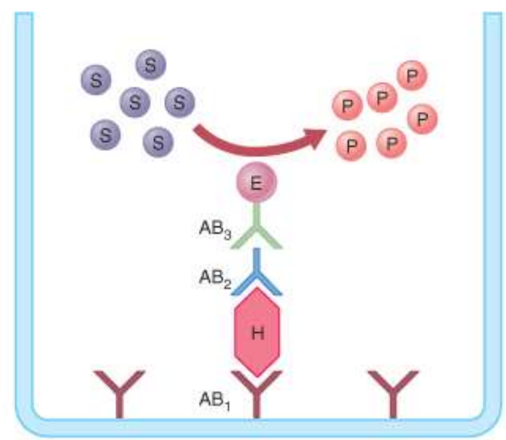


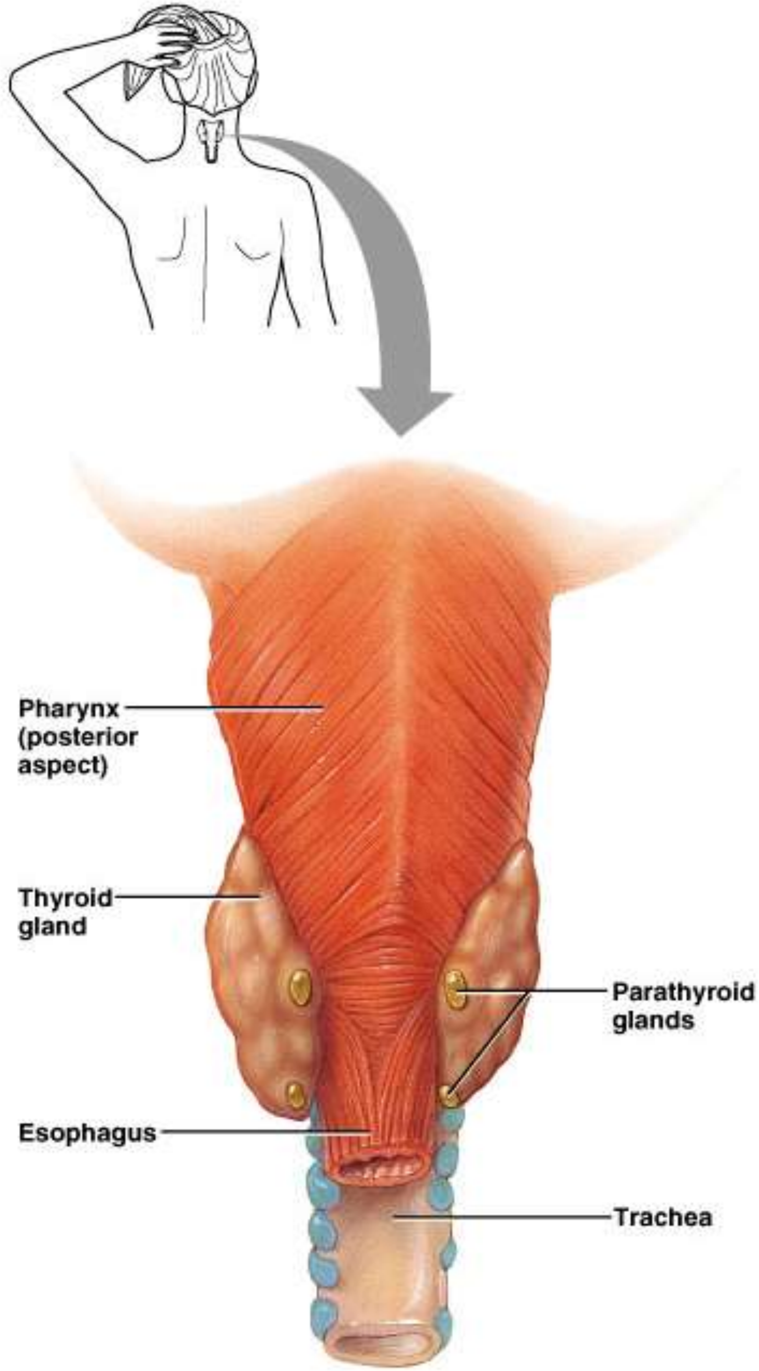
- Patients with Hashimoto's hypothyroidism often are deficient in vitamin D.
- http://drclark.typepad.com/dr_david_clark/2012/07/hashimotos-autoimmune-thyroiditis-and-vitamin-d-deficiency.html

Diagnosing the etiology (cause) of hypo/hyperthyroidism



- Methods of measuring plasma concentration of hormones:
  - RIA (radioimmunoassay)
  - ELISA (enzyme-linked immunosorbent assay)
- Sample a small amount of patient's blood; sent to lab
- Concentration is determined, recorded as Pico molar concentration





(a)

Parathyroid Glands

- Four glands imbedded on the posterior surface of the thyroid gland

Figure 25.4a

Parathyroid Glands

- Parathyroid hormone (PTH)
    - Increases blood concentration of Ca^{2+}
- **There are three ways that the parathyroid gland raises blood calcium levels**

1) Stimulates osteoclasts to move bone calcium from the skeleton to the bloodstream

2) Stimulates the kidneys to stop excreting calcium

3) Stimulates the intestines to absorb more calcium from diet

    - Activates vitamin D which increases calcium uptake by intestines

PARATHYROID GLANDS

- **The antagonist of parathyroid hormone is calcitonin,** which is produced in the thyroid gland, and stimulates osteoblasts to take calcium from the blood and deposit it in bone.
- Parathyroid hormone is released by a **humeral mechanism.**
- If blood calcium levels are low, parathyroid hormone is released.
- If blood calcium levels are high, parathyroid hormone stops being released.

THYMUS GLAND

- Hormones produced by this organ stimulate the production of T cells (a type of white blood cell).
- This gland is mostly active in children under the age of three.

The Adrenal Glands

- Located on the superior surface of the kidneys
- Two endocrine glands in one
  - Adrenal medulla – a knot of nervous tissue within the gland. Secretes catecholamines.
  - Adrenal cortex – bulk of the adrenal gland. Secretes most of the steroid hormones.

Adrenal glands MECHANISM OF RELEASE

- Both adrenal glands together weigh only one gram!
- They use neuronal, hormonal, and humoral mechanisms.
- **The adrenal medulla uses a neuronal mechanism, since it is an extension of the nervous system.** If the cells there are detached, they will differentiate into a neuron!
- The **adrenal cortex uses a hormonal mechanism, except aldosterone uses a humoral mechanism.**

The Adrenal Gland

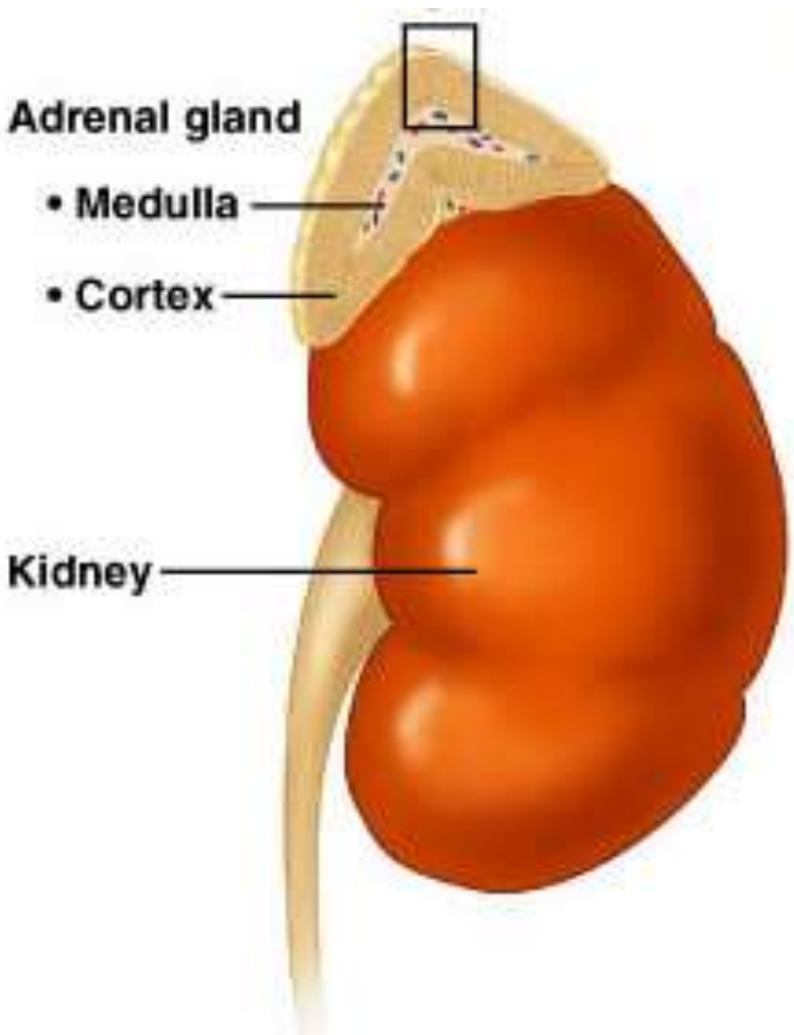


Figure 25.9a

The Adrenal Glands

- Located on the superior surface of the kidneys
- Two endocrine glands in one (different embryological origin)

– **ADRENAL MEDULLA – a knot of sympathetic nervous tissue**
  - **Secretes catecholamines (mostly epinephrine)**
    – Active in "fight, flight, and fright" response

– **ADRENAL CORTEX – bulk of the adrenal gland**
  - **Secretes aldosterone** (salt and water balance for blood pressure)
  - **Secretes androgens and estrogens** (sex hormones)
  - **Secretes cortisol** (anti-stress and anti-inflammation hormone)

The Adrenal Medulla

- Secretes catecholamines such as epinephrine and norepinephrine:

  ADRENALIN (AKA epinephrine "above the kidney"; Greek).

  This is the neurotransmitter for the sympathetic nervous system.

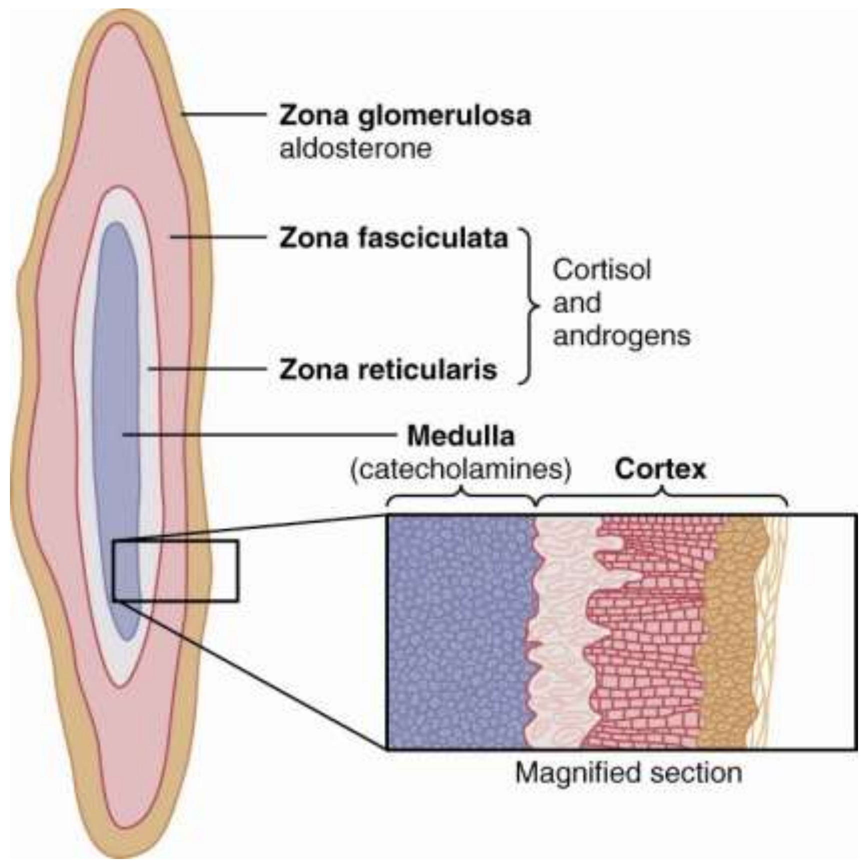
  The adrenal medulla also has sympathetic neurons which synapse on it, so when you are spooked, the neurons fire and stimulates the adrenal medulla to release more epinephrine to increase the effects of the sympathetic nervous system.

Adrenal Medulla

- The adrenal medulla releases **catecholamines (epinephrine and norepinephrine)**.
- These catecholamines are released when the sympathetic nervous system is activated ("fight or flight").
- When you run from a predator, is that when you want insulin to take glucose from blood? No, you want to keep it there so the brain can get the glucose. The brain needs to think of a way to escape, and thinking burns glucose.
- **Therefore, epinephrine is antagonistic to insulin**
- Cells that don't get the glucose during fight or flight break down fatty acids to get their ATP. These fatty acids will be taken to the liver for gluconeogenesis to elevate the depleted blood glucose levels. Glycogen will also be broken down to glucose to elevate the depleted blood glucose levels.
- Epinephrine has the same effect as the sympathetic nervous system:
  - Heart rate and force increases.
  - Digestion slows
  - respiratory passages open (bronchiole dilation)
  - BP goes up (from vasoconstriction in less-needed organs).

Adrenal Cortex layers

- The bulk of the adrenal gland is the adrenal cortex. It has layers, from superficial to deep: “GFR”
- **G = Zona glomerulosa: makes aldosterone**
- **F = Zona fasciculate**
- **R = Zona reticularis**
  - The zona fasciculate and zona reticularis both make sex hormones and cortisol
- (Don’t confuse this mnemonic with “GFR” in the kidney, which stands for glomerular filtration rate)

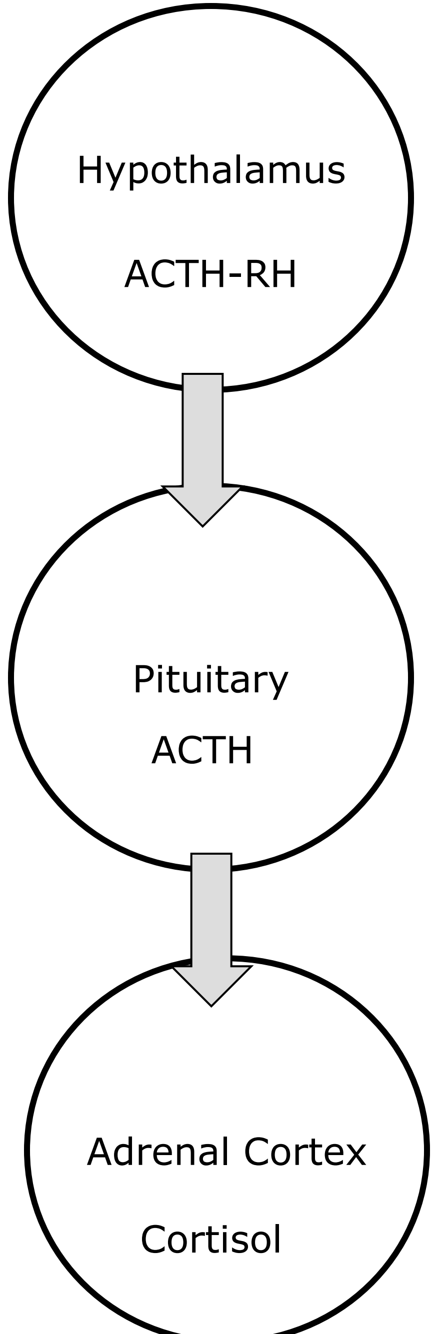


Adrenal Cortex

- Secretes a variety of hormones- **all are steroids** (steroids are made from cholesterol) and are grouped into three main categories:
  - Glucocorticoids
    - **Cortisol** – secreted in response to ACTH from the pituitary gland. Cortisol stimulates fat and protein catabolism to use for gluconeogenesis.
  - Mineralocorticoids
    - **Aldosterone** -Sodium/water reabsorbed
  - Androgens and Estrogens
    - **Male sex hormones (Androgens)**
    - **Female sex hormones (estrogen)**

The Adrenal Cortex

- **CORTISOL** helps the body deal with stressful situations like fasting, anxiety, trauma, and infection. It keeps the blood protein and glucose levels high enough to support the brain's activities. When the brain perceives a stressful situation, the hypothalamus makes the pituitary to secrete ACTH, which travels to the adrenal gland and signals it to release cortisol to most of the cells of the body. It is also known as hydrocortisone, which decreases inflammation.
- **ALDOSTERONE** increases blood volume during hemorrhage or drop in blood pressure. It causes kidney to reabsorb more sodium; water follows with it, so the blood volume increases.
- **SEX HORMONES** for the opposite sex: Males produce estrogen here, and females produce testosterone.



Cortisol: Hormonal Mechanism

- ACTH-RH is released by the hypothalamus.
- ACTH is released by pituitary.
- Cortisol (also called corticotropic hormone or CT).
- Cortisol affects almost all cells in body.

- Note: When ACTH plus cholesterol is present, you can take cortisol and turn it into aldosterone if you need to.
  - It does not do this unless the blood pressure is too low, because aldosterone is under a **humeral** mechanism (turned on by high **blood** levels of potassium or A2).

Glucocorticoids (cortisol)

- Glucocorticoids (GC) are a class of steroid hormones that bind to the glucocorticoid receptor (GR), which is present in almost every cell in the body.
- The name glucocorticoid (glucose + cortex + steroid) derives from their role in the raising glucose levels, their synthesis in the adrenal cortex, and their steroidal structure. They suppress the immune system (they are anti-inflammatory).
- Cortisol (also known as hydrocortisone) is one of the most important glucocorticoids.
- Others are prednisone, prednisolone, dexamethasone, and triamcinolone, which are also commonly used medicines for anti-inflammation.

Effects of Increased Glucocorticoids

- Cortisol is called an anti-stress hormone because it does several things:
- Stimulates **protein and fat catabolism (breakdown)**
  - The breakdown products are then taken to the liver for **gluconeogenesis** in the liver
- **Inhibits glucose uptake by the body but not the brain**
- **It elevates blood glucose (diabetogenic effect)**
- **It suppresses the immune response**
  - That means it is an **anti-inflammatory agent**
- It is prescribed as a medicine to suppress inflammation and the immune system.

Cortisol

- **Cortisol (also known as corticosterol and also known as hydrocortisone)**
- The hypothalamus releases ACTH-RH, pituitary releases ACTH, adrenal gland releases cortisol. The adrenal gland also can release androgens, estrogens, and aldosterone. All of those might be released if there is excess ACTH.
- **Excess androgens do not affect males, but females might develop more masculine features.**
- **Excess estrogens do not affect females, but males might develop more feminine features.**

- What is “stress” that causes cortisol production? Stress can be emotional or physical. Examples of physical stress can range from fighting an infection to having a minor injury that needs to remodel tissue.
- Cortisol tells tissues to stop using glucose (except brain), and to break down fatty acids instead, in order to get their energy.
- Cortisol also tells the skeletal muscle to start breaking down, and to release the free amino acids into bloodstream.
- The liver takes in these free amino acids and fatty acids and converts them into new glucose molecules that you did not acquire from your food. Since these are new glucose molecules being formed, this process is called **gluconeogenesis** (“generation of new glucose”).
- The new glucose molecules are released back into the blood (blood glucose levels rise) so the other tissues can have some energy.

Prednisone

- If a person has a lot of cortisol or prednisone in the body, blood sugar levels rise too much, and sugar spills out in the urine. Also there are some symptoms of diabetes. But you have some cortisol in you now to **help maintain normally elevated blood glucose levels between meals.**
- **In high doses only, exogenous (medicinal) prednisone may be given for asthma because it suppresses smooth muscle from constricting**, and bronchioles cannot close up.

Prednisone

- Prednisone makes you hungry. You also have a hard time sleeping because brain is stimulated. If the person abruptly stop taking prednisone, it can lead to same symptoms of Addison's disease (low cortisol levels). Their BP drops, blood glucose drops, can go to hospital. A person on high dose for 4 or more weeks must be tapered off.
- There are two ways to use prednisone: high dose, short duration (okay to stop abruptly)
- Lower dose, longer duration (need to wean off).

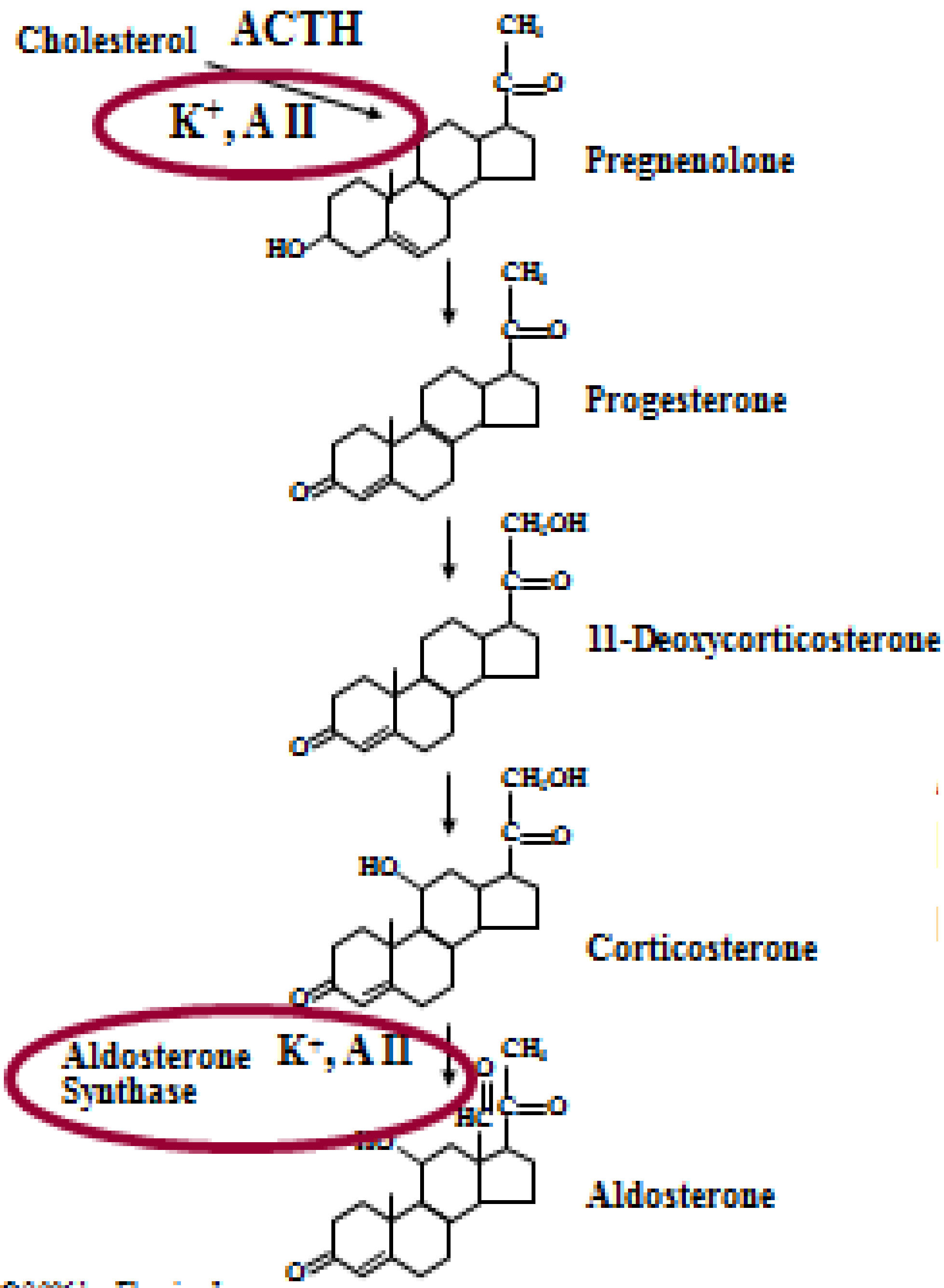
Aldosterone

Aldosterone (a mineralocorticoid) targets the cells of kidney, increases the amount of salt and water that is reabsorbed.

- It elevates blood pressure.
- The Z. Glomerulosa makes aldosterone. It has a **humeral release mechanism**. A few things trigger it, especially high potassium plasma levels and A2. That signals the kidneys to reabsorb sodium and water increases blood volume. How does this happen?

How Low BP is Raised: The renal-angiotensin system

- When **baroreceptors** detect low blood pressure, the kidney releases an enzyme called **renin.**
- In the meantime, **angiotensinogen** is made by the liver and released into the blood.
- Renin cuts angiotensinogen into angiotensin-1 (A1), which travels through blood to the pulmonary capillary bed, where cells have angiotensin converting enzyme (ACE) that cuts A1 into A2 (the active form).
  - Any word that ends in –ogen means it is a longer, inactive protein, called a zymogen.
  - To become activated, they need to be cut by an enzyme into a smaller segment.
- A2 then causes vasoconstriction of the peripheral blood vessels so the body's blood will pool up to the core organs.
- Also, these **high levels of A2** stimulates the adrenal cortex to make more **aldosterone**, and also stimulates the posterior pituitary gland to release **ADH**. These events will raise the blood pressure.
- When blood pressure is too high, the patient might be given an ACE inhibitor such as Captopril, or a renin inhibitor such as Aliskiren, or an A2 antagonist, such as Azilsartan.



Prednisone, cortisone, cortisol, and aldosterone are all similar in structure. One can be used to make the others.

If ACTH is demanding more cortisol, but the body cannot make enough, it may start making androgens/estrogens instead.

Sex (Male and Female) Hormones

- Male and Female sex hormones are present in both males and females; the pituitary gland affects these hormones in both sexes.
- Male sex hormones (androgens, such as testosterone) are made in the testes of males, and made in the adrenal gland of females.
- Female sex hormones are made in the ovary of females and in the adrenal gland of males.

Androgens

- Androgens are called male sex hormones because they cause male secondary sexual characteristics to develop, such as facial hair and low voice.
- The main steroid secreted by the adrenal gland that makes sex hormones is called DHEA.
- DHEA can be converted into **testosterone or estrogen**.
- A large amount of testosterone is made in the testes in males.
- A small amount of testosterone is made in adrenal cortex in males and females.
- If the adrenal cortex hyper-secretes testosterone and other androgens, it won't impact a male, because the testes make more than that already.
- However, in females, hypersecretion causes masculinization (such as facial hair and low voice).

Estrogen

- Estrogens are one of the female sex hormones because they cause female secondary sexual characteristics to develop, such as breasts.
- A large amount of estrogen is made in the ovaries in females.
- A small amount of estrogen is made by adrenal cortex in males and females.
- The androgen, DHEA, can be converted into **estrogen**.
- If the adrenal cortex hypersecretes estrogen, it won't impact a female's sex characteristics, because the ovaries make more than that already.
- However, in males, hypersecretion causes feminization (such as breast development).

Hypothalamus

↓ **ACTH-RH**

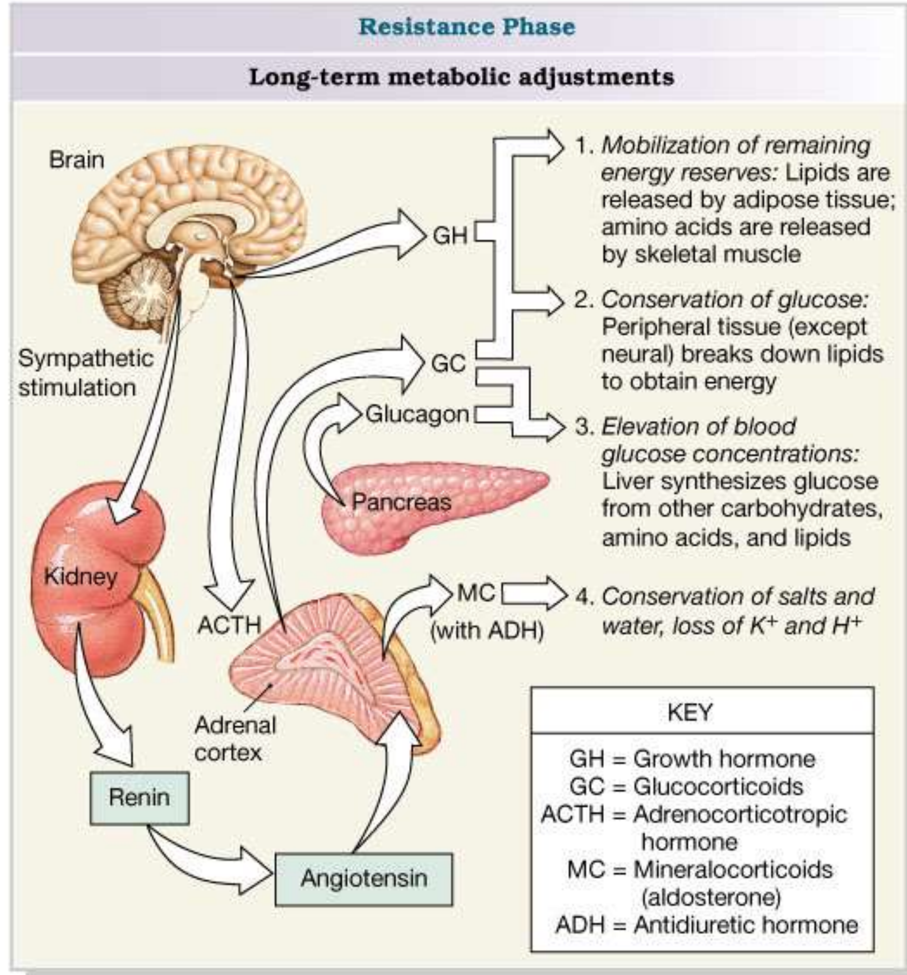
Pituitary Gland

↓ **ACTH**

Adrenal Cortex

↓

Cortisol



Adrenal Gland

- The adrenal cortex also makes aldosterone and sex hormones.

Adrenal Gland Disorders

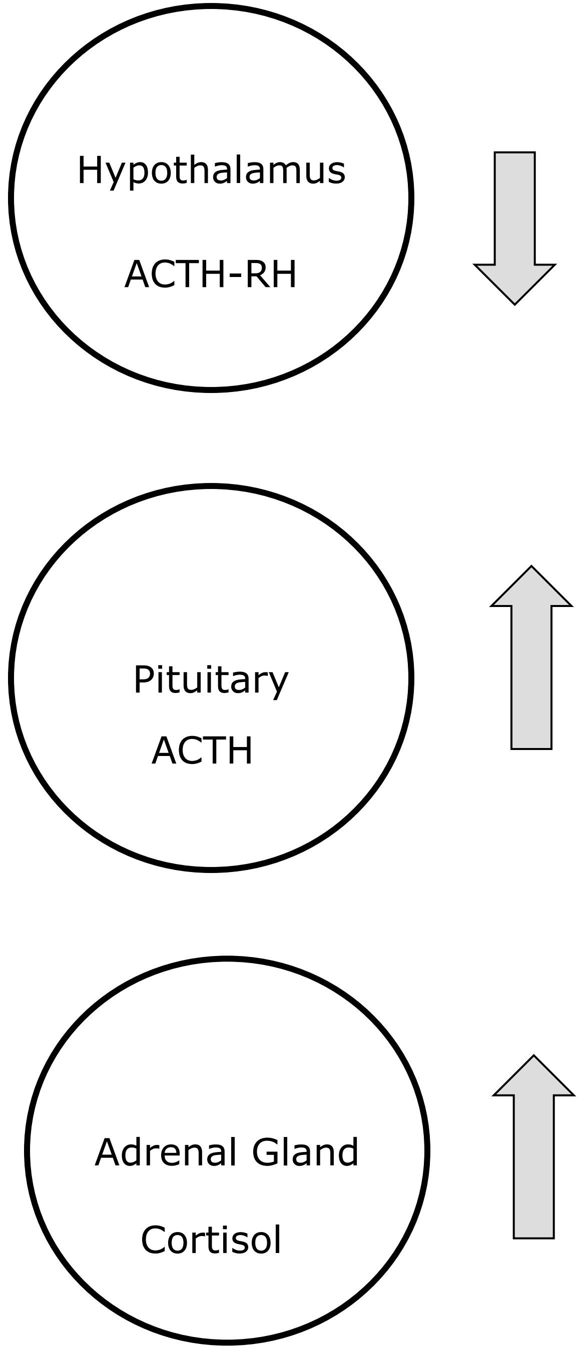
- Cushing’s syndrome/Disease
  - Hypersecretion of cortisol
  - High blood glucose
  - High blood pressure
  - Features of the opposite sex
  - Round “moon” face and “buffalo hump”
- Addison’s disease
  - Hyposecretion of cortisol
  - Low blood glucose
  - Low blood pressure results
  - Also get hyperpigmentation

CUSHING’S DISEASE

- Excess ACTH caused only by a **pituitary tumor**. Patient has **excess cortisol, high blood pressure, high blood glucose**, and too much aldosterone is produced. More salt and water is reabsorbed by the kidney, so the blood volume increases. In this disorder, the **hypothalamus (ACTH-RH) levels are low, the other hormone levels (ACTH, cortisol, androgens, and aldosterone) are high.**

ACTH Over-secreting pituitary tumor

CUSHING'S DISEASE

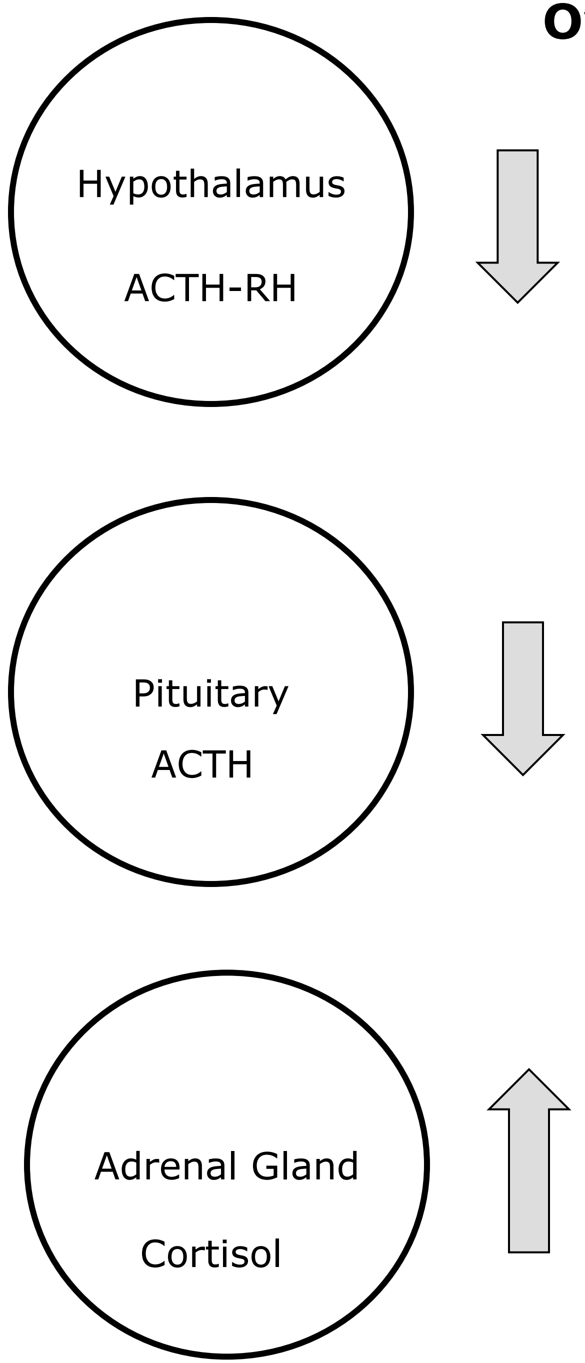


ACTH-RH **is low**

ACTH **is high**

Cortisol **is high (hyperadrenalism)**

Over-secreting adrenal tumor



ACTH-RH **will be low**

ACTH **levels will be low**.

Cortisol **will be high (hyperadrenalism)**

CUSHING'S SYNDROME

CUSHING’S SYNDROME (Andrenogenital Syndrome)

- Excess cortisol secretion, but not caused by the pituitary gland. It could be caused by primary hyperadrenalism (adrenal gland is not working right), an adrenal tumor, or even by a tumor in the lungs that releases ACTH (called an ectopic ACTH producing tumor).
- In Cushing’s Syndrome**, all adrenal cortical hormones (cortisol, androgens, and aldosterone) are elevated, but ACTH-RH and ACTH levels are low.**

Excessive Adrenal Hormones

- <u>Cushing's Disease-</u> pituitary tumor (excess ACTH)

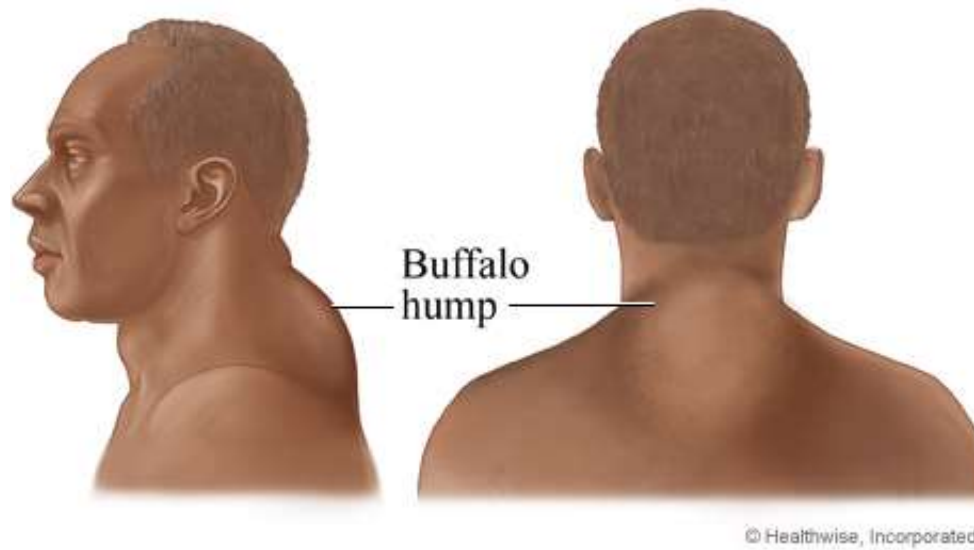
- <u>Cushing's Syndrome</u>
  - •Ectopic ACTH producing tumor (lungs)
  - •Iatrogenic (side-effect of some medical treatment)
  - •Primary hyperadrenalism
  - •Over-secreting adrenal tumor-, all adrenocortical hormones elevated; Andrenogenital syndrome
- Signs/symptoms: buffalo hump, moon face, muscle loss/weakness, thin skin with striae, hyperglycemia, immune suppression

Symptoms of Cushing's Disease and Cushing's Syndrome

•Fat deposition around waist (central obesity), scapula (**buffalo hump**), and "**moon" shaped face**. There is **muscle loss** and weakness (cortisol tells muscles to break down), thin skin with **striae**, (High levels of cortisol leads to destruction of collagen, get thin and striae on skin), **hyperglycemia, immune suppression**. Excessive amounts of adrenal stimulation causes release of male steroids, causing male secondary characteristics, but only in females. **Adult onset disease in females causes masculinization**, including facial hair, thicker jaw and skull.

Cushing's Syndrome

Michael Santoro and his twin sister, Paula, who had Cushing's Syndrome.



Cushing's Syndrome

Figure 1) Left and right *A 52-year-old human immunodeficiency virus (HIV)-1-infected man presented with a football-sized mass in the dorsal cervical area ('buffalo hump'). It had existed as a minor area of fullness for several years, but had dramatically increased in size over the preceding year, after the protease inhibitor indinavir was added to his antiviral regimen*

Central Obesity

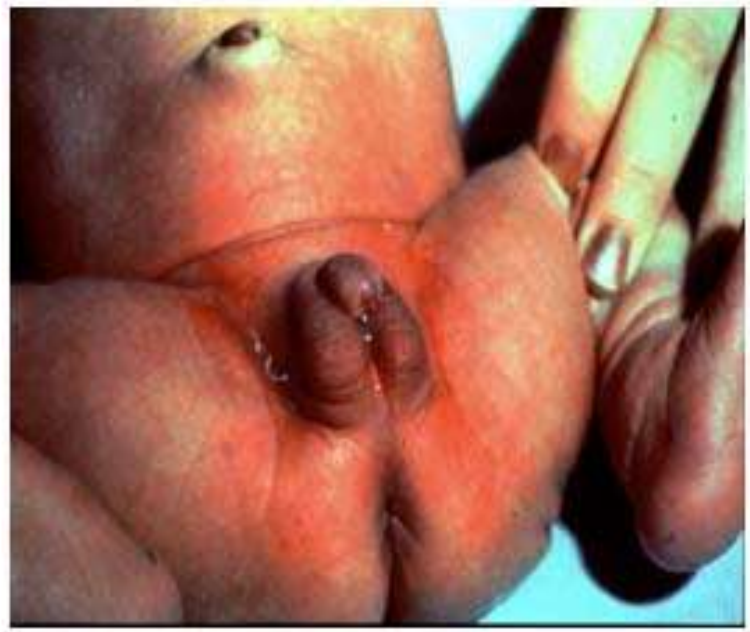
- The immediate cause of obesity is net energy imbalance—the organism consumes more usable calories than it expends. The fundamental cause of obesity is a combination of the organism's genes and environment. Obesity plays an important role in the impairment of lipid and carbohydrate metabolism shown in high-fat diets. It has also been shown that the quality protein intake in a 24-hour period is inversely related to percent central abdominal fat. Quality protein uptake is defined as the ratio of essential amino acids to daily dietary protein.
- The fat cells in the greater omentum will release their fatty acids and trigycerides into the portal circulation, where the blood leads straight to the liver. Thus, the excess of triglycerides and accumulate there. In the liver, most of it will be stored as fat. This concept is known as 'lipotoxicity'. Hypercortisolism, such as in Cushing's syndrome also leads to central obesity. Many prescription drugs, such as dexamethasone and other steroids, can also have side effects resulting in central obesity, especially in the presence of elevated insulin levels.
- The prevalence of abdominal obesity is increasing in western populations, due to a combination of low physical activity and high-energy diets. Waist measurement is and height and weight are used to determine a person’s health. BMI will illustrate the best estimate of your total body fat, while waist measurement gives an estimate of risk of obesity-related disease.

Congenital adrenal hyperplasia

- **Congenital adrenal hyperplasia (CAH)** in a female fetus causes the clitoris to enlarge and the labia major fuse into a scrotal sac. These babies have a mutation in a gene, some enzyme is not expressed which is required to convert cholesterol into corticosteroids, so cholesterol is shunted to the pathway that is not compromised: androgen production. Boys are not affected; girls need a surgery and cortisol for living. If the presence of ACTH is driving the pathway, and it is blocked at this enzyme, the ACTH can only be used to make androgens.

CAH- Excessive and Deficient?

- Congenital Adrenal Hyperplasia (CAH)
  - Autosomal recessive trait (congenital)
  - Deficiency of any of the five enzymes necessary for cortisol production.
  - Increased ACTH (leads to adrenal hyperplasia) MAP IT!
  - Leads to overstimulation of adrenal androgen pathways.
  - Males seldom diagnosed at birth, females have ambiguous genitalia (enlarged clitoris, fused labia, etc).
  - With treatment, surgery, sex characteristics and fertility is normal



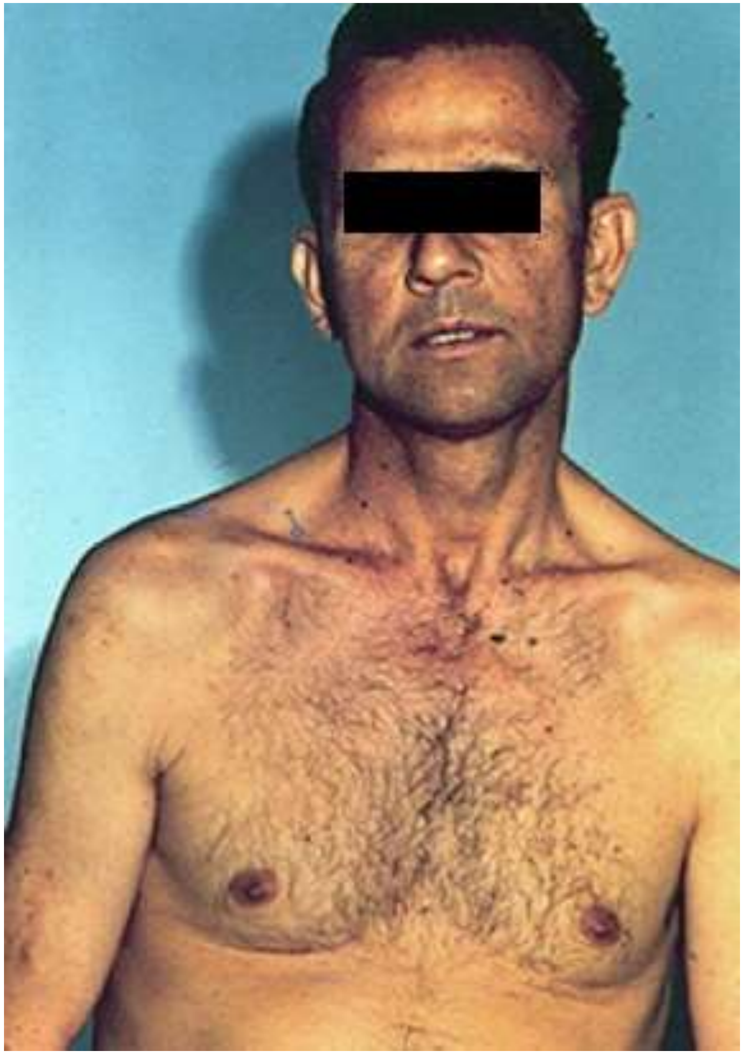
http://www.dshs.state.tx.us/newborn/cah2.shtm

Increased androgen production results in ambiguous genitalia in newborn girls.

ADDISON'S DISEASE

- Also called **Primary Adrenal Insufficiency** and hypoadrenalism; mainly see effects on the hands, fingers, and gums.
- Addison's disease may be caused by anything that disturbs the production of adrenal hormones (for some reason, Tuberculosis attacks the adrenal glands as well as the lungs, and can cause hypoadrenalism).
- In Addison's disease, the adrenal cortex does not respond to pituitary orders. **Cortisol levels are low, but pituitary ACTH and hypothalamus ACTH-RH hormones are high.**
- **Symptoms of Addison's disease are decreased glucose levels, a drop in blood pressure from water and salt imbalance, and darkening of the skin.**

Addison’s Disease



- Thirty-two-year-old man with Addison's disease with generalized hyperpigmentation, most marked on areas exposed to sunlight, such as face and neck. Courtesy of David N Orth, MD.

Adrenal Gland Deficiencies

- Primary Adrenal Insufficiency: Addison's Disease
  - primary hypoadrenalism; entire adrenal gland is destroyed due to atrophy or autoimmune disorder
  - Tuberculosis –disease attacks adrenal gland
  - **ACTH is increased**
- Secondary adrenal insufficiency
  - **deficiency of ACTH**
  - Rapid withdrawal of pharmacologic doses of cortisol
- Signs/symptoms: Water/salt imbalance, plasma volume depletion, low blood glucose, pigmentation, Addisonian crisis (low blood pressure, low blood glucose, need to go to the hospital)

Adrenal Gland Deficiencies

Addison's disease:

- Note the generalised skin pigmentation (in a Caucasion patient) but especially the deposition in the palmer skin creases, nails and gums.

- She was treated many years ago for pulmonary TB. What are the other causes of this condition?

Secondary Adrenal Insufficiency

- In **Secondary Adrenal Insufficiency**, the problem is in pituitary; it is not secreting enough ACTH, maybe because of a tumor. Cortisol levels drop, but hypothalamus ACTH-RH increases.
- A person can also get secondary hypoadrenalism from rapid withdrawal of cortisol meds.
- Symptoms are the same as for primary adrenal insufficiency, except blood tests show that **pituitary ACTH levels are low, cortisol is low, and hypothalamus ACTH-RH is high.**

Conn’s syndrome (hyperaldosteronism)

- Too much aldosterone is secreted.
- Too much salt and water is reabsorbed, person develops high blood pressure.
- Cortisone levels are not effected, so they do not have elevated blood glucose.

The Pineal Gland

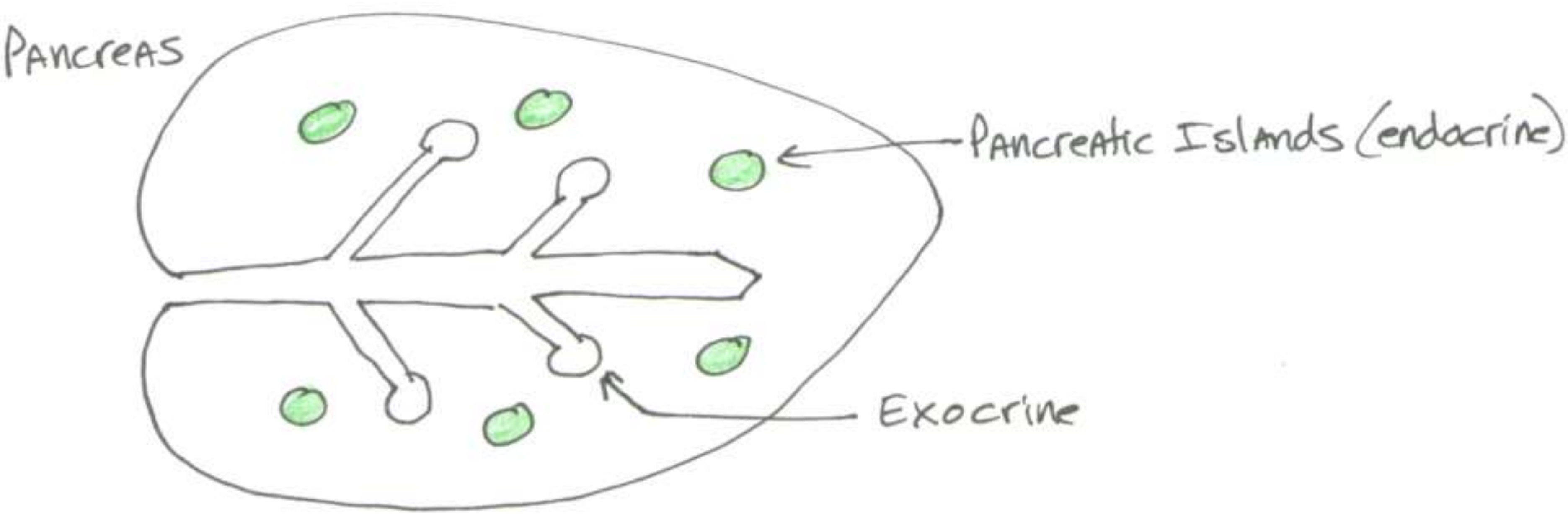
- Shaped like a pinecone
- Pinealocytes secrete melatonin
  - A hormone that regulates circadian rhythms (sense of daytime and night; it regulates sleep cycle)
- “Pineal sand” is radiopaque
  - Mineral deposits within pineal gland.
  - Used as a landmark to identify other brain structures in X-Rays

Pineal Sand

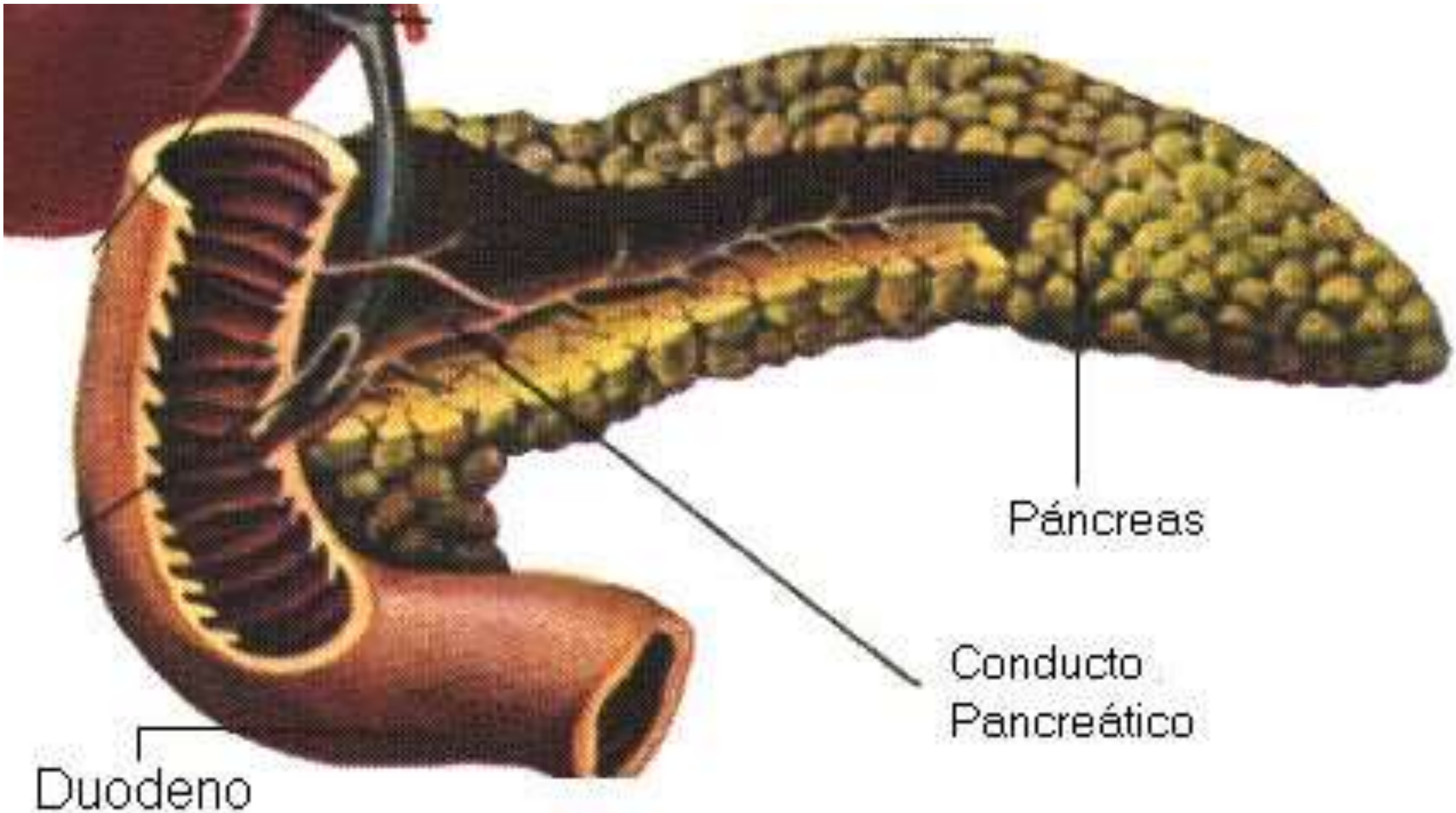
The Pancreas

- Is an endocrine and exocrine gland
  - Exocrine cells – acinar cells – secrete digestive enzymes into a duct.
  - Endocrine cells – pancreatic islets (islets of Langerhans)
    - About one million islets – scattered throughout the pancreas
    - Secrete insulin
    - Secretes glucogon

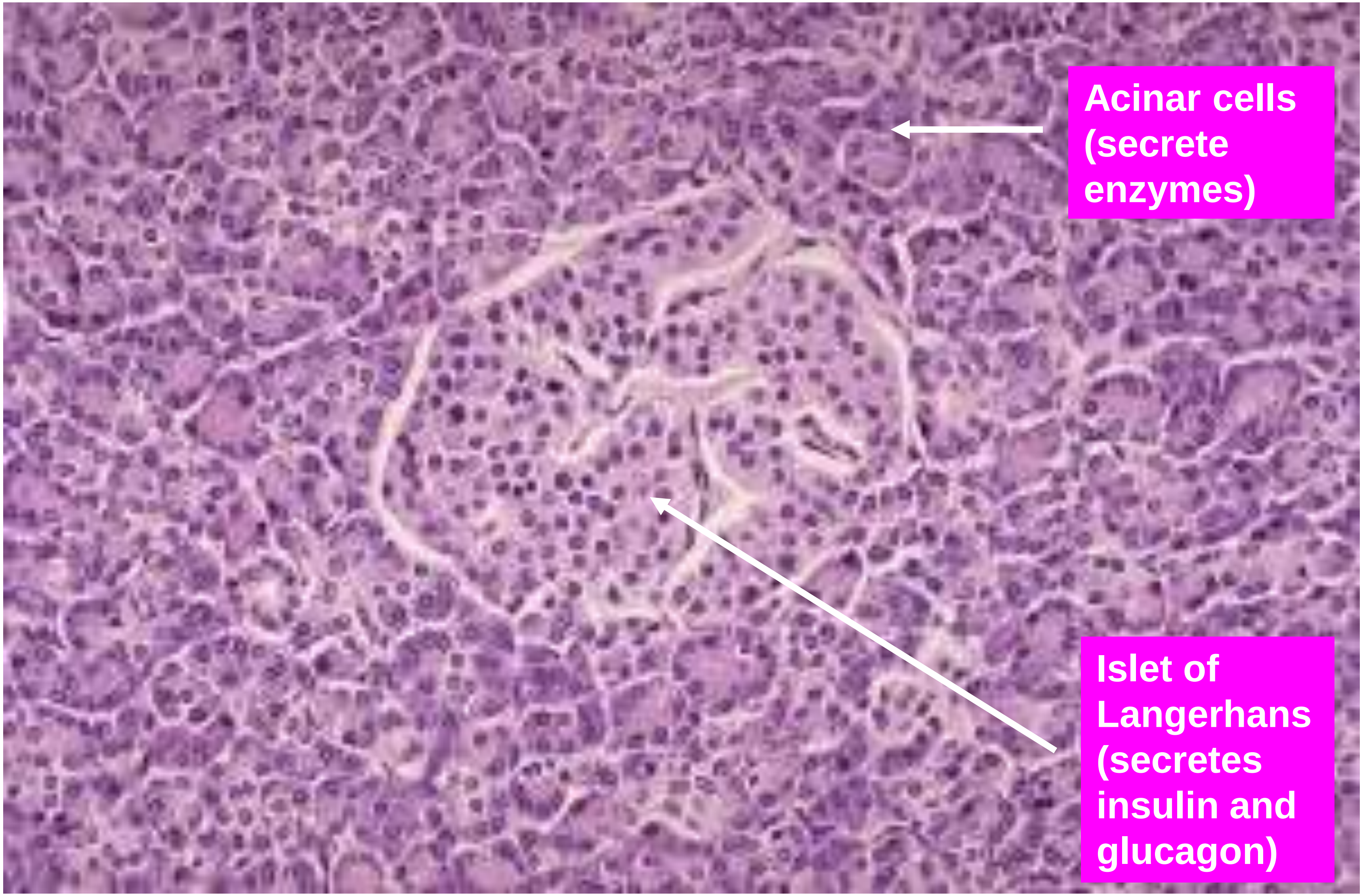
Pancreas



Pancreas



Pancreas


Acinar cells (secrete enzymes)
Islet of Langerhans (secretes insulin and glucagon)

Endocrine Hormones of the Pancreas

- Glucagon
  - Signals liver to break down glycogen into glucose
  - Raises blood sugar
- Insulin
  - Signals most body cells to take up glucose from the blood
  - Promotes storage of glucose as glycogen in liver
  - Lowers blood sugar

Regulation of Blood Glucose Levels

- When blood glucose is high, the pancreas secretes **insulin**, which makes the cells to take in the sugar from the bloodstream. If the blood sugar levels remain high, the excess sugar is taken to the liver and converted to glycogen for storage.
- When blood glucose is low, the pancreas secretes **glucagon**, which tells the liver to take the glycogen and break it back down into glucose and release it into the bloodstream.
- Gluconeogenesis is when the liver takes fatty acids (leftover from fat metabolism) and joins them to amino acids (from broken down proteins), and makes new glucose molecules that you did not get from eating glucose. These new glucose molecules are then released into the bloodstream to elevate blood glucose levels.
- Summary:
  – When blood glucose is high, **insulin** lowers blood glucose levels.
  – When blood glucose is low, glucagon causes **glycogen breakdown** and **gluconeogenesis** to raise blood glucose levels.

Diabetes

- **DIABETES INSIPIDUS**
  - pituitary gland does not secrete antidiuretic hormone, or the kidney does not respond to the hormone. It can be caused by damage to the pituitary or kidney damage.
- **DIABETES MELLITUS**
  - hereditary lack of insulin secretion in the pancreas, or resistance to insulin by the body's cells.
    - **Type I diabetes** (insulin dependent, develops in children)
      - **Destruction of pancreatic islets by autoimmune disorders.**
      - **Need insulin injections daily throughout life.**
    - **Type II diabetes** (not insulin dependent, develops in adults)
      - **Consequence of obesity: cells are less sensitive to insulin.**
      - **Initially treated with diet and exercise.**
      - **Oral medicines or injected insulin may be needed.**


Your sugar level is off the chart. Don't worry. We'll whip you back into shape in no time...
DOCTORATE
Reynolds

- Type 1 Diabetes VIDEO
- Type 2 Diabetes VIDEO

VIDEOS

- Endocrine System 3 mins
- Pancreas, testes, and ovaries 5 mins

The Gonads

- Ovaries
  - Secrete progesterone
    - Prepares uterus for pregnancy
  - Secrete estrogen
    - Female secondary sex characteristics
    - Stores enough for several months
- Testes
  - Secrete androgens (e.g. testosterone)
    - Promotes the formation of sperm
    - Maintains secondary sex characteristics
    - Testes are the primary sex organs in the male, NOT the penis

DANGERS OF STEROIDS

- Steroids that weightlifters take are synthetic testosterone, and they are taken in doses 100x larger than a prescription, so they are dangerous.
- Although they increase muscle size, they increase rage and aggression, cause kidney and liver disease, cancer, severe acne, high blood pressure, high cholesterol, impotence, baldness, decreases the size of testicles and causes a low sperm count and sterility.
- In males, it causes baldness and increases the breasts.
- In women it causes hair on their face and chest, and decreases the breasts.
- In children, it stunts the growth.
- In everyone, they can shorten the life span by several decades.

News Articles

- Effects of Stress
- http://ehealthmd.com/library/stress/STR_affect.html
- Effects of Steroids on Behavior
- http://kidshealth.org/parent/emotions/behavior/steroids.html#
- Facts about steroids
- http://www.drugabuse.gov/infofacts/Steroids.html

Other Endocrine Glands

- Many of the glands we talked about have no other function than to make hormones. But almost all organs are endocrine glands in addition to their other functions.
- **Heart** pumps blood and produces hormones
- **Liver** makes enzymes, produces hormones
- **GI tract** digests food and produces hormones.
- **Kidney:** excretes wastes, produces hormones
- **Dermis** Involved in vitamin D synthesis, makes hormones
- **Bones** stores calcium and produces hormones.
- **Placenta** oxygenates and produces hormones.

- The only thing that does NOT make hormones are epithelial glands that have ducts (hormone glands are by definition without ducts).

The rest of this PPT is not on the test or quiz

Other Hormones are made in these organs

- **Heart**
- **Liver**
- **GI tract**
- **Kidney**
- **Dermis**
- **Bones**
- **Placenta**

Heart Hormones: Natriuretic Peptides

- In response to a rise in blood pressure, the heart releases two peptides:
- **A-type Natriuretic Peptide (ANP)**
- This hormone is released from stretched atria (hence the "A").

- **B-type Natriuretic Peptide (BNP)**
- This hormone is released from the ventricles. (It was first discovered in brain tissue; hence the "B".)

- Both hormones lower blood pressure by
- relaxing arterioles
- inhibiting the secretion of renin and aldosterone
- inhibiting the reabsorption of sodium ions by the kidneys.
- The latter two effects reduce the reabsorption of water by the kidneys. So the volume of urine increases as does the amount of sodium excreted in it. The net effect of these actions is to reduce blood pressure by reducing the volume of blood in the circulatory system.

- These effects give ANP and BNP their name (natrium = sodium; uresis = urinate).

Liver Hormones

- **Angiotensinogen** (precursor molecule that will raise blood pressure)
- **Thrombopoietin** (stimulates development of platelets)
- **Hepcidin** (blocks the release of iron from intracellular stores in the body so iron is not lost, especially during bacterial infections. Bacteria often need to take our iron to sustain themselves)

GI tract Hormones

- Stomach secretes
  - **Gastrin** (Tells parietal cells to secrete HCl)
- Duodenum secretes
  - **Secretin** (tells pancreas to secrete bicarbonate)
  - **CCK** (Tells pancreas to secrete digestive enzymes, and gallbladder to release bile)
  - **GIP** (Tells pancreas to release insulin and also causes fat to be broken down into fatty acids)
  - **Motilin** (Initiates peristalsis and tells chief cells in stomach to secrete pepsinogen).

Kidney Hormones

- **Erythropoietin (EPO):** Stimulates production of red blood cells
- **Calcitriol (Vitamin D3):** Promotes the absorption of calcium from food in the intestines
- **Renin**: Converts angiotensinogen (from liver) into angiotensin-1, and also tells ACE (lung enzyme) to cut angiotensin-1 into angiotensin-2, which causes vasoconstriction, which raises blood pressure.
- **Adenosine**: Causes vasoconstriction of afferent arterioles in the glomerulus. This decreases water loss, and increases blood pressure.

Dermis Hormone

- When ultraviolet radiation strikes the skin, it triggers the conversion of dehydrocholesterol (a cholesterol derivative) into calciferol.
- Calciferol travels in the blood to the liver where it is converted into a precursor of vitamin D3.
- This compound travels to the kidneys where it is converted into calcitriol (vitamin D3). This final step is promoted by the parathyroid hormone (PTH).
- Calcitriol acts on the cells of the intestine to promote the absorption of calcium from food, and it also acts on bone to mobilize calcium from the bone to the blood.
- Although called a vitamin, calciferol qualifies as a hormone because it is made in certain cells, carried in the blood, and affects target cells.

Bone Hormones

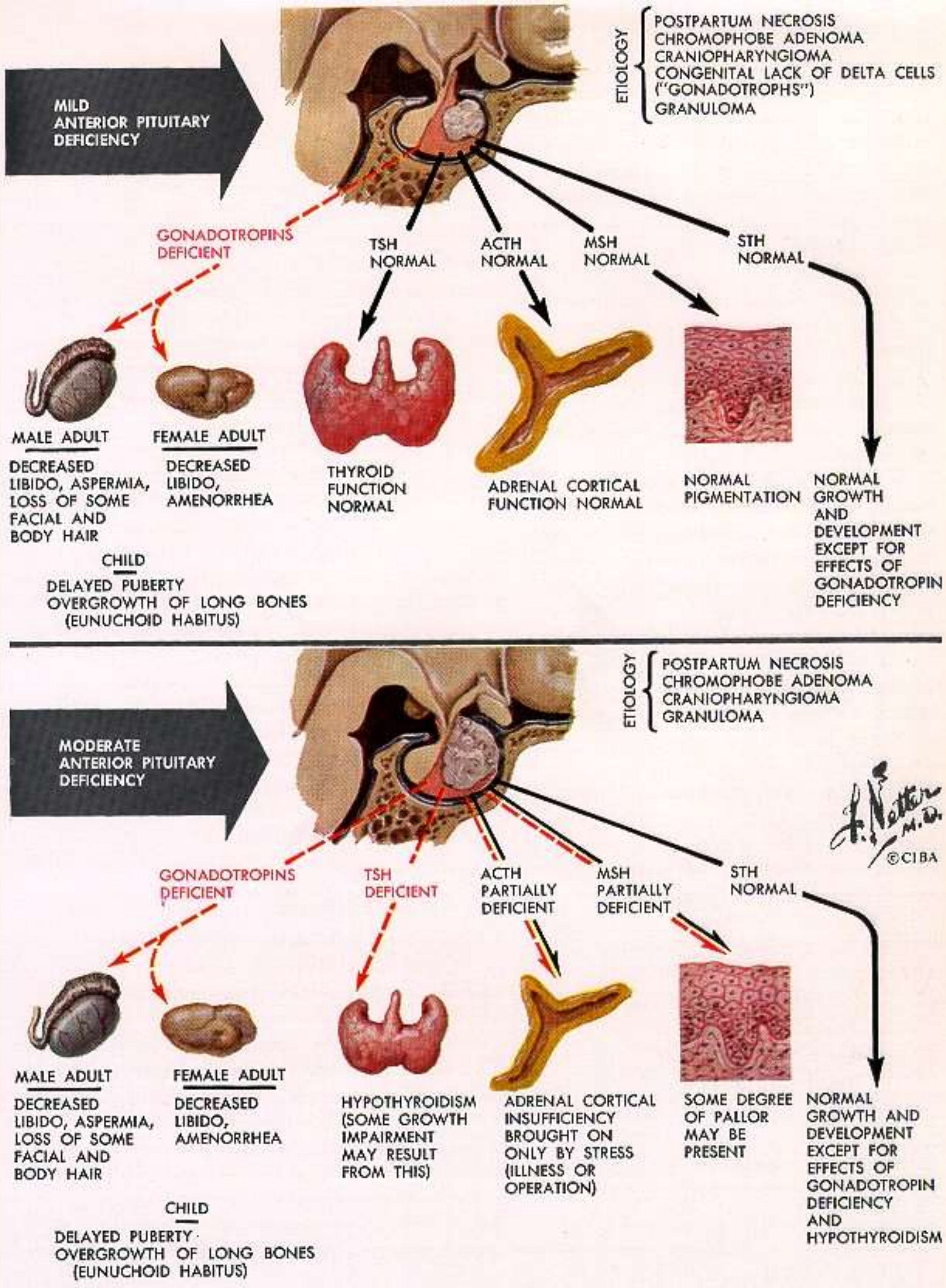
- **Fibroblast Growth Factor** (Tells kidneys to reduce phosphate reabsorption)
- **Osteocalcin** (lowers blood glucose and decreases fat deposition)

Placenta Hormones

- **Human Chorionic Gonadotropin (hCG)** ensures that the corpus luteum continues to secrete progesterone so the endometrial lining won’t slough off. hCG also suppresses the maternal immunologic response so that placenta is not rejected.
- **Human Placental Lactogen** promotes mammary gland growth in preparation for lactation.

Sample Study Chart

Hormone	Where Made	Target Organ	Effect
ADH	Posterior pituitary	Kidney	Increases water reabsorption
Parathyroid	Parathyroid gland	Bone, kidney, intestines	Increases blood calcium levels
Thyroid	Thyroid gland	Most cells	Increases metabolic rate
ACTH	Anterior pituitary	Adrenal cortex	Stimulates release of cortisol
Cortisol	Adrenal cortex	Most cells	Affects glucose and protein blood levels and metabolic rates



MILD ANTERIOR PITUITARY DEFICIENCY
ETIOLOGY
POSTPARTUM NECROSIS
CHROMOPHOBE ADENOMA
CRANIOPHARYNGIOMA
CONGENITAL LACK OF DELTA CELLS ("GONADOTROPHS")
GRANULOMA
GONADOTROPINS DEFICIENT
TSH NORMAL
ACTH NORMAL
MSH NORMAL
STH NORMAL
MALE ADULT
DECREASED LIBIDO, ASPERMIA, LOSS OF SOME FACIAL AND BODY HAIR
FEMALE ADULT
DECREASED LIBIDO, AMENORRHEA
CHILD
DELAYED PUBERTY
OVERGROWTH OF LONG BONES (EUNUCHOID HABITUS)
THYROID FUNCTION NORMAL
ADRENAL CORTICAL FUNCTION NORMAL
NORMAL PIGMENTATION
NORMAL GROWTH AND DEVELOPMENT EXCEPT FOR EFFECTS OF GONADOTROPIN DEFICIENCY
MODERATE ANTERIOR PITUITARY DEFICIENCY
ETIOLOGY
POSTPARTUM NECROSIS
CHROMOPHOBE ADENOMA
CRANIOPHARYNGIOMA
GRANULOMA
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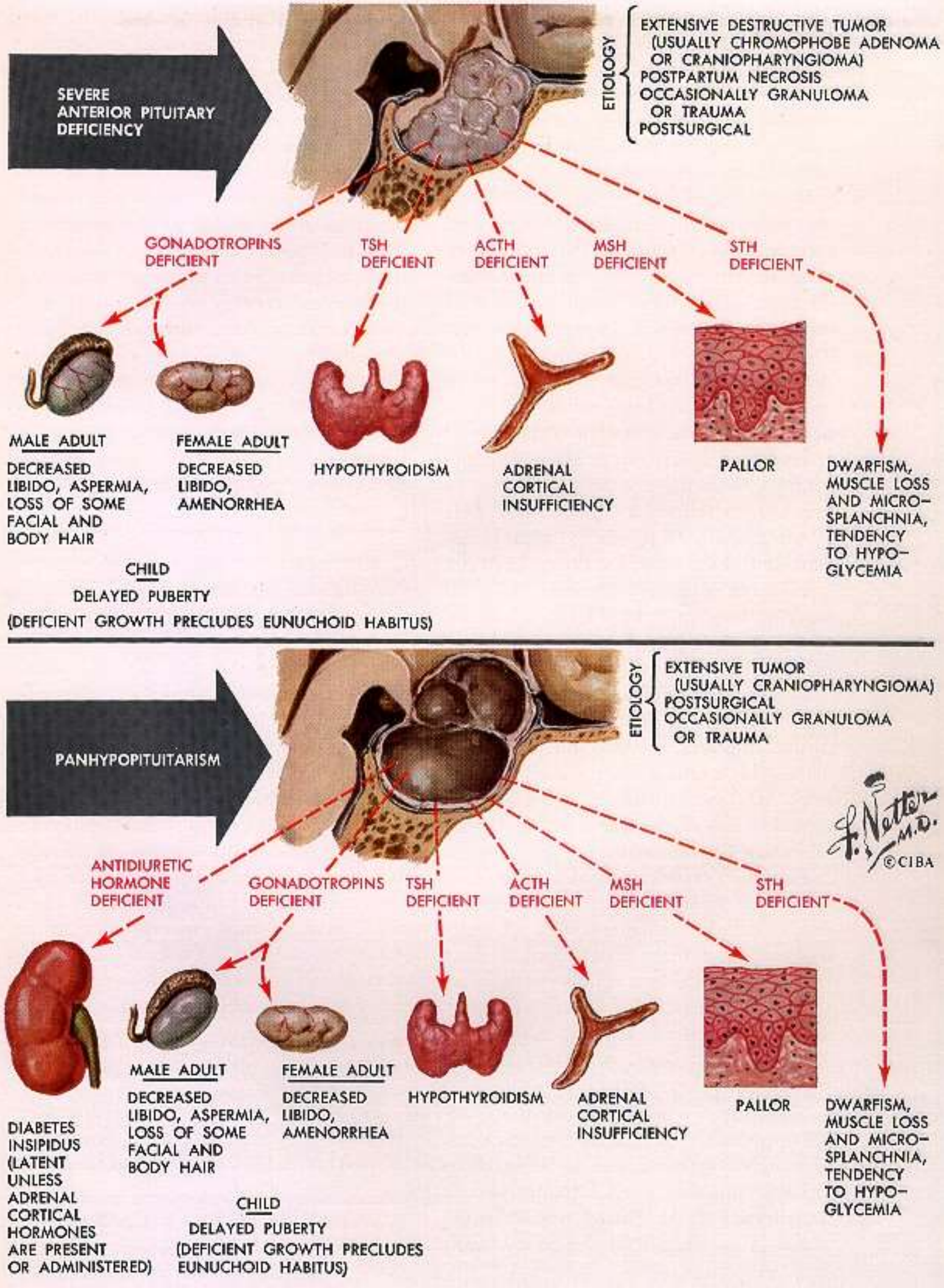
GONADOTROPINS DEFICIENT
TSH DEFICIENT
ACTH PARTIALLY DEFICIENT
MSH PARTIALLY DEFICIENT
STH NORMAL
MALE ADULT
DECREASED LIBIDO, ASPERMIA, LOSS OF SOME FACIAL AND BODY HAIR
FEMALE ADULT
DECREASED LIBIDO, AMENORRHEA
CHILD
DELAYED PUBERTY
OVERGROWTH OF LONG BONES (EUNUCHOID HABITUS)
HYPOTHYROIDISM (SOME GROWTH IMPAIRMENT MAY RESULT FROM THIS)
ADRENAL CORTICAL INSUFFICIENCY BROUGHT ON ONLY BY STRESS (ILLNESS OR OPERATION)
SOME DEGREE OF PALLOR MAY BE PRESENT
NORMAL GROWTH AND DEVELOPMENT EXCEPT FOR EFFECTS OF GONADOTROPIN DEFICIENCY AND HYPOTHYROIDISM


SEVERE ANTERIOR PITUITARY DEFICIENCY
ETIOLOGY
EXTENSIVE DESTRUCTIVE TUMOR (USUALLY CHROMOPHOBE ADENOMA OR CRANIOPHARYNGIOMA)
POSTPARTUM NECROSIS
OCCASIONALLY GRANULOMA OR TRAUMA
POSTSURGICAL
GONADOTROPINS DEFICIENT
TSH DEFICIENT
ACTH DEFICIENT
MSH DEFICIENT
STH DEFICIENT
MALE ADULT
DECREASED LIBIDO, ASPERMIA, LOSS OF SOME FACIAL AND BODY HAIR
FEMALE ADULT
DECREASED LIBIDO, AMENORRHEA
HYPOTHYROIDISM
ADRENAL CORTICAL INSUFFICIENCY
PALLOR
DWARFISM, MUSCLE LOSS AND MICRO-SPLANCHNIA, TENDENCY TO HYPO-GLYCEMIA
CHILD
DELAYED PUBERTY
(DEFICIENT GROWTH PRECLUDES EUNUCHOID HABITUS)
PANHYPOPITUITARISM
ETIOLOGY
EXTENSIVE TUMOR (USUALLY CRANIOPHARYNGIOMA)
POSTSURGICAL
OCCASIONALLY GRANULOMA OR TRAUMA
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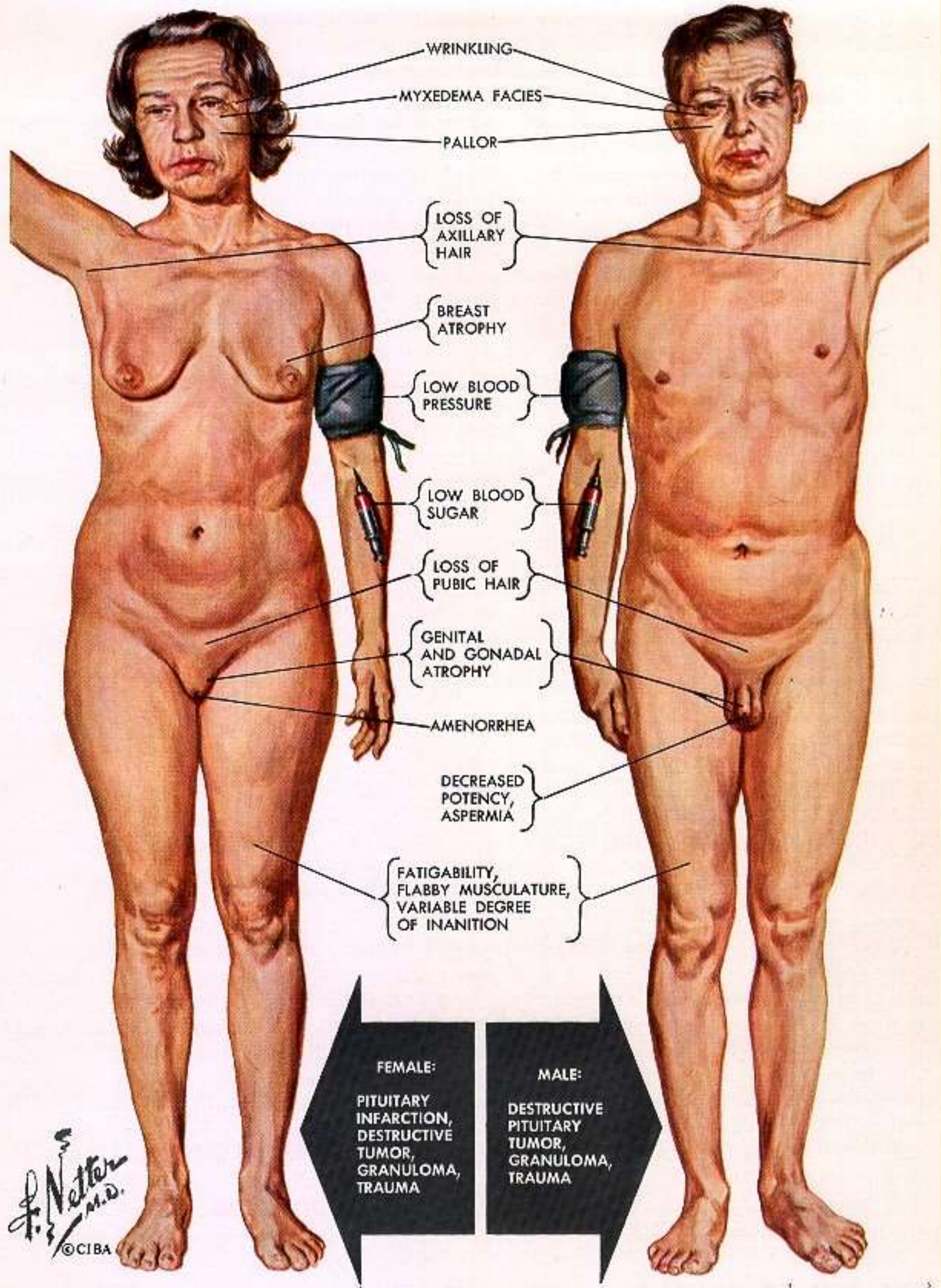
ANTIDIURETIC HORMONE DEFICIENT
GONADOTROPINS DEFICIENT
TSH DEFICIENT
ACTH DEFICIENT
MSH DEFICIENT
STH DEFICIENT
MALE ADULT
DECREASED LIBIDO, ASPERMIA, LOSS OF SOME FACIAL AND BODY HAIR
FEMALE ADULT
DECREASED LIBIDO, AMENORRHEA
HYPOTHYROIDISM
ADRENAL CORTICAL INSUFFICIENCY
PALLOR
DWARFISM, MUSCLE LOSS AND MICRO-SPLANCHNIA, TENDENCY TO HYPO-GLYCEMIA
DIABETES INSIPIDUS (LATENT UNLESS ADRENAL CORTICAL HORMONES ARE PRESENT OR ADMINISTERED)
CHILD
DELAYED PUBERTY
(DEFICIENT GROWTH PRECLUDES EUNUCHOID HABITUS)


WRINKLING
MYXEDEMA FACIES
PALLOR
LOSS OF AXILLARY HAIR
BREAST ATROPHY
LOW BLOOD PRESSURE
LOW BLOOD SUGAR
LOSS OF PUBIC HAIR
GENITAL AND GONADAL ATROPHY
AMENORRHEA
DECREASED POTENCY, ASPERMIA
FATIGABILITY, FLABBY MUSCULATURE, VARIABLE DEGREE OF INANITION
FEMALE:
PITUITARY INFARCTION, DESTRUCTIVE TUMOR, GRANULOMA, TRAUMA
MALE:
DESTRUCTIVE PITUITARY TUMOR, GRANULOMA, TRAUMA
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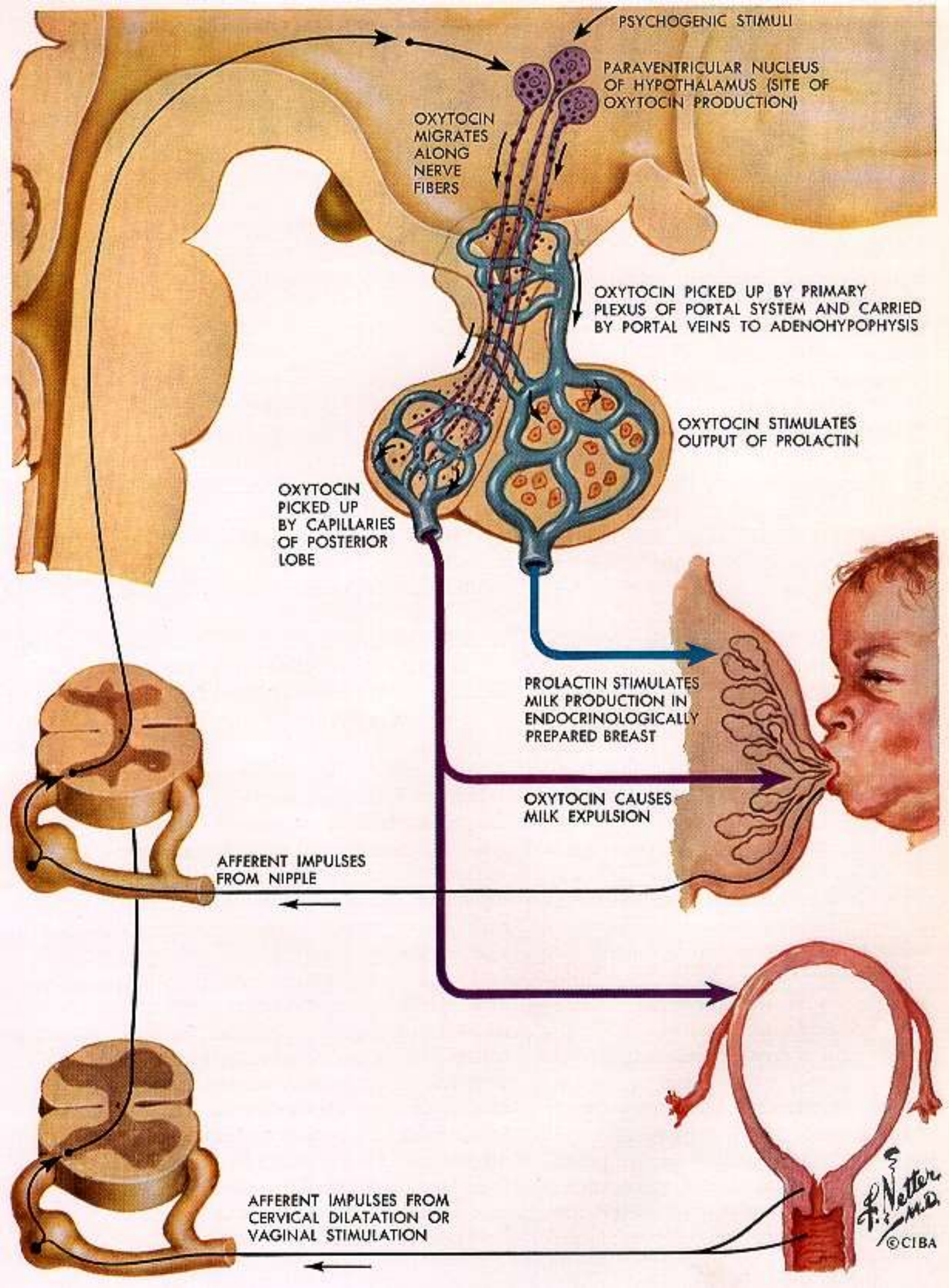
a)



PSYCHOGENIC STIMULI
PARAVENTRICULAR NUCLEUS OF HYPOTHALAMUS (SITE OF OXYTOCIN PRODUCTION)
OXYTOCIN MIGRATES ALONG NERVE FIBERS
OXYTOCIN PICKED UP BY PRIMARY PLEXUS OF PORTAL SYSTEM AND CARRIED BY PORTAL VEINS TO ADENOHYPOPHYSIS
OXYTOCIN STIMULATES OUTPUT OF PROLACTIN
OXYTOCIN PICKED UP BY CAPILLARIES OF POSTERIOR LOBE
PROLACTIN STIMULATES MILK PRODUCTION IN ENDOCRINOLOGICALLY PREPARED BREAST
OXYTOCIN CAUSES MILK EXPULSION
AFFERENT IMPULSES FROM NIPPLE
AFFERENT IMPULSES FROM CERVICAL DILATATION OR VAGINAL STIMULATION
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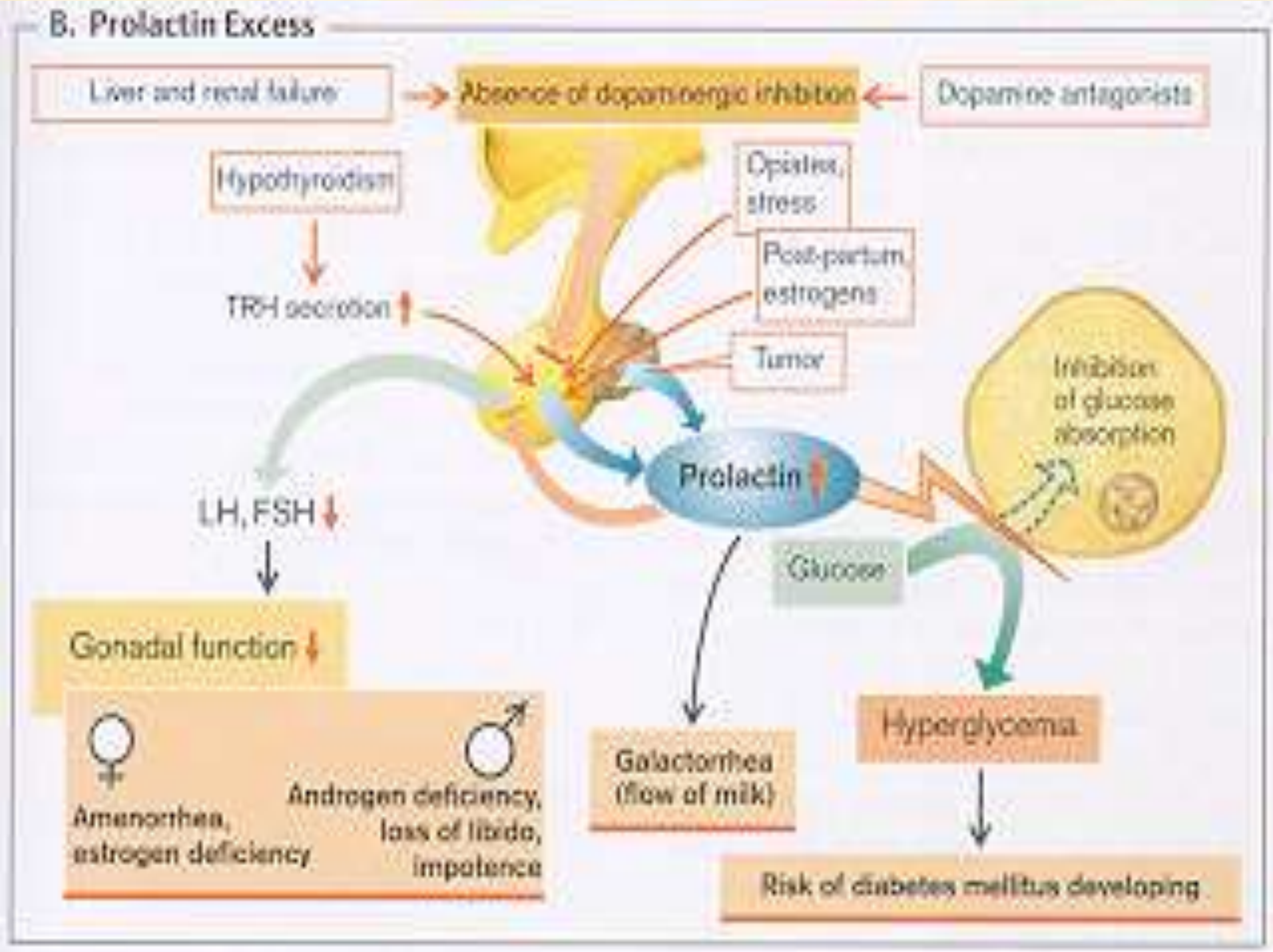
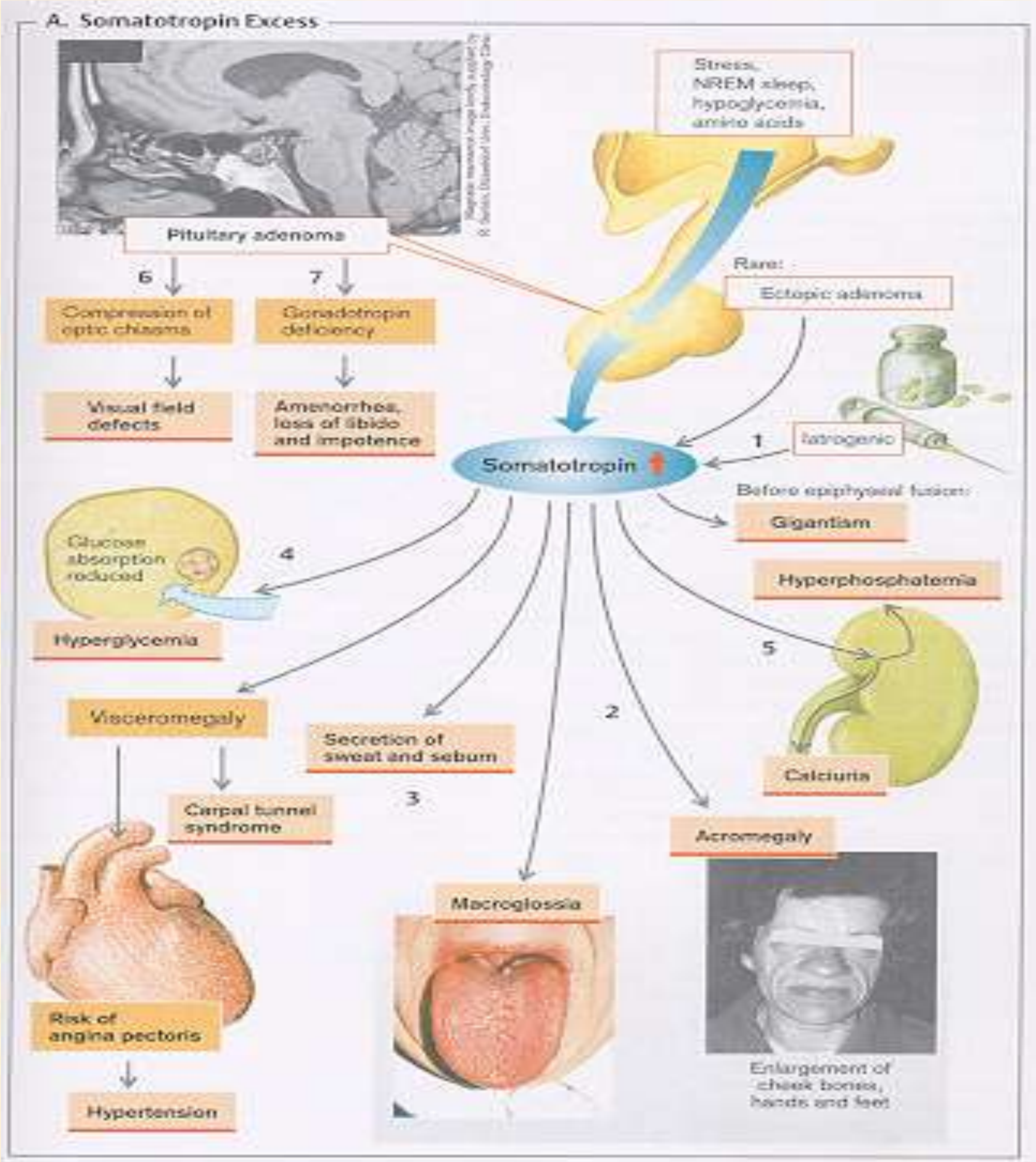
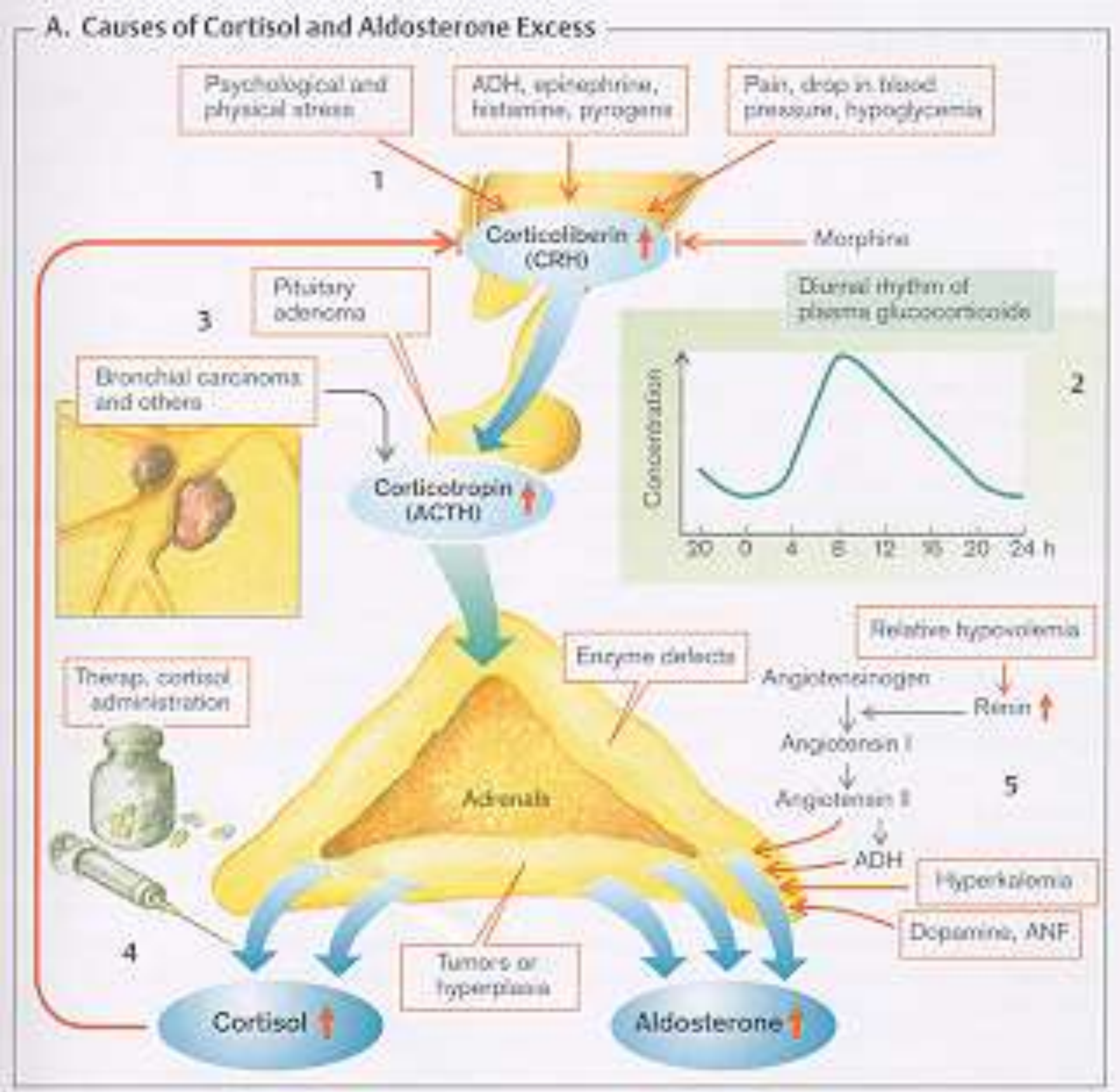
a)

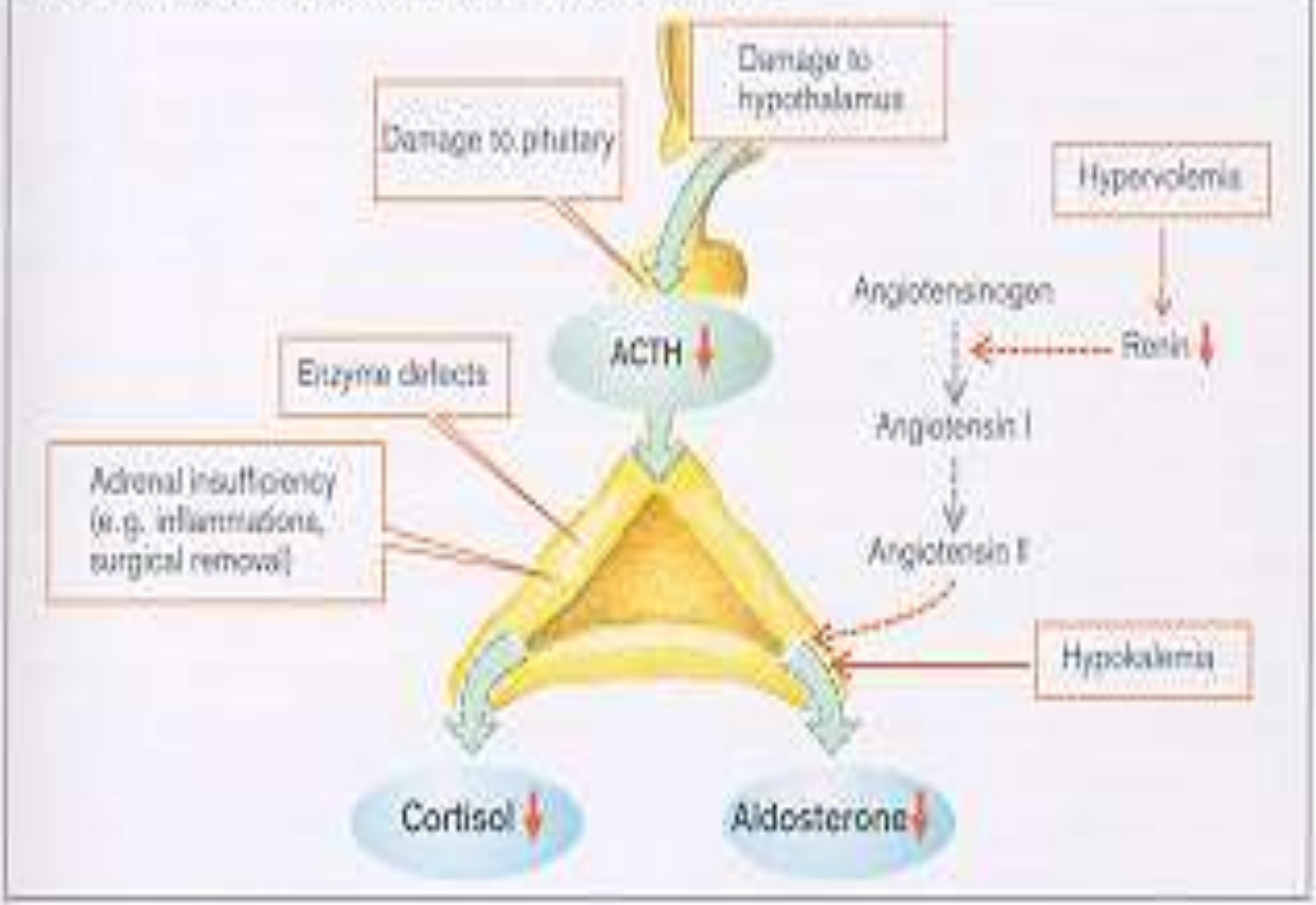

B. Prolactin Excess
Liver and renal failure
Absence of dopaminergic inhibition
Dopamine antagonists
Hypothyroidism
Opiates, stress
Post-partum estrogens
TRH secretion
Tumor
Inhibition of glucose absorption
Prolactin
LH, FSH
Glucose
Gonadal function
Amenorrhea, estrogen deficiency
Androgen deficiency, loss of libido, impotence
Galactorrhea (flow of milk)
Hyperglycema
Risk of diabetes mellitus developing

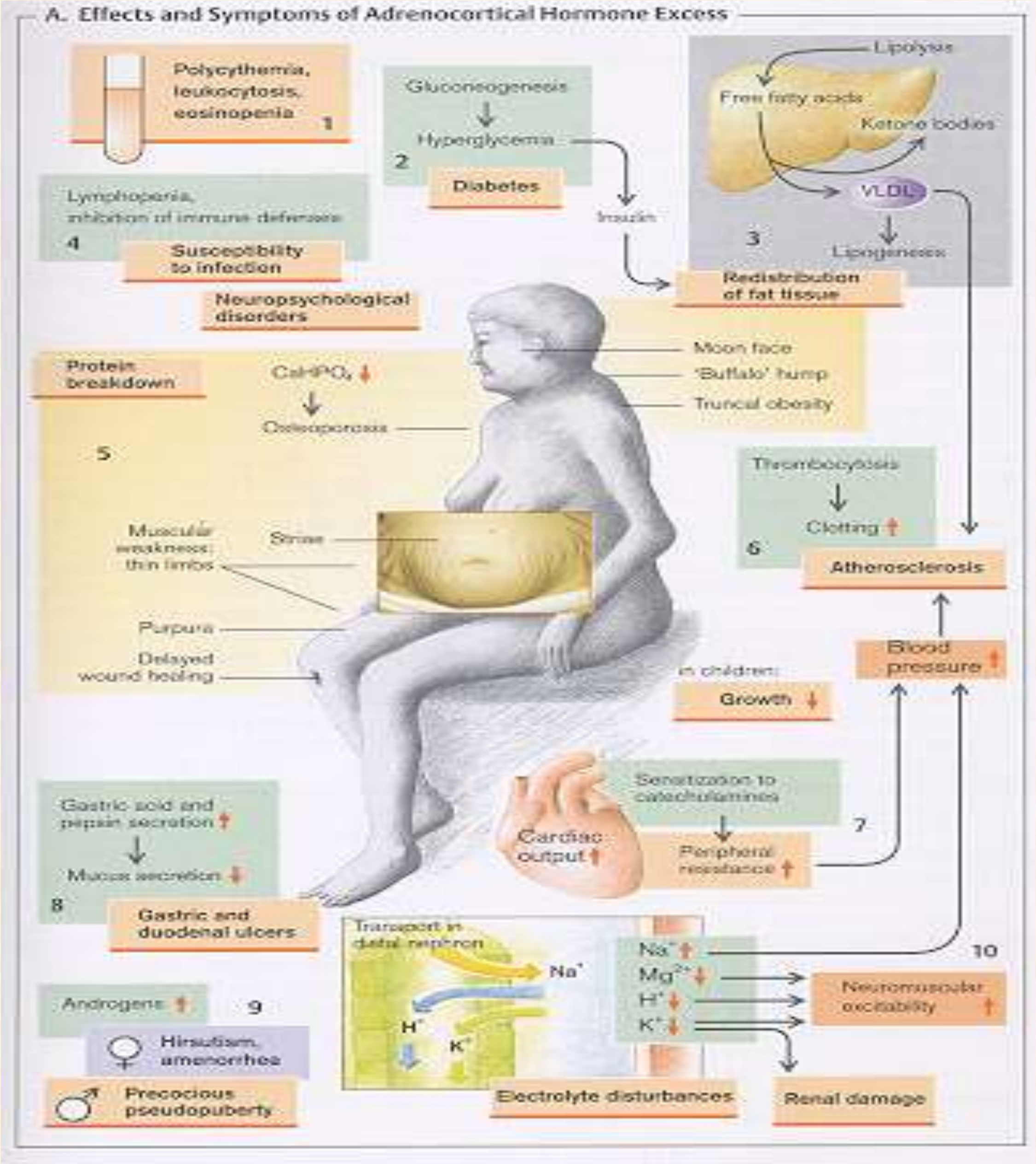
b)

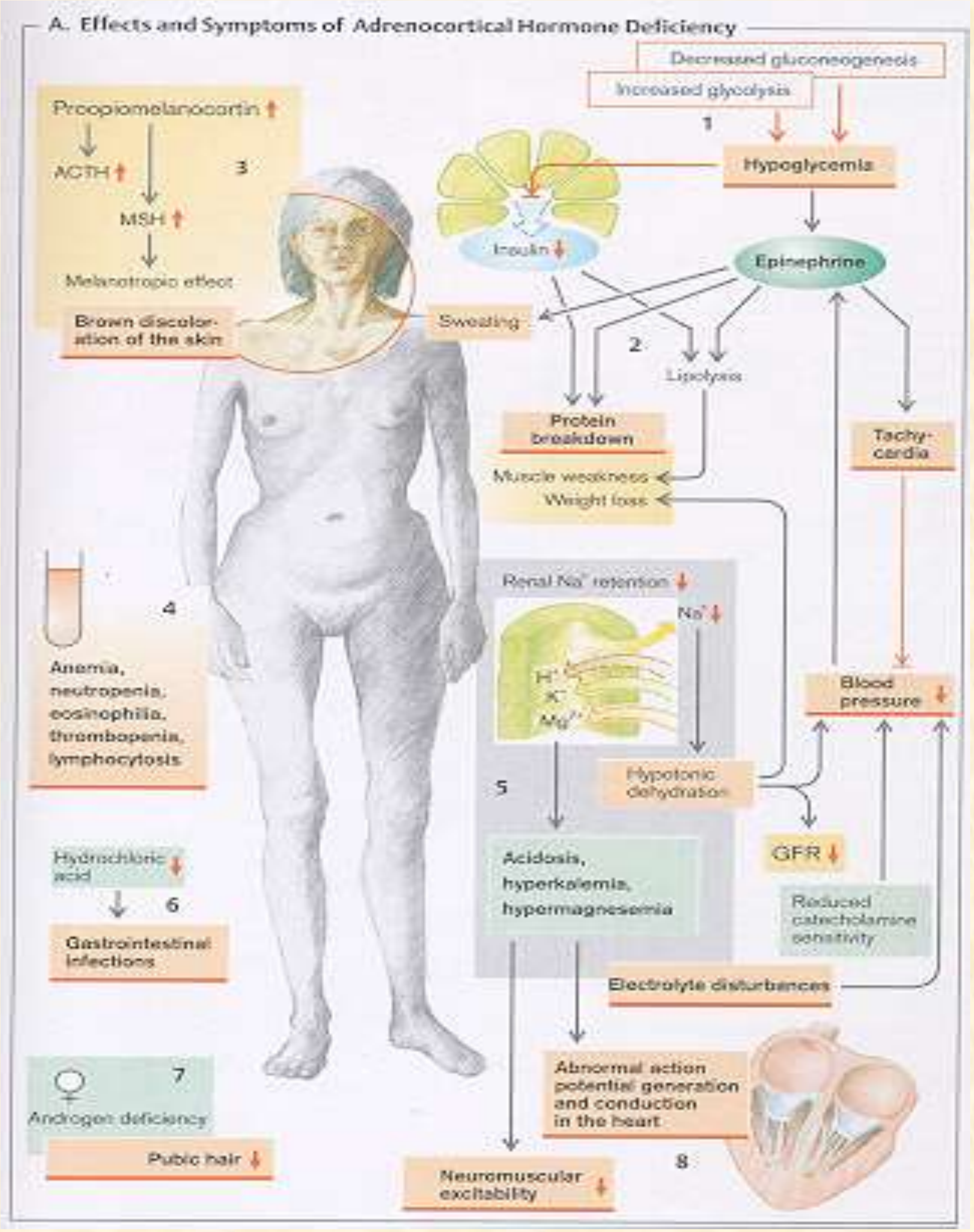

A. Somatotropin Excess
Pituitary adenoma
Stress,
NREM sleep,
hypoglycemia,
amino acids
Rare:
Ectopic adenoma
6
7
Compression of optic chiasma
Gonadotropin deficiency
Visual field defects
Amenorrhea, loss of libido and impotence
Somatotropin
1
Iatrogenic
Before epiphyseal fusion:
Gigantism
Hyperphosphatemia
Glucose absorption reduced
4
Hyperglycemia
5
Visceromegaly
Secretion of sweat and sebum
2
Calciuria
Carpal tunnel syndrome
3
Acromegaly
Macroglossia
Risk of angina pectoris
Hypertension
Enlargement of cheek bones, hands and feet

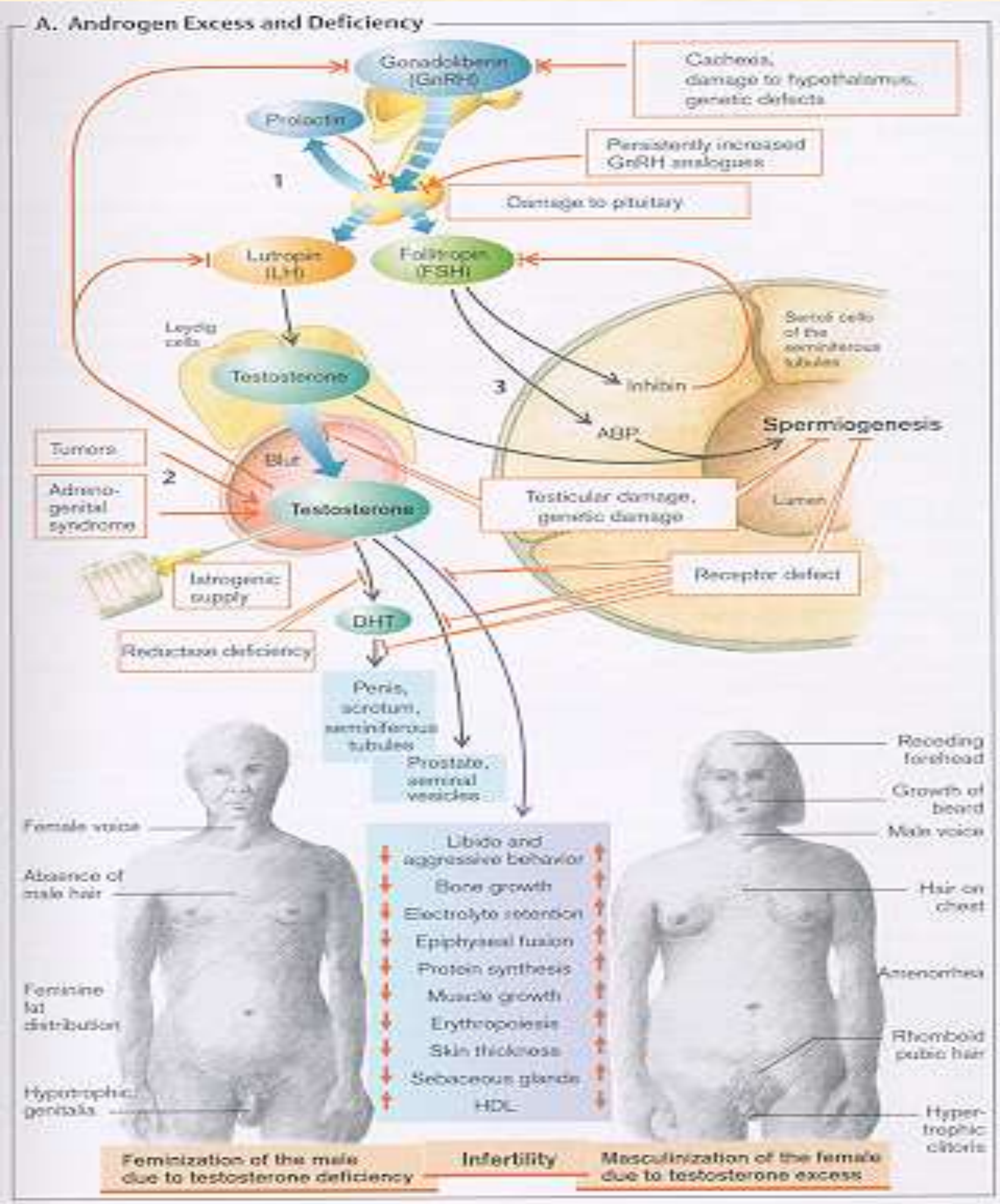

A. Causes of Cortisol and Aldosterone Excess
Psychological and physical stress
ADH, epinephrine, histamine, pyrogens
Pain, drop in blood pressure, hypoglycemia
1
Corticoliberin (CRH)
Morphine
Pituitary adenoma
3
Diurnal rhythm of plasma glucocorticoids
2
Concentration
20 0 4 8 12 16 20 24 h
Bronchial carcinoma and others
Corticotropin (ACTH)
Relative hypovolemia
Enzyme defects
Angiotensinogen
Renin
Therap. cortisol administration
Angiotensin I
Angiotensin II
5
Adrenals
ADH
Hyperkalemia
Dopamine, ANF
4
Tumors or hyperplasia
Cortisol
Aldosterone

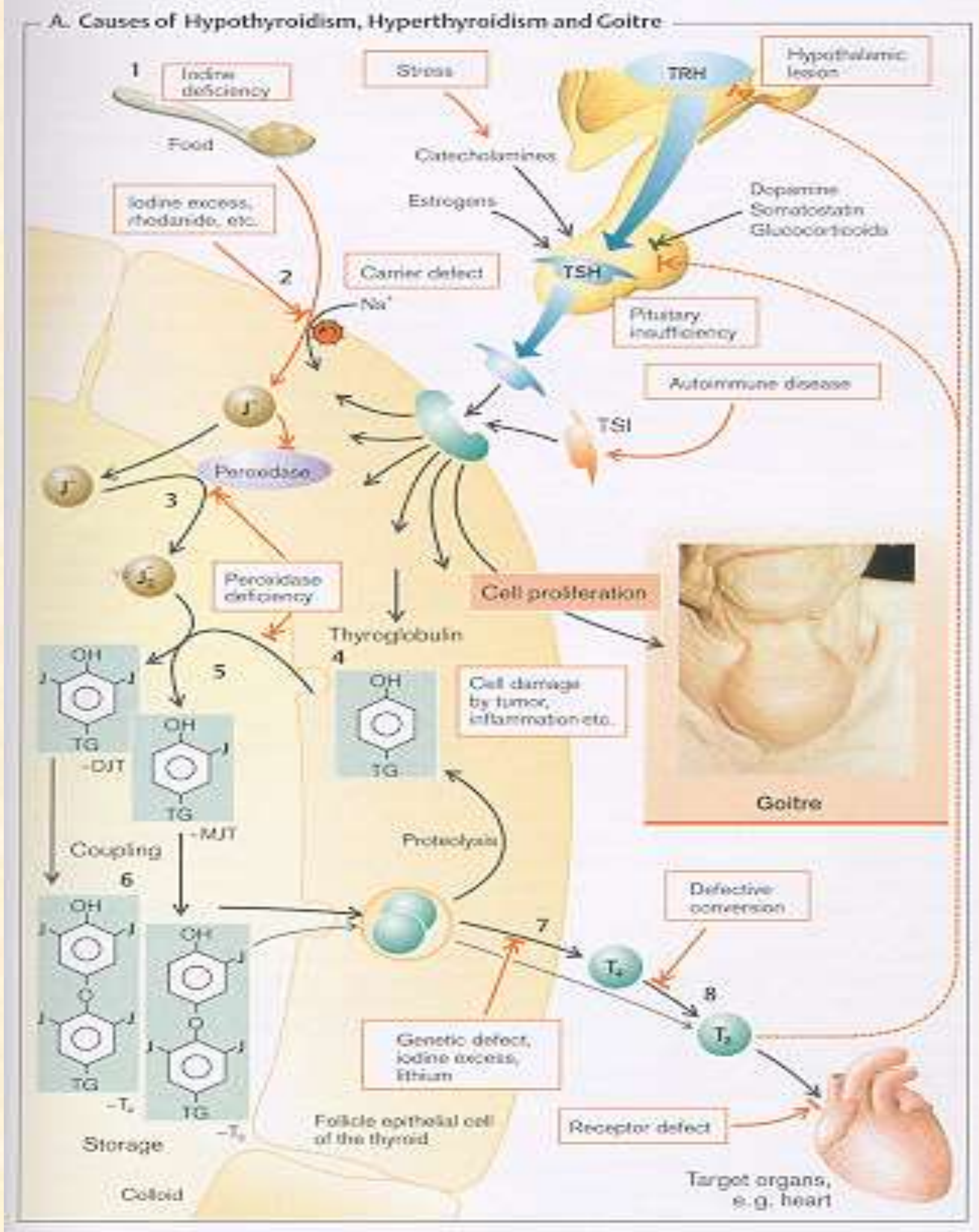
B. Causes of Cortisol and Aldosterone Deficiency

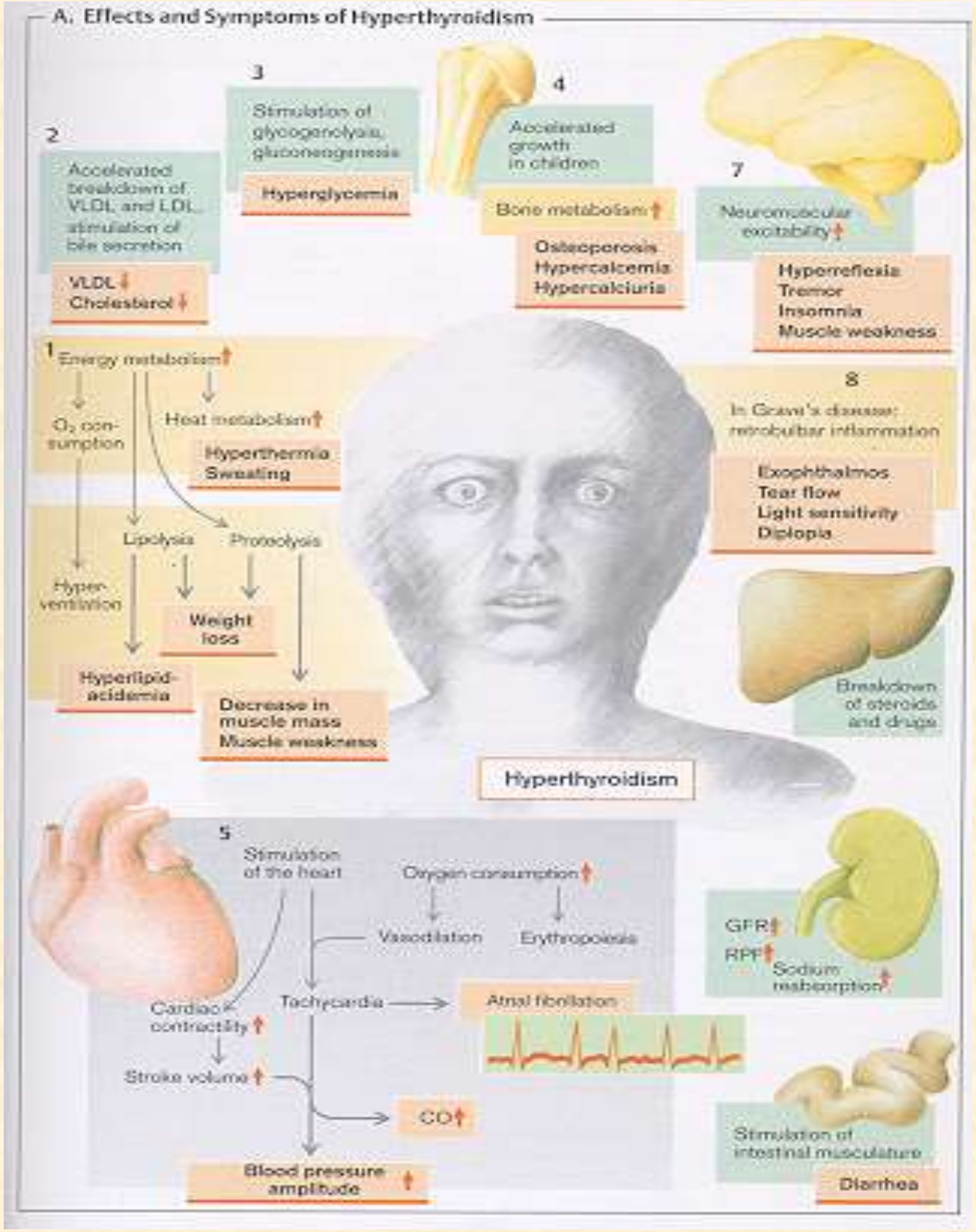


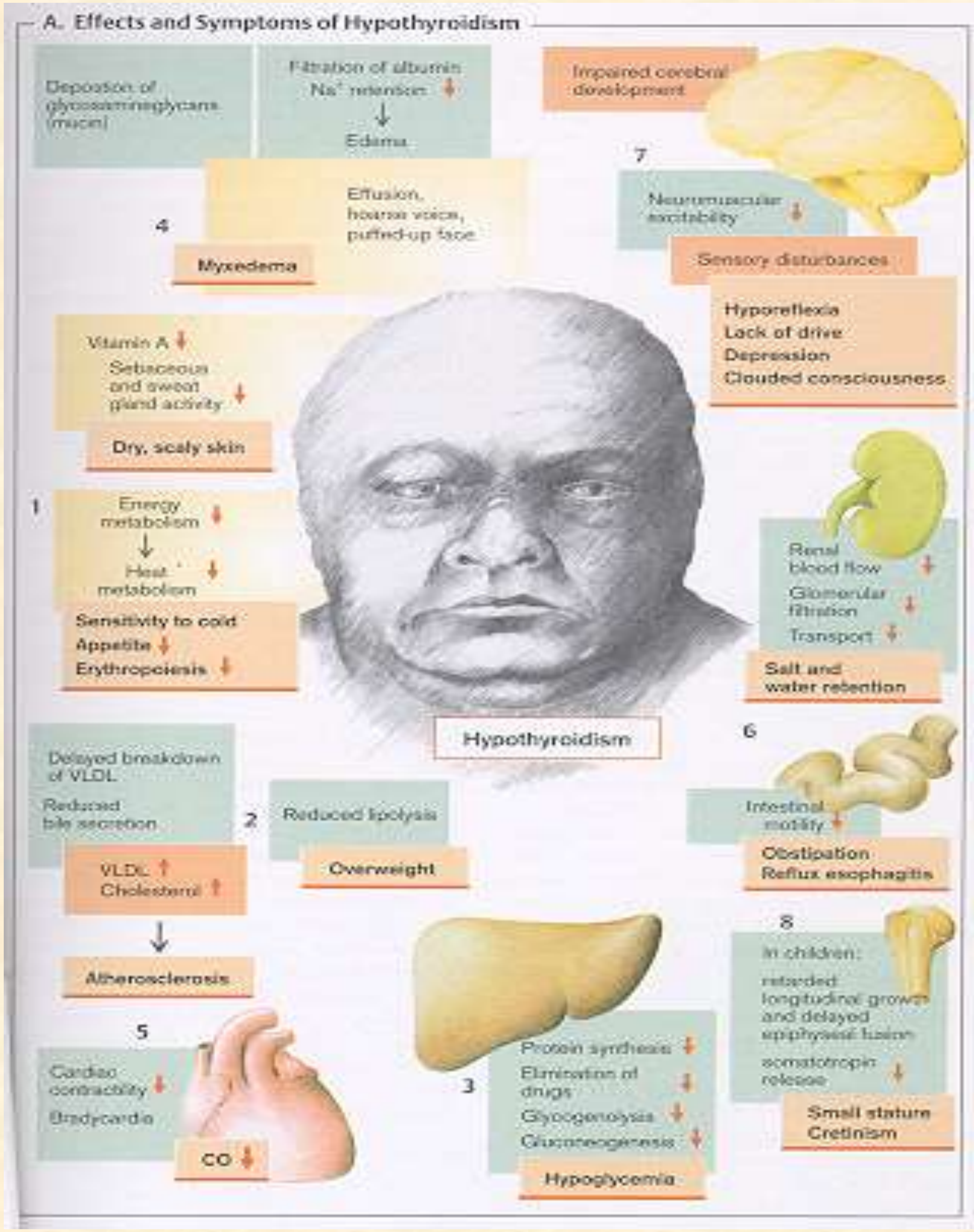

A. Effects and Symptoms of Adrenocortical Hormone Excess
Polycythemia, leukocytosis, eosinopenia
1
Gluconeogenesis
Hyperglycemia
2
Diabetes
Lipolysis
Free fatty acids
Ketone bodies
VLDL
3
Lipogenesis
Insulin
Lymphopenia, inhibition of immune defenses
4
Susceptibility to infection
Neuropsychological disorders
Redistribution of fat tissue
Moon face
'Buffalo' hump
Truncal obesity
Protein breakdown
CaHPO4
Osteoporosis
5
Thrombocytosis
Clotting
6
Atherosclerosis
Muscular weakness: thin limbs
Striae
Purpura
Delayed wound healing
in children:
Growth
Blood pressure
Sensitization to catecholamines
Cardiac output
Peripheral resistance
7
Gastric acid and pepsin secretion
Mucus secretion
8
Gastric and duodenal ulcers
Transport in distal nephron
Na+
H+
K+
Na+
Mg2+
H+
K+
10
Neuromuscular excitability
Androgens
9
Hirsutism, amenorrhea
Precocious pseudopuberty
Electrolyte disturbances
Renal damage

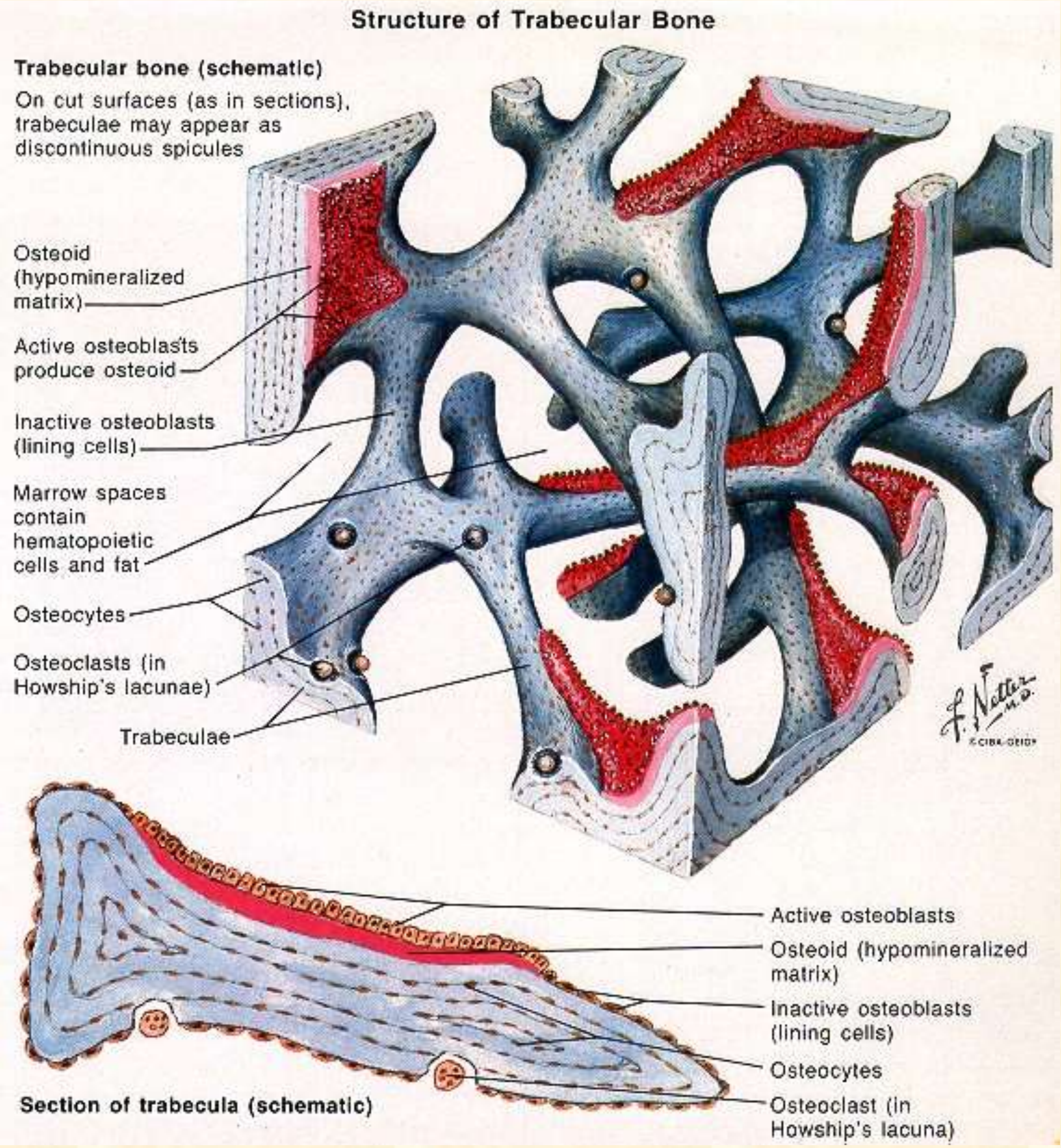

A. Effects and Symptoms of Adrenocortical Hormone Deficiency
Decreased gluconeogenesis
Increased glycolysis
1
Hypoglycemia
Proopiomelanocortin ↑
ACTH ↑
3
MSH ↑
Melanotropic effect
Brown discoloration of the skin
Insulin ↓
Epinephrine
Sweating
2
Lipolysis
Protein breakdown
Tachycardia
Muscle weakness
Weight loss
Renal Na⁺ retention ↓
Na⁺ ↓
4
Anemia, neutropenia, eosinophilia, thrombopenia, lymphocytosis
Blood pressure ↓
Hypotonic dehydration
5
GFR ↓
Hydrochloric acid ↓
6
Gastrointestinal infections
Acidosis, hyperkalemia, hypermagnesemia
Reduced catecholamine sensitivity
Electrolyte disturbances
7
Androgen deficiency
Abnormal action potential generation and conduction in the heart
Pubic hair ↓
8
Neuromuscular excitability ↓


A. Androgen Excess and Deficiency
Gonadoliberin (GnRH)
Cachexia, damage to hypothalamus, genetic defects
Prolactin
Persistently increased GnRH analogues
1
Damage to pituitary
Lutropin (LH)
Follitropin (FSH)
Leydig cells
Sertoli cells of the seminiferous tubules
Testosterone
3
Inhibin
ABP
Spermiogenesis
Tumors
2
Blut
Adreno-genital syndrome
Testosterone
Testicular damage, genetic damage
Lumen
Iatrogenic supply
Receptor defect
DHT
Reductase deficiency
Penis, scrotum, seminiferous tubules
Prostate, seminal vesicles
Receding forehead
Growth of beard
Female voice
Male voice
Absence of male hair
Hair on chest
Libido and aggressive behavior
Bone growth
Electrolyte retention
Epiphyseal fusion
Protein synthesis
Muscle growth
Erythropoiesis
Skin thickness
Sebaceous glands
HDL
Amenorrhea
Feminine fat distribution
Rhomboid pubic hair
Hypotrophic genitals
Hyper-trophic clitoris
Feminization of the male due to testosterone deficiency
Infertility
Masculinization of the female due to testosterone excess


A. Causes of Hypothyroidism, Hyperthyroidism and Goitre
1
Iodine deficiency
Food
Stress
Catecholamines
TRH
Hypothalamic lesion
Iodine excess, rhodanide, etc.
Estrogens
Dopamine
Somatostatin
Glucocorticoids
2
Carrier defect
Na⁺
TSH
Pituitary insufficiency
Autoimmune disease
TSI
Peroxidase
3
Peroxidase deficiency
Cell proliferation
Thyroglobulin
4
5
OH
TG
-DJT
-MJT
Cell damage by tumor, inflammation etc.
Goitre
Coupling
6
Proteolysis
Defective conversion
7
8
Genetic defect, iodine excess, lithium
Receptor defect
Storage
Follicle epithelial cell of the thyroid
Colloid
Target organs, e.g. heart


A. Effects and Symptoms of Hyperthyroidism
3
Stimulation of glycogenolysis, gluconeogenesis
Hyperglycemia
4
Accelerated growth in children
Bone metabolism ↑
Osteoporosis
Hypercalcemia
Hypercalciuria
2
Accelerated breakdown of VLDL and LDL, stimulation of bile secretion
VLDL ↓
Cholesterol ↓
7
Neuromuscular excitability ↑
Hyperreflexia
Tremor
Insomnia
Muscle weakness
1
Energy metabolism ↑
O_2 consumption
Heat metabolism ↑
Hyperthermia
Sweating
Lipolysis
Proteolysis
Hyperventilation
Weight loss
Hyperlipidacidemia
Decrease in muscle mass
Muscle weakness
8
In Grave's disease: retrobulbar inflammation
Exophthalmos
Tear flow
Light sensitivity
Diplopia
Breakdown of steroids and drugs
Hyperthyroidism
5
Stimulation of the heart
Oxygen consumption ↑
Vasodilation
Erythropoiesis
Tachycardia
Atrial fibrillation
Cardiac contractility ↑
Stroke volume ↑
CO ↑
Blood pressure amplitude ↑
GFR ↑
RPF ↑
Sodium reabsorption ↑
Stimulation of intestinal musculature
Diarrhea


A. Effects and Symptoms of Hypothyroidism
Impaired cerebral development
7
Neuromuscular excitability
Sensory disturbances
Hyporeflexia
Lack of drive
Depression
Clouded consciousness
Effusion, hoarse voice, puffed-up face
4
Myxedema
Edema
Vitamin A
Sebaceous and sweat gland activity
Dry, scaly skin
1
Energy metabolism
Heat metabolism
Sensitivity to cold
Appetite
Erythropoiesis
Renal blood flow
Glomerular filtration
Transport
Salt and water retention
Hypothyroidism
6
Delayed breakdown of VLDL
Reduced bile secretion
2
Reduced lipolysis
Overweight
VLDL
Cholesterol
Atherosclerosis
Intestinal motility
Obstipation
Reflux esophagitis
8
In children:
retarded longitudinal growth and delayed epiphyseal fusion
somatotropin release
Small stature
Cretinism
5
Cardiac contractility
Bradycardia
CO
3
Protein synthesis
Elimination of drugs
Glycogenolysis
Gluconeogenesis
Hypoglycemia


Structure of Trabecular Bone
Trabecular bone (schematic)
On cut surfaces (as in sections), trabeculae may appear as discontinuous spicules
Osteoid (hypomineralized matrix)
Active osteoblasts produce osteoid
Inactive osteoblasts (lining cells)
Marrow spaces contain hematopoietic cells and fat
Osteocytes
Osteoclasts (in Howship's lacunae)
Trabeculae
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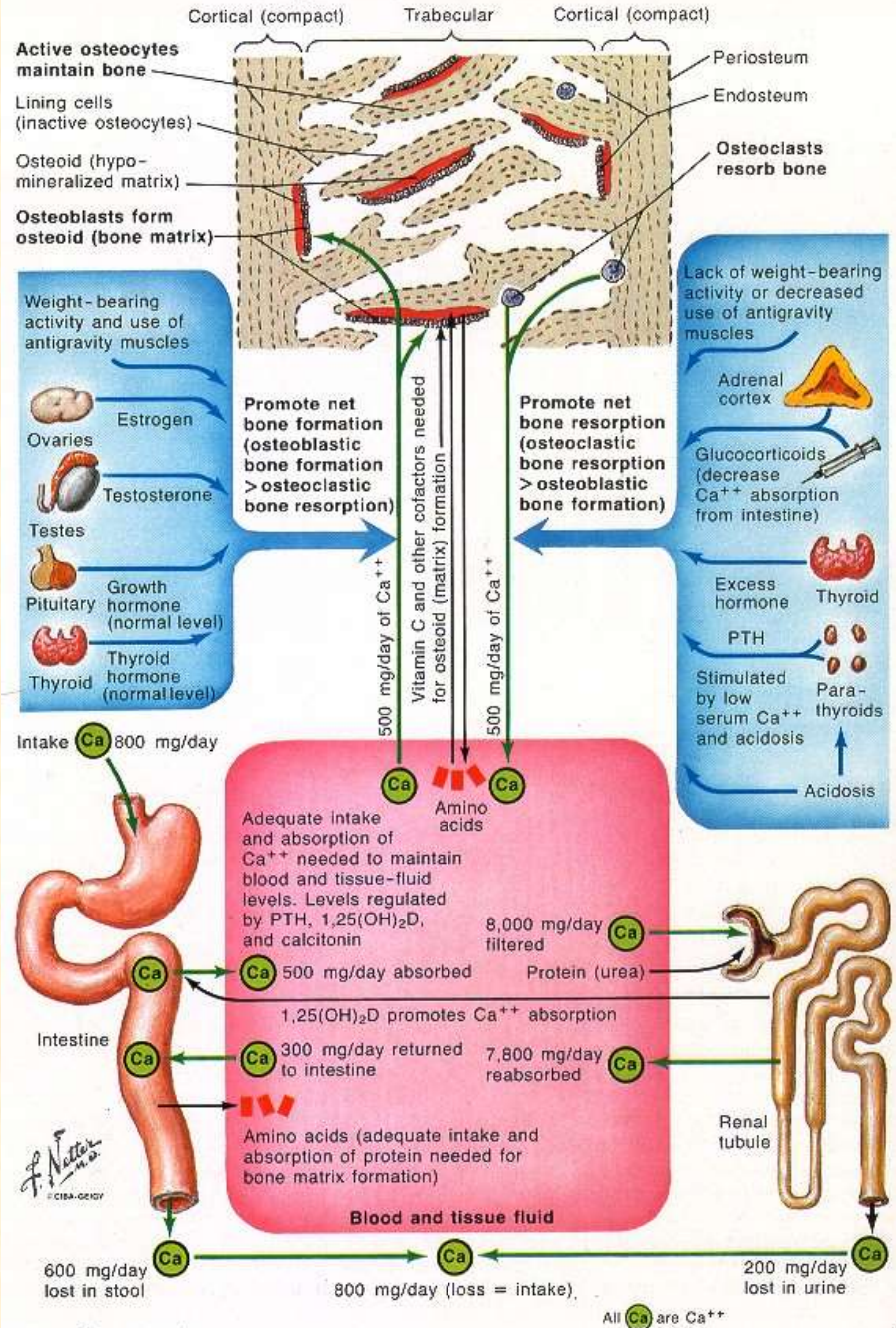
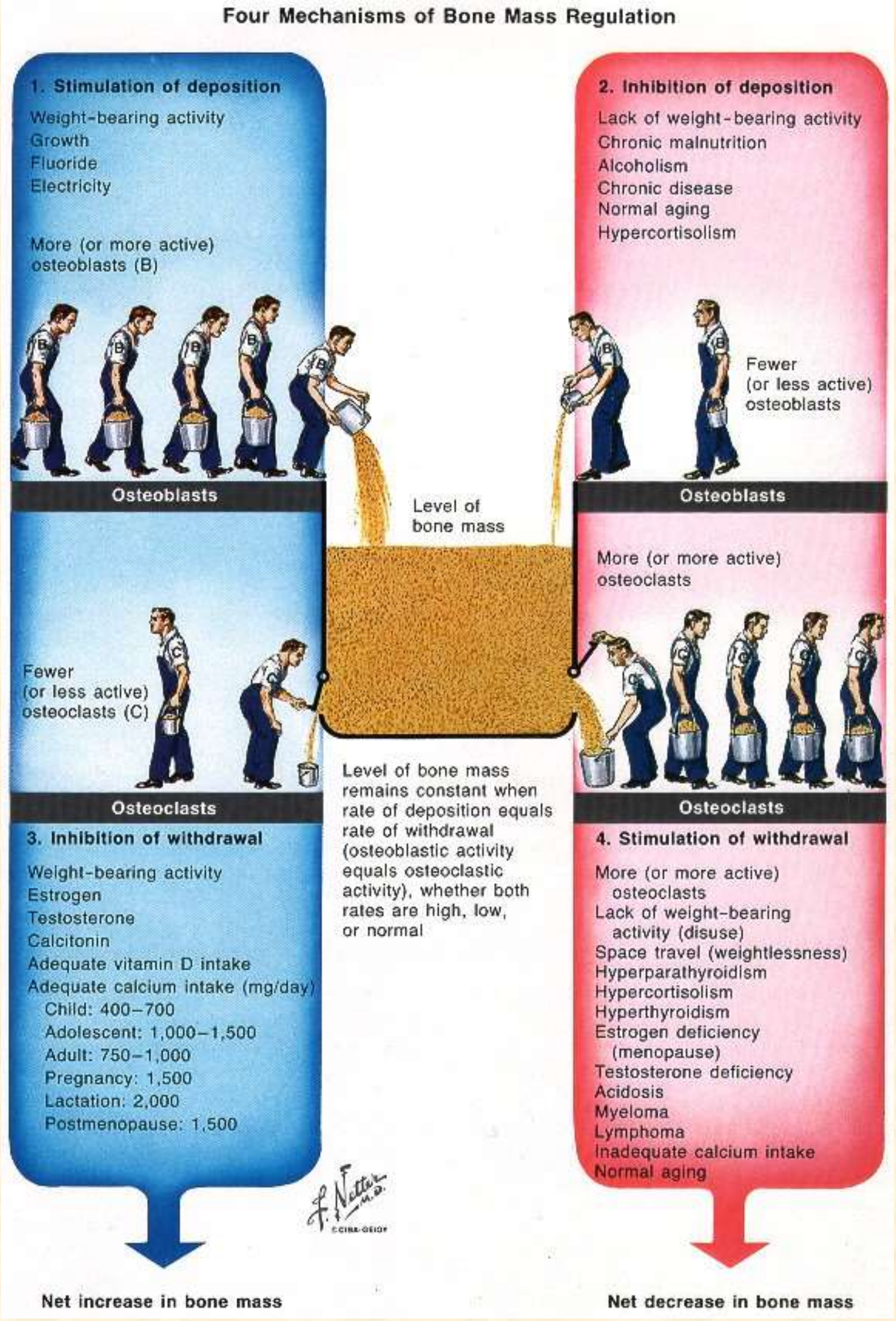
Active osteoblasts
Osteoid (hypomineralized matrix)
Inactive osteoblasts (lining cells)
Osteocytes
Osteoclast (in Howship's lacuna)
Section of trabecula (schematic)

Dynamics of Bone Homeostasis




Four Mechanisms of Bone Mass Regulation
1. Stimulation of deposition
Weight-bearing activity
Growth
Fluoride
Electricity
More (or more active) osteoblasts (B)
2. Inhibition of deposition
Lack of weight-bearing activity
Chronic malnutrition
Alcoholism
Chronic disease
Normal aging
Hypercortisolism
Fewer (or less active) osteoblasts
Osteoblasts
Osteoblasts
Level of bone mass
More (or more active) osteoclasts
Fewer (or less active) osteoclasts (C)
Level of bone mass remains constant when rate of deposition equals rate of withdrawal (osteoblastic activity equals osteoclastic activity), whether both rates are high, low, or normal
Osteoclasts
Osteoclasts
3. Inhibition of withdrawal
Weight-bearing activity
Estrogen
Testosterone
Calcitonin
Adequate vitamin D intake
Adequate calcium intake (mg/day)
Child: 400–700
Adolescent: 1,000–1,500
Adult: 750–1,000
Pregnancy: 1,500
Lactation: 2,000
Postmenopause: 1,500
4. Stimulation of withdrawal
More (or more active) osteoclasts
Lack of weight-bearing activity (disuse)
Space travel (weightlessness)
Hyperparathyroidism
Hypercortisolism
Hyperthyroidism
Estrogen deficiency (menopause)
Testosterone deficiency
Acidosis
Myeloma
Lymphoma
Inadequate calcium intake
Normal aging
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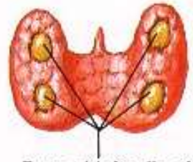
Net increase in bone mass
Net decrease in bone mass

Regulation of Calcium and Phosphate Metabolism

	Parathyroid hormone (PTH) (peptide)	$1,25(OH)_2D$ (steroid)	Calcitonin (peptide)
Hormone	 From chief cells of parathyroid glands	 From proximal tubule of kidney	 From parafollicular cells of thyroid gland
Factors stimulating production	Decreased serum Ca^{++}	Elevated PTH Decreased serum Ca^{++} Decreased serum P_i	Elevated serum Ca^{++}
Factors inhibiting production	Elevated serum Ca^{++} Elevated $1,25(OH)_2D$	Decreased PTH Elevated serum Ca^{++} Elevated serum P_i	Decreased serum Ca^{++}
End-organs for hormone action:  Intestine	No direct effect Acts indirectly on bowel by stimulating production of $1,25(OH)_2D$ in kidney	Strongly stimulates intestinal absorption of Ca^{++} and P_i	?
 Kidney	Stimulates $25(OH)D\text{-}1\alpha\text{-}OH_{ase}$ in mitochondria of proximal tubular cells to convert $25(OH)D$ to $1,25(OH)_2D$ Increases fractional reabsorption of filtered Ca^{++} Promotes urinary excretion of P_i	?	?
 Bone	Stimulates osteoclastic resorption of bone Stimulates recruitment of preosteoclasts	Strongly stimulates osteoclastic resorption of bone	Inhibits osteoclastic resorption of bone ? Role in normal human physiology
Net effect on calcium and phosphate concentrations in extracellular fluid and serum	**Increased serum calcium** **Decreased serum phosphate**	**Increased serum calcium** **Increased serum phosphate**	**Decreased serum calcium (transient)**

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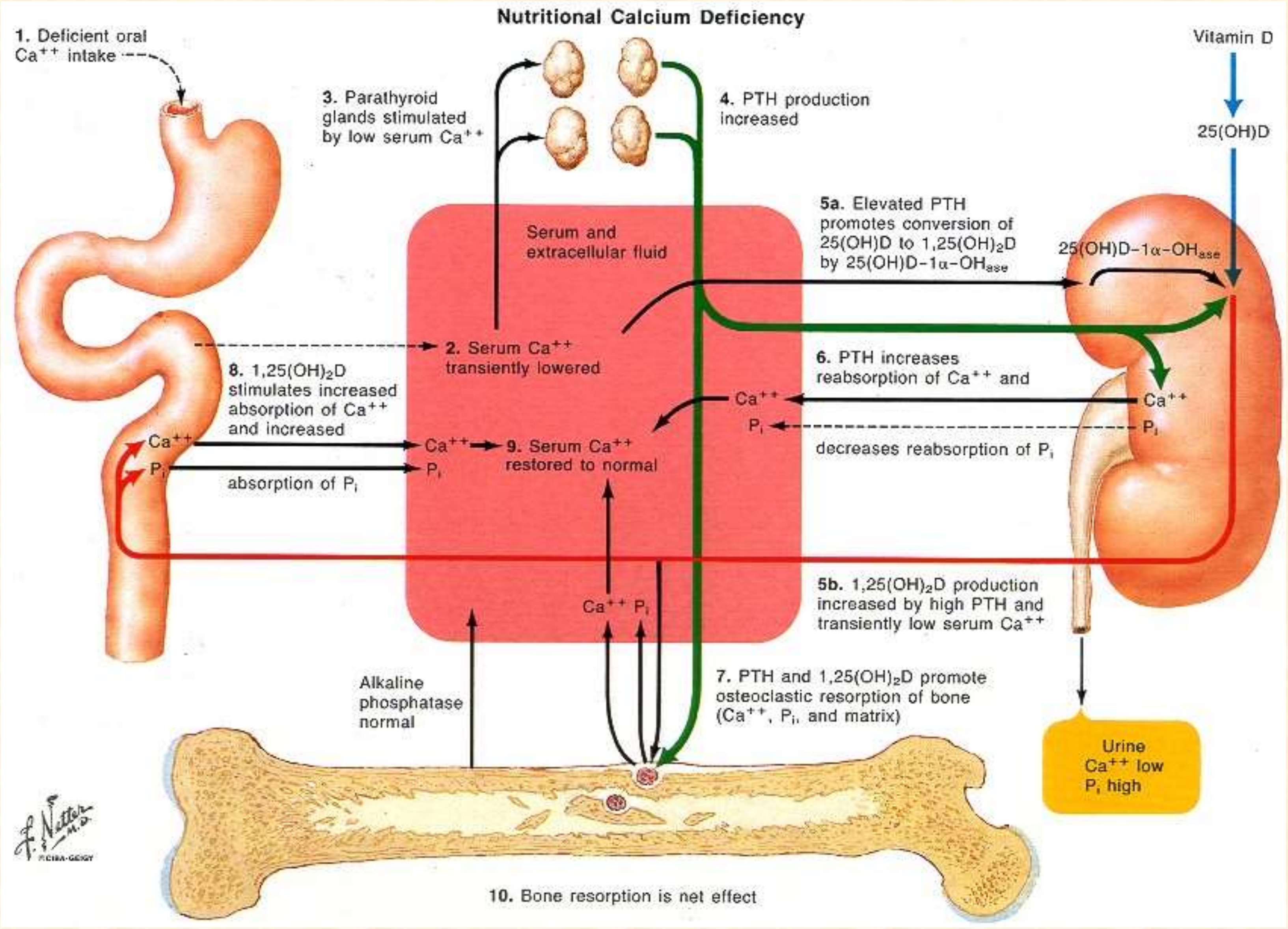
Nutritional Calcium Deficiency
1. Deficient oral Ca++ intake
3. Parathyroid glands stimulated by low serum Ca++
4. PTH production increased
Vitamin D
25(OH)D
Serum and extracellular fluid
5a. Elevated PTH promotes conversion of 25(OH)D to 1,25(OH)2D by 25(OH)D-1α-OHase
25(OH)D-1α-OHase
2. Serum Ca++ transiently lowered
6. PTH increases reabsorption of Ca++ and
8. 1,25(OH)2D stimulates increased absorption of Ca++ and increased
Ca++
Pi
Ca++
Pi
decreases reabsorption of Pi
Ca++
Pi
Ca++
9. Serum Ca++ restored to normal
Pi
absorption of Pi
5b. 1,25(OH)2D production increased by high PTH and transiently low serum Ca++
Ca++ Pi
7. PTH and 1,25(OH)2D promote osteoclastic resorption of bone (Ca++, Pi, and matrix)
Alkaline phosphatase normal
Urine
Ca++ low
Pi high
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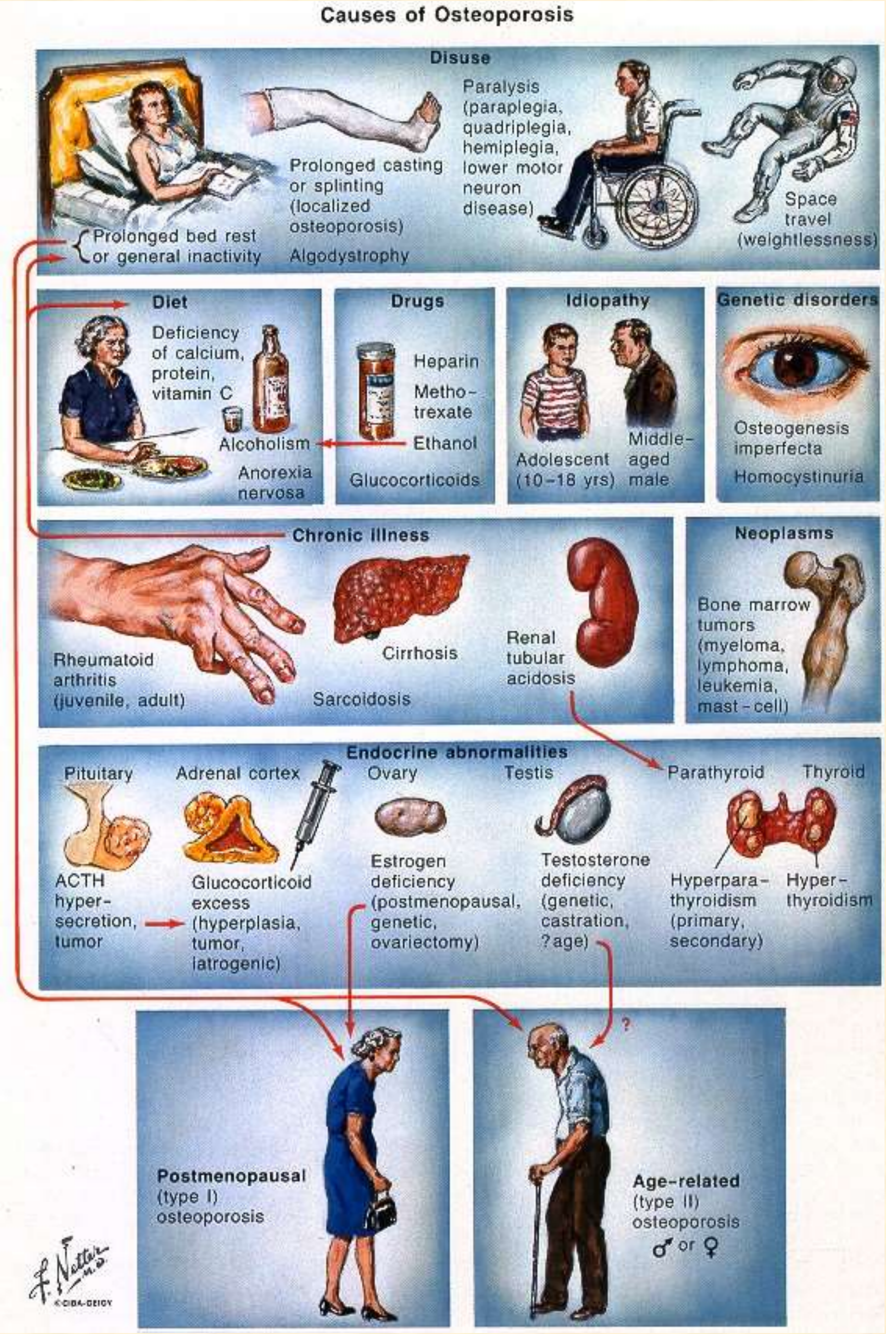
10. Bone resorption is net effect


Causes of Osteoporosis
Disuse
Prolonged bed rest or general inactivity
Prolonged casting or splinting (localized osteoporosis)
Algodystrophy
Paralysis (paraplegia, quadriplegia, hemiplegia, lower motor neuron disease)
Space travel (weightlessness)
Diet
Deficiency of calcium, protein, vitamin C
Alcoholism
Anorexia nervosa
Drugs
Heparin
Metho-trexate
Ethanol
Glucocorticoids
Idiopathy
Adolescent (10–18 yrs)
Middle-aged male
Genetic disorders
Osteogenesis imperfecta
Homocystinuria
Chronic illness
Rheumatoid arthritis (juvenile, adult)
Cirrhosis
Sarcoidosis
Renal tubular acidosis
Neoplasms
Bone marrow tumors (myeloma, lymphoma, leukemia, mast-cell)
Endocrine abnormalities
Pituitary
Adrenal cortex
Ovary
Testis
Parathyroid
Thyroid
ACTH hyper-secretion, tumor
Glucocorticoid excess (hyperplasia, tumor, iatrogenic)
Estrogen deficiency (postmenopausal, genetic, ovariectomy)
Testosterone deficiency (genetic, castration, ?age)
Hyperpara-thyroidism (primary, secondary)
Hyper-thyroidism
?
Postmenopausal (type I) osteoporosis
Age-related (type II) osteoporosis ♂ or ♀
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