Endocrine System

Specialized tissues and glands that produce and release **hormones**

Hormones regulate many activities and allow for communication between cells

Endocrine signaling

Hormone released into the blood

Hormone circulates in the blood to its **target cell**

Hormone binds to specific receptor of the target cell

Hormone affects the activity of its target cell

Major Classes of Hormones

Protein

Water-soluble
Most circulate freely within the blood
e.g. antidiuretic hormone, growth hormone, insulin, oxytocin

Amine

Water-soluble
Most circulate freely within the blood
e.g. norepinephrine, thyroid hormones

Steroid

Relatively small, lipid-soluble
Need to be carried in the blood
e.g. testosterone, estrogens, cortisol

Hormone-like Substances: Prostaglandins

Prostaglandins ARE NOT hormones (function like hormones)

Fatty acid derivatives that are synthesized by most tissues of the body

Cyclooxygenases (COX-1 and COX-2) needed to synthesize prostaglandins

Synthesized from the following fatty acids:

Gamma linolenic acid (omega-6 fatty acid)

Used to synthesize series-1 prostaglandins (**PG-1**) – e.g. PGH_1

Arachidonic acid (omega-6 fatty acid)

Used to synthesize series-2 prostaglandins (**PG-2**) – e.g. PGE_2

Eicosapentaenoic acid (omega-3 fatty acid)

Used to synthesize series-3 prostaglandins (**PG-3**) – e.g. PGA_3

Series 1 and 3 prostaglandins

e.g. dilate airways, reduce inflammation and pain

Series 2 prostaglandins

e.g. constrict airways, cause inflammation, pain and fever

Why are prostaglandins not hormones?

Are NOT released into the blood (because of their very short half-life)

Do not use endocrine signaling for communication

Signaling must be paracrine or autocrine

Paracrine signalling

Release of a substance from a cell that acts on a nearby cell

Autocrine signaling

Release of a substance from a cell that acts on that same cell

Hormonal Regulation Mechanisms

Feedback Mechanisms

Negative Feedback

Positive Feedback

Loss of Negative Feedback

Hyposecretion

Hypersecretion

Patterns of Regulation

Non-hormonal

Neural

Hormonal

Patterns of Hormone Release

Chronic

Acute

Cyclic

Feedback Mechanisms

Negative feedback

Most common feedback mechanism

Homeostatic

Maintenance of the normal range in response to a deviation from normal range

Hormones are regulated in a negative feedback manner

Allows for normal blood hormone level

Allows hormones to maintain homeostasis

Hormone will stimulate its own release when level is too low

Hormone will inhibit its own release when level is too high

Positive feedback

Non-homeostatic (i.e. does not maintain homeostasis)

Increase above normal range that is necessary to facilitate a “special event”

Some hormones are released in a positive feedback manner

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Patterns of Hormone Release

Chronic

Acute

Cyclic

Loss of Negative Feedback

Hyposecretion
Too little secretion

Leads to loss of homeostasis

Hypersecretion
Too much secretion

Leads to loss of homeostasis

Hormonal Regulation Mechanisms

Feedback Mechanisms

Negative Feedback

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Chronic

Acute

Cyclic

Patterns of Regulation

What regulates the release of a hormone

Non-hormonal

Something (other than a hormone) regulates release of a hormone
e.g. low blood glucose stimulates the release of cortisol

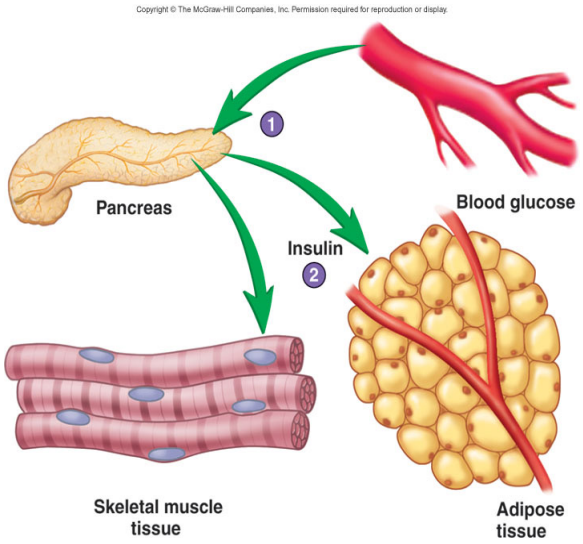
Hormonal

Hormone from one gland regulates release of a hormone from another gland
e.g. adrenocorticotropic hormone stimulates the release of cortisol

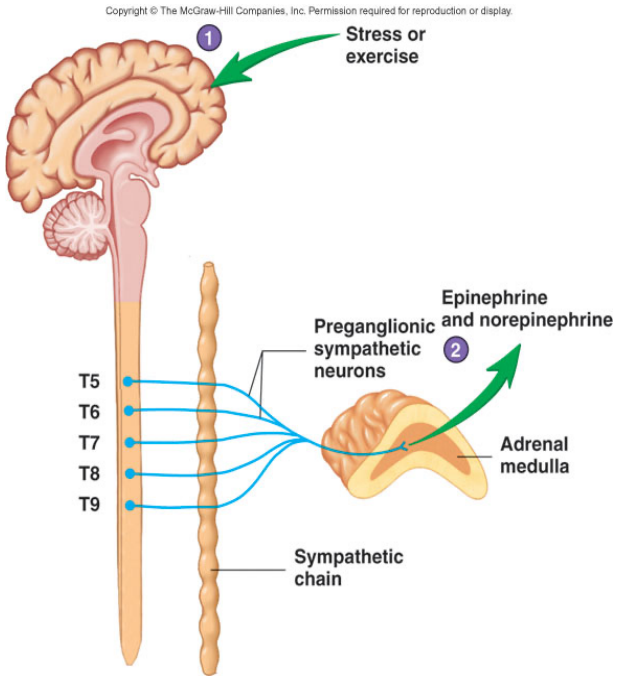
Neural

Nervous system regulates release of a hormone
e.g. sympathetic nervous system stimulates the release of epinephrine

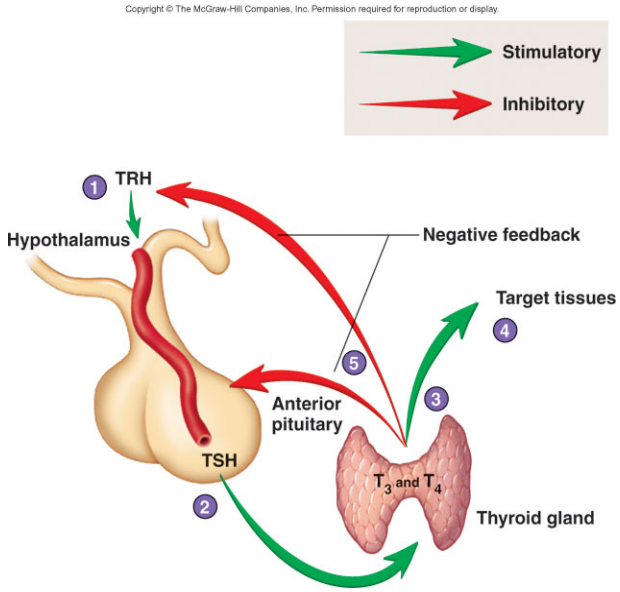
Major Patterns of Hormone Regulation



Non-hormonal



Neural



Hormonal

Hormonal Regulation Mechanisms

Feedback Mechanisms

Negative Feedback

Positive Feedback

Loss of Negative Feedback

Hyposecretion

Hypersecretion

Patterns of Regulation

Non-hormonal

Neural control

Hormonal

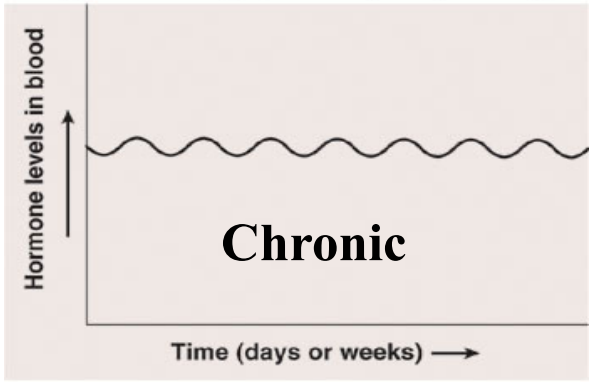
Patterns of Hormone Release

Chronic

Acute

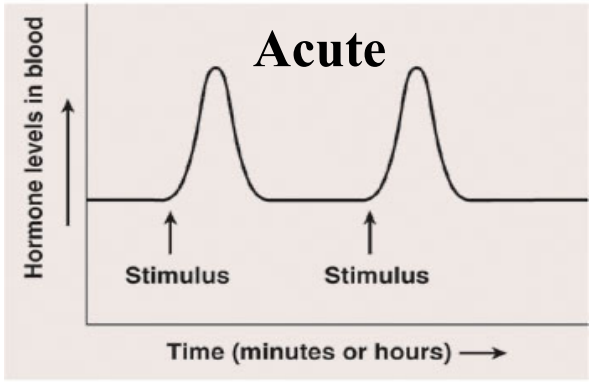
Cyclic

Patterns of Hormone Secretion



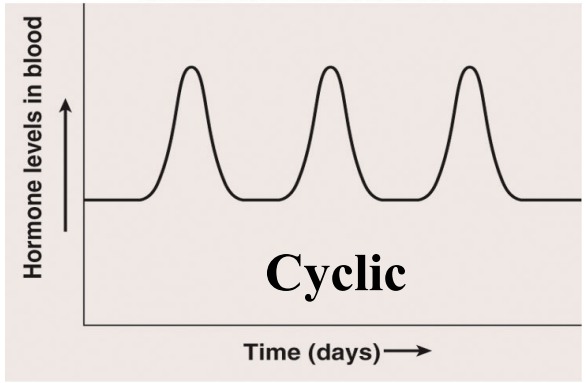
Chronic hormone regulation.

Release of hormone is relatively constant
Concentration of circulating hormone is relatively constant
e.g. release of thyroid hormones throughout the day



Acute hormone regulation.

Release of hormone in response to an "unplanned" stimulus
Rapid, transient increase in circulating hormone
e.g. increased release of growth hormone in response to exercise



Cyclic hormone regulation.

Release of hormone in response to a "planned" stimulus
At set times during the day or week or month
Increase or decrease in circulating hormone at set times
e.g. increased release of growth hormone during deep sleep

Determining what gland is dysfunctioning
(i.e. determine what gland has lost negative feedback)

Assume gland X releases hormone X

Assume gland Y releases hormone Y

Assume release of hormone X stimulates release of hormone Y

Therefore, gland X regulates gland Y

Therefore, given negative feedback (i.e. to maintain homeostasis)

Decreased level of hormone Y should stimulate gland X

Increases the release of hormone X

Increases the level of hormone Y towards normal

Increased level of hormone Y should inhibit gland X

Decreases the release of hormone X

Decreases the level of hormone Y towards normal

Scenario 1: Dysfunction of gland X

Abnormal blood work

Increased level of hormone X (i.e. hypersecretion of hormone X)

Increased level of hormone Y (i.e. hypersecretion of hormone Y)

Reasoning it is dysfunction of gland X

A high level of hormone Y should inhibit gland X

That should decrease the level of hormone X

However, the level of hormone X is high

Why?

Gland X lost negative feedback

Causes increased level of hormone Y

Scenario 2: Dysfunction of gland Y

Abnormal Blood Work

Decreased level of hormone X (i.e. hyposecretion of hormone X)

Increased level of hormone Y (i.e. hypersecretion of hormone Y)

Reasoning it is dysfunction of gland Y

A high level of hormone Y should inhibit gland X

That should decrease the level of hormone X

The level of hormone X is indeed low

Therefore gland X is functioning properly

Should decrease the level of hormone Y

However, the level of hormone Y is high

Why?

Gland Y lost negative feedback

Causes increased level of hormone Y

Scenario 3: Dysfunction of gland X

Abnormal blood work

Decreased level of hormone X (i.e. hyposecretion of hormone X)

Decreased level of hormone Y (i.e. hyposecretion of hormone Y)

Reasoning it is dysfunction of gland X

A low level of hormone Y should stimulate gland X

That should increase the level of hormone X

However, the level of hormone X is low

Why?

Gland X lost negative feedback

Causes decreased level of hormone Y

Scenario 4: Dysfunction of gland Y

Abnormal blood work

Increased level of hormone X (i.e. hypersecretion of hormone X)

Decreased level of hormone Y (i.e. hyposecretion of hormone Y)

Reasoning it is dysfunction of gland Y

A low level of hormone Y should stimulate gland X

That should increase the level of hormone X

The level of hormone X is indeed high

Therefore gland X is functioning properly

Should increase the level of hormone Y

However, the level of hormone Y is low

Why?

Gland Y lost negative feedback

Causes decreased level of hormone Y

Is it the gland in question or something else?

Primary condition

If a hormone is causing signs and symptoms and there is something wrong with the gland that is producing that hormone . . . It is a **primary condition**

Secondary condition

If a hormone is causing signs and symptoms and there is nothing wrong with the gland that is producing that hormone . . . But instead something else is causing that gland to be dysfunctional . . . It is a **secondary condition**

Endocrine Glands and Specialized Structures

Hypothalamus

Pituitary Gland (Posterior Pituitary and Anterior Pituitary)

Thyroid Gland

Parathyroid Glands

Adrenal Gland

Pancreas

Hypothalamus

Directly or indirectly regulates most endocrine activity

Produces eight neurohormones

A neurohormone is a hormone produced by neurons

Six releasing and inhibiting hormones that regulate the anterior pituitary

1. **Gonadotropin releasing hormone (GnRH)**
   Stimulates production and release of luteinizing hormone
   Stimulates production and release of follicle stimulating hormone
2. **Prolactin inhibiting hormone (PRH) / Dopamine**
   Stimulates production and release of prolactin
3. **Growth hormone releasing hormone (GHRH)**
   Stimulates production and release of growth hormone
4. **Growth hormone inhibiting hormone (GHIH)**
   Inhibits production and release of growth hormone
5. **Thyrotropin releasing hormone (TRH)**
   Stimulates production and release of thyroid stimulating hormone
6. **Corticotropin releasing hormone (CRH)**
   Stimulates production and release of adrencorticotropic hormone
7. **Oxytocin**
8. **Antidiuretic hormone (ADH)**

Endocrine Glands and Specialized Structures

Hypothalamus

Pituitary Gland (Posterior Pituitary and Anterior Pituitary**)**

Thyroid Gland

Parathyroid Glands

Adrenal Gland

Pancreas

Pituitary Gland

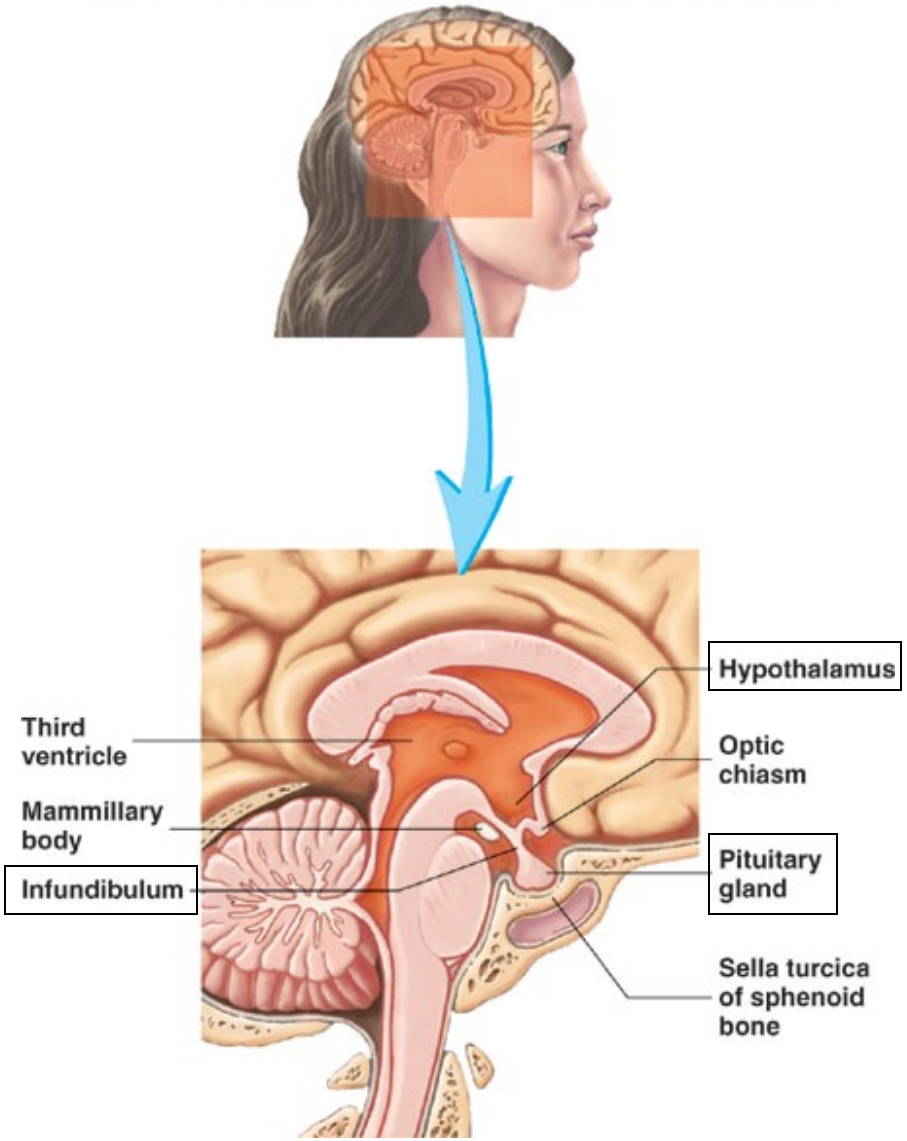
Connected to the hypothalamus by infundibulum / pituitary stalk

Composed of two separate lobes (function independently of each other)

Posterior pituitary / Neurohypophysis

Anterior pituitary / Adenohypophysis

Hypothalamus



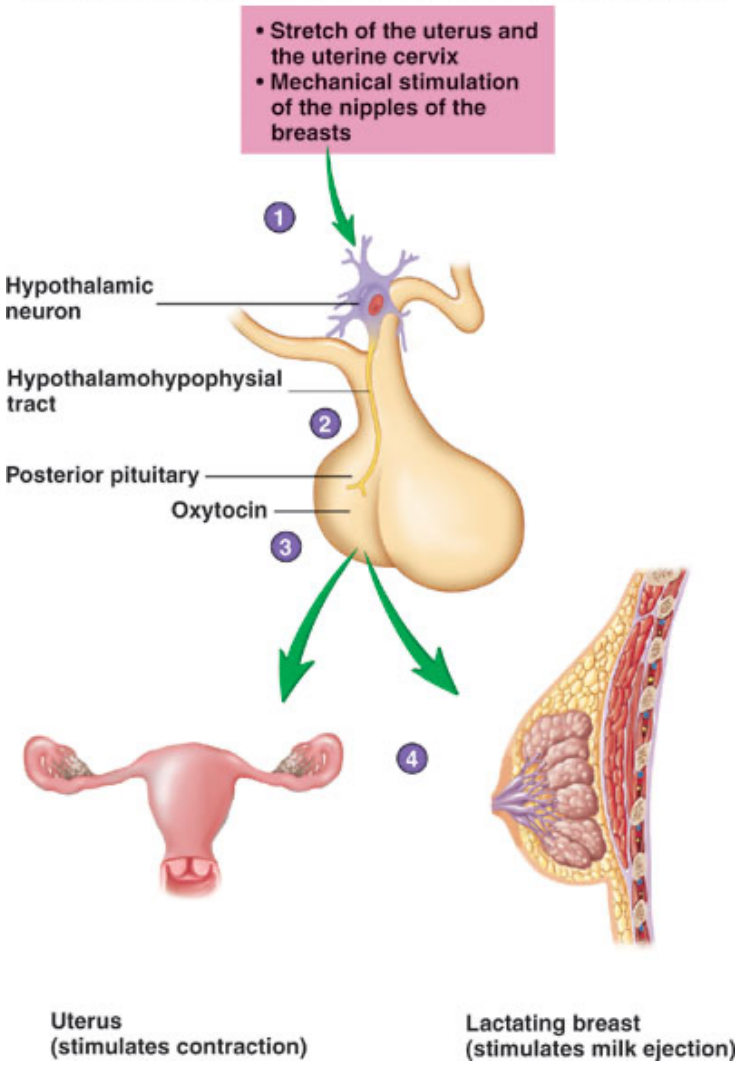
Posterior Pituitary

Neurohormones released (not produced) by the posterior pituitary

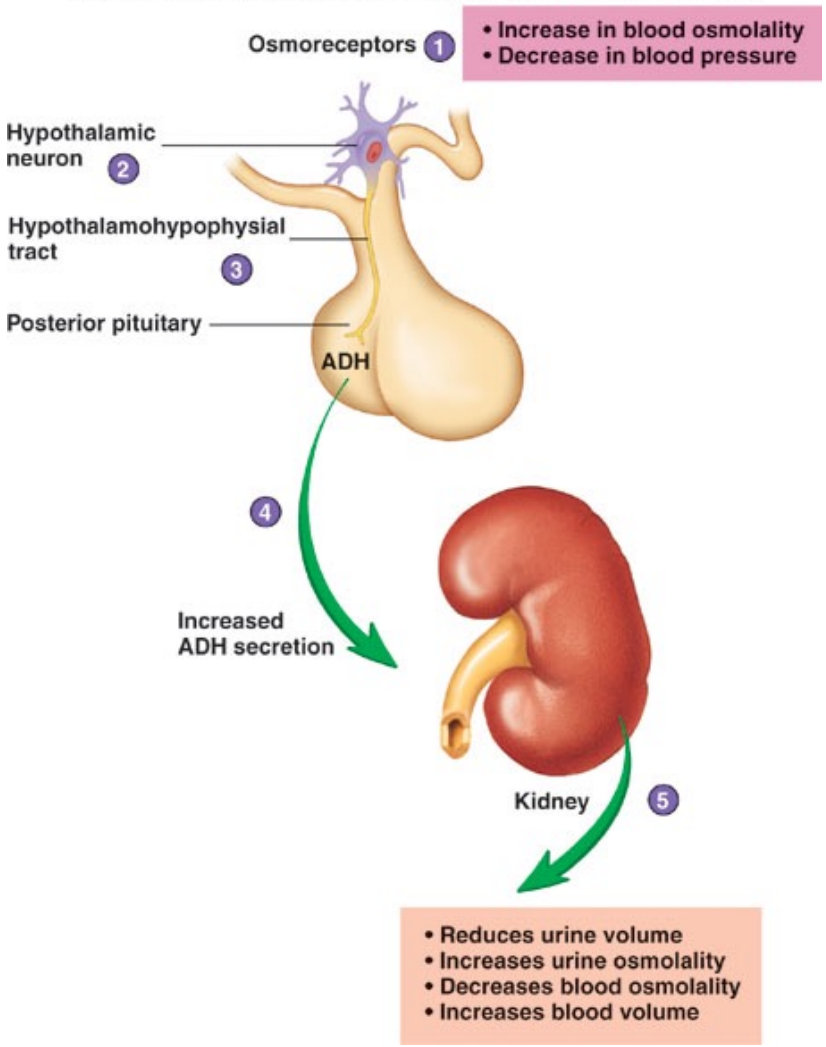
Oxytocin

Antidiuretic Hormone (ADH) / Vasopressin

Oxytocin



ADH / Vasopressin



Oxytocin

- Functions
  - Stimulates uterine contractions
  - Stimulates release of milk from lactating breast
  - Facilitates sperm transport in male reproductive tract
  - Plays a role in bonding (“the love hormone”)
- What stimulates oxytocin
  - Dilation of cervix
  - Distension of uterus
  - Suckling of breasts
  - Intimacy
- What inhibits oxytocin
  - None known
- Effects of oxytocin hypersecretion
  - None known
- Effects of oxytocin hyposecretion
  - Difficulty with birthing and lactation
  - Decreased sperm transport in male reproductive tract

ADH / Vasopresin

Functions
- Regulates blood osmolarity (most important function)
  - Regulates transport of water into the blood by kidneys
- Regulates blood pressure (minor function)
  - Regulates transport of water into the blood by kidneys
  - Regulates blood vessel tone

What stimulates ADH
- High blood osmolarity
- Low blood volume
- Low blood pressure
- Stress

What inhibits ADH
- Low blood osmolarity
- High blood volume
- High blood pressure

ADH

Effects of ADH hypersecretion

Syndrome of inappropriate ADH (SIADH)

Large volume of water transported into the blood

Large decrease in blood osmolarity

Large increase in blood volume

Large increase in blood pressure

Effects of ADH hyposecretion

Diabetes insipidus

Small volume of water transported into the blood

Large increase in blood osmolality

Large decrease in blood volume

Large decrease in blood pressure

Endocrine Glands and Specialized Structures

Hypothalamus

Pituitary Gland (Posterior Pituitary and **Anterior Pituitary)**

Thyroid Gland

Parathyroid Glands

Adrenal Gland

Pancreas

Hormones Produced and Released by the Anterior Pituitary

Gonadotropic Hormones (Luteinizing Hormone and Follicle Stimulating Hormone)

Prolactin

Growth Hormone / Somatotropin

Thyroid Stimulating Hormone

Adrenocorticotropin Hormone

Luteinizing Hormone (LH)

Functions
- Stimulates testosterone production
  - Indirectly stimulates sperm production
- Helps regulate the menstrual cycle

What stimulates LH
- GnRH
- Low blood testosterone

What inhibits LH
- Low GnRH
- High blood testosterone

Effects of LH hypersecretion
- High blood testosterone
- **Amenorrhea** (lack of a menstrual cycle)

Effects of LH hyposecretion
- Low blood testosterone (decreases sperm production)
- Decreased libido
- Erectile dysfunction
- Amenorrhea

Follicle Stimulating Hormone (FSH)

- Functions
  - Stimulates sperm production
  - Stimulates estrogen production
  - Helps regulate the menstrual cycle
- What stimulates FSH
  - GnRH
  - Low blood estrogen (females)
  - Low blood testosterone (males)
- What inhibits FSH
  - High blood estrogen
  - Inhibin (hormone released from testes and ovaries)
  - Low GnRH
  - Stress (via inhibition of GnRH)
- Effects of FSH hypersecretion
  - High blood estrogen
  - Amenorrhea
- Effects of FSH hyposecretion
  - Low blood estrogen
  - Amenorrhea
  - Decrease in sperm production

Hormones Produced and Released by the Anterior Pituitary

Gonadotropic Hormones (Luteinizing Hormone and Follicle Stimulating Hormone)

Prolactin

Growth Hormone / Somatotropin

Thyroid Stimulating Hormone

Adrenocorticotropin Hormone

Prolactin

Functions
- Stimulates milk production in lactating females
- Stimulates breast development
- Modulates the production of testosterone and sperm in males

What stimulates prolactin
- Estrogen
- Suckling of breasts
- Decreased levels of PIH

What inhibits prolactin
- PIH (continually inhibits prolactin)

Prolactin

- Effects of prolactin hypersecretion
  - Inhibits GnRH (effects via LH and FSH)
    - Low blood estrogen
      - Amenorrhea (lactational amenorrhea)
    - Low testosterone
      - Decreased libido
      - Erectile dysfunction
      - Decreased sperm production
  - **Galactorrhea** (spontaneous milk production)
- Effects of prolactin hyposecretion
  - Females
    - Lack of milk production during lactation
  - Males
    - No known effect

Hormones Produced and Released by the Anterior Pituitary

Gonadotropic Hormones (Luteinizing Hormone and Follicle Stimulating Hormone)

Prolactin

Growth Hormone / Somatotropin

Thyroid Stimulating Hormone

Adrenocorticotropin Hormone

Growth Hormone / Somatotropin

Functions
- Stimulates and regulates growth and repair of tissues
- Stimulates amino acid uptake and their synthesis into proteins
- Increases blood fatty acids via lipolysis
- Increases blood glucose via gluconeogenesis

What stimulates GH
- GHRH
- Deep sleep (*highest level during deep sleep*)
- Low blood glucose (**hypoglycemia**)
- High blood amino acids (especially arginine)
- Low blood fatty acids
- Stress
- Exercise

What inhibits GH
- GHIH
- High blood glucose (**hyperglycemia**)
- Low blood amino acids
- High blood fatty acids
- Sleep-wake transition (*lowest level shortly before waking*)

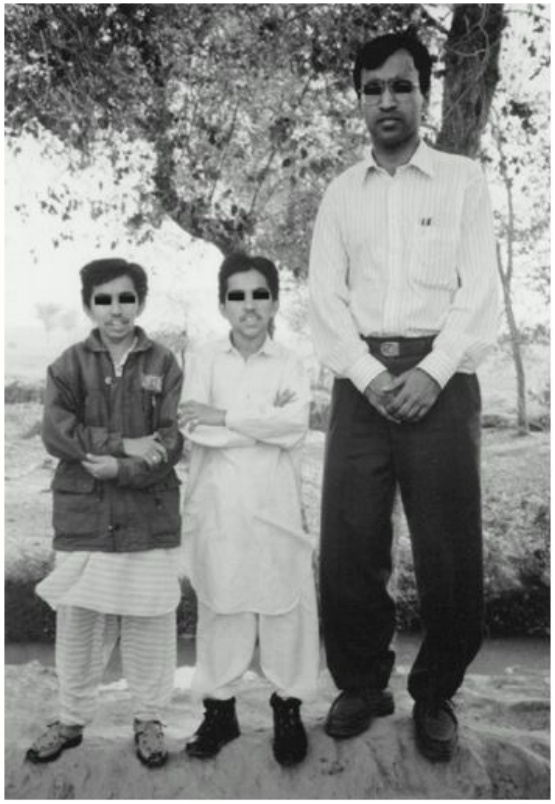
Growth Hormone / Somatotropin

Effects of growth hormone hypersecretion
- Abnormal lengthening and thickening of bones
- Abnormal organ growth (can lead to organ failure)
- Elevated blood fatty acids (can lead to cardiovascular disease)
- Elevated blood glucose (can lead to type 2 diabetes)
- Caused by (often due to benign tumor of the anterior pituitary)
  - **Pituitary gigantism / giantism**
    - Elevated growth hormone during childhood
  - **Acromegaly**
    - Elevated growth hormone after puberty

Effects of GH hyposecretion
- **Pituitary dwarfism**
  - Decreased growth hormone level during childhood
  - Proportional but short in stature
  - Can treat with human growth hormone
    - Must be done prior to growth plate ossifying
  - Accounts for approximately 30% of dwarfism
    - *Other 70% is due to **achondroplastic dwarfism***

Giantism and Acromegaly

Pituitary Dwarfism



Pituitary Dwarfism

Achondroplastic Dwarfism

Hormones Produced and Released by the Anterior Pituitary

Gonadotropic Hormones (Follicle Stimulating Hormone and Luteinizing Hormone)

Prolactin

Growth Hormone / Somatotropin

Thyroid Stimulating Hormone

Adrenocorticotropin Hormone

Thyroid Stimulating Hormone (TSH)

Function
- Stimulates thyroid hormones (thyroxine and triiodothyronine)

What stimulates TSH
- TRH
- Low thyroxine and triiodothyronine

What inhibits TSH
- Low TRH
- High thyroxine and triiodothyronine
- Stress

Effects of TSH hypersecretion
- Increased levels of thyroxine and triiodothyronine

Effects of TSH hyposecretion
- Decreased levels of thyroxine and triiodothyronine

Hormones Produced and Released by the Anterior Pituitary

Gonadotropic Hormones (Follicle Stimulating Hormone and Luteinizing Hormone)

Prolactin

Growth Hormone / Somatotropin

Thyroid Stimulating Hormone

Adrenocorticotropin Hormone

Adrenocorticotopic Hormone (ACTH)

- Functions
  - Stimulates all adrenal cortex hormones (especially cortisol)
  - Stimulates melanocytes
- What stimulates ACTH
  - CRH
  - Inflammation
  - Stress
  - Hypoglycemia
  - Sleep-wake transition (*highest level just before waking*)
  - Low aldosterone, cortisol, DHEA
- What inhibits ACTH
  - Low CRH level
  - Deep sleep (*lowest levels during deep sleep*)
  - High aldosterone, cortisol, DHEA
- Effects of ACTH hypersecretion
  - High level of adrenal cortex hormones (especially cortisol)
  - Darkened skin
- Effects of ACTH hyposecretion
  - Low level of adrenal cortex hormones (especially cortisol)

Endocrine Glands and Specialized Structures

Hypothalamus

Pituitary Glands (Anterior Pituitary and Posterior Pituitary)

Thyroid Gland

Parathyroid Glands

Adrenal Gland

Pancreas

Thyroid Gland

Thyroxine and Triiodothyronine

Thyroxine (T_4)

Contains four iodine molecules as part of its structure

Approximately 90% of produced and secreted thyroid hormone

Triiodothyronine (T_3)

Contains three iodine molecules as part of its structure

Most produced from de-iodination of T_4

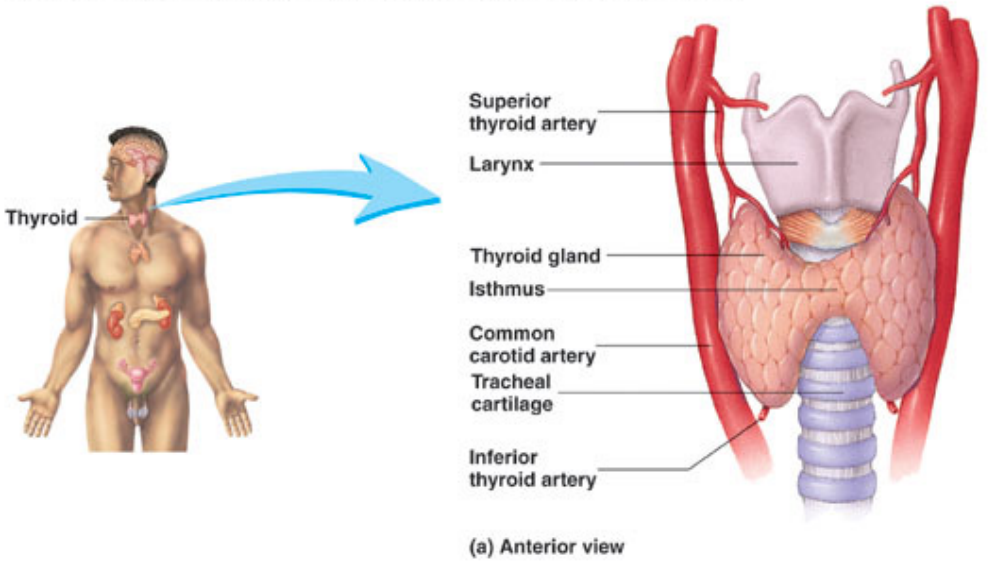
Five times more biologically active than than T_4

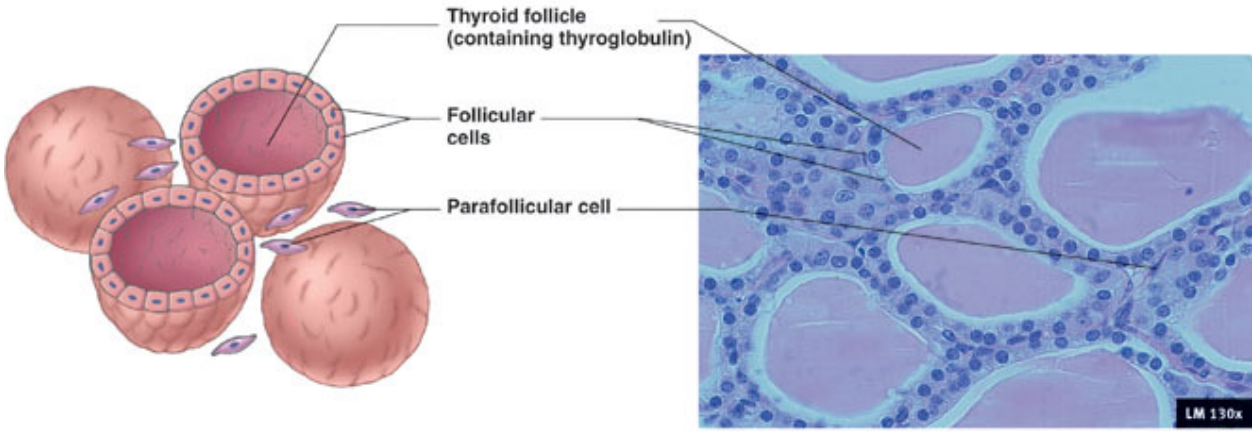
Produced in follicular cells

Stored in **colloid**

Enough stored to last approximately three months

Thyroid Gland





T_3 and T_4

Functions of T_3 and T_4
- Regulate metabolism
- Help regulate body temperature (via metabolic regulation)
- Protein synthesis
- Glucose synthesis via gluconeogenesis and glycogenolysis
- Decrease plasma cholesterol

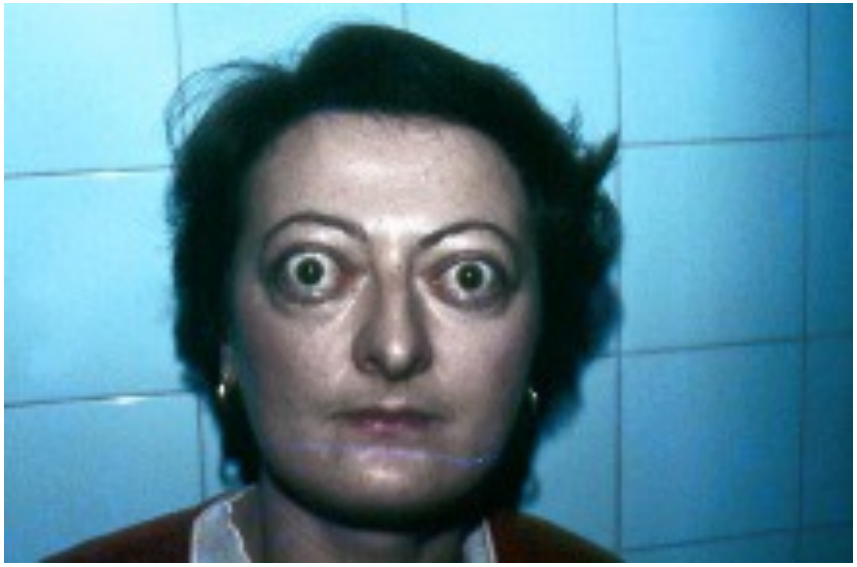
What stimulates T_3 and T_4
- TSH / Thyrotropin
- Increased blood iodine
- Hypoglycemia
- Cold

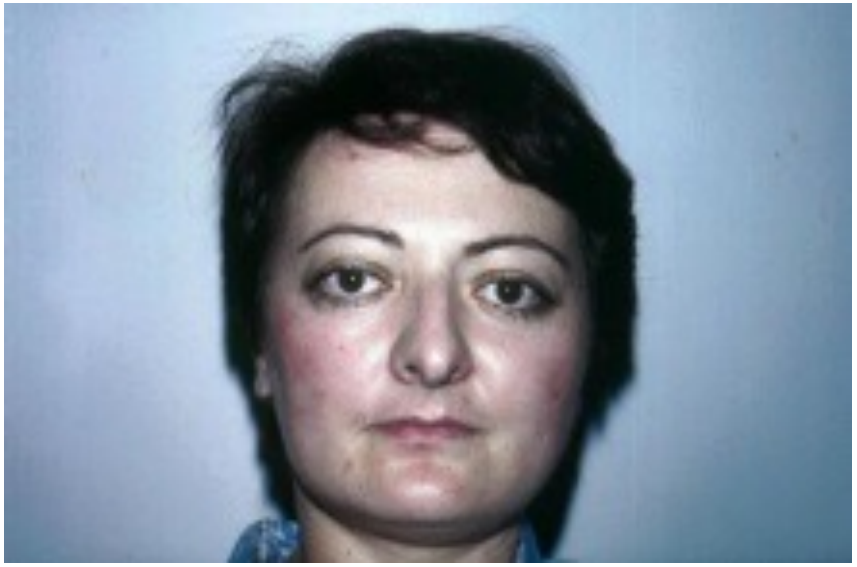
What inhibits T_3 and T_4
- Decreased level of TSH
- Decreased blood iodine
- Hyperglycemia
- Stress (via inhibition of TSH)

T_3 and T_4

Effects of T_3 and T_4 hypersecretion

- Excess sweating
- Heat intolerance
- Weight loss
- High heart rate
- Increased ventilation
- Nervousness
- Diarrhea
- **Exophthalmos** (protruding of the eyes)
  - Mucoprotein and fluid deposition behind the eye
- **Goiter**
  - Enlarged thyroid gland
- Caused by
  - **Grave's disease**
    - Autoantibodies are produced that mimic TSH
      - Agonist of the TSH receptor
  - Pituitary tumor
    - Increased TSH
  - Thyroid tumor (almost always causes hypersecretion)






Before
After

T_3 and T_4

Effects of T_3 and T_4 hyposecretion
- Cold intolerance
- Dry skin and hair
- Increased plasma cholesterol
- Low heart rate
- Decreased ventilation
- Lethargy
- Constipation
- Goiter if due to primary hypothyroidism
  - Causes an increase in TSH
    - Causes thyroid gland to enlarge
- Caused by
  - **Primary hypothyroidism** (~ 90% of cases)
    - Inability of thyroid gland to produce T_3 and T_4
      - Congenital errors in T_3 and T_4 synthesis
      - Iodine deficiency
        - Can lead to **cretinism** in unborn and infants
    - **Hashimoto's disease**
      - Autoimmune
  - **Secondary hypothyroidism**
    - Decreased TRH or TSH
      - Also causes thyroid gland to atrophy

Is it the gland in question or something else?

Primary condition

If a hormone is causing signs and symptoms and there is something wrong with the gland that is producing that hormone . . . It is a **primary condition**

Secondary condition

If a hormone is causing signs and symptoms and there is nothing wrong with the gland that is producing that hormone . . . But instead something else is causing that gland to be dysfunctional . . . It is a **secondary condition**

Endocrine Glands and Specialized Structures

Hypothalamus

Pituitary Gland (Anterior Pituitary and Posterior Pituitary)

Thyroid Gland

Parathyroid Glands

Adrenal Gland

Pancreas

Calcitonin

Thyroid hormone not involved with metabolism
Produced by **parafollicular cells / extrafollicular cells**
Function is unclear based on physiological levels
No known problems with hypersecretion or hyposecretion

Endocrine Glands and Specialized Structures

Hypothalamus

Pituitary Glands (Anterior Pituitary and Posterior Pituitary)

Thyroid Gland

Parathyroid Glands

Adrenal Gland

Pancreas

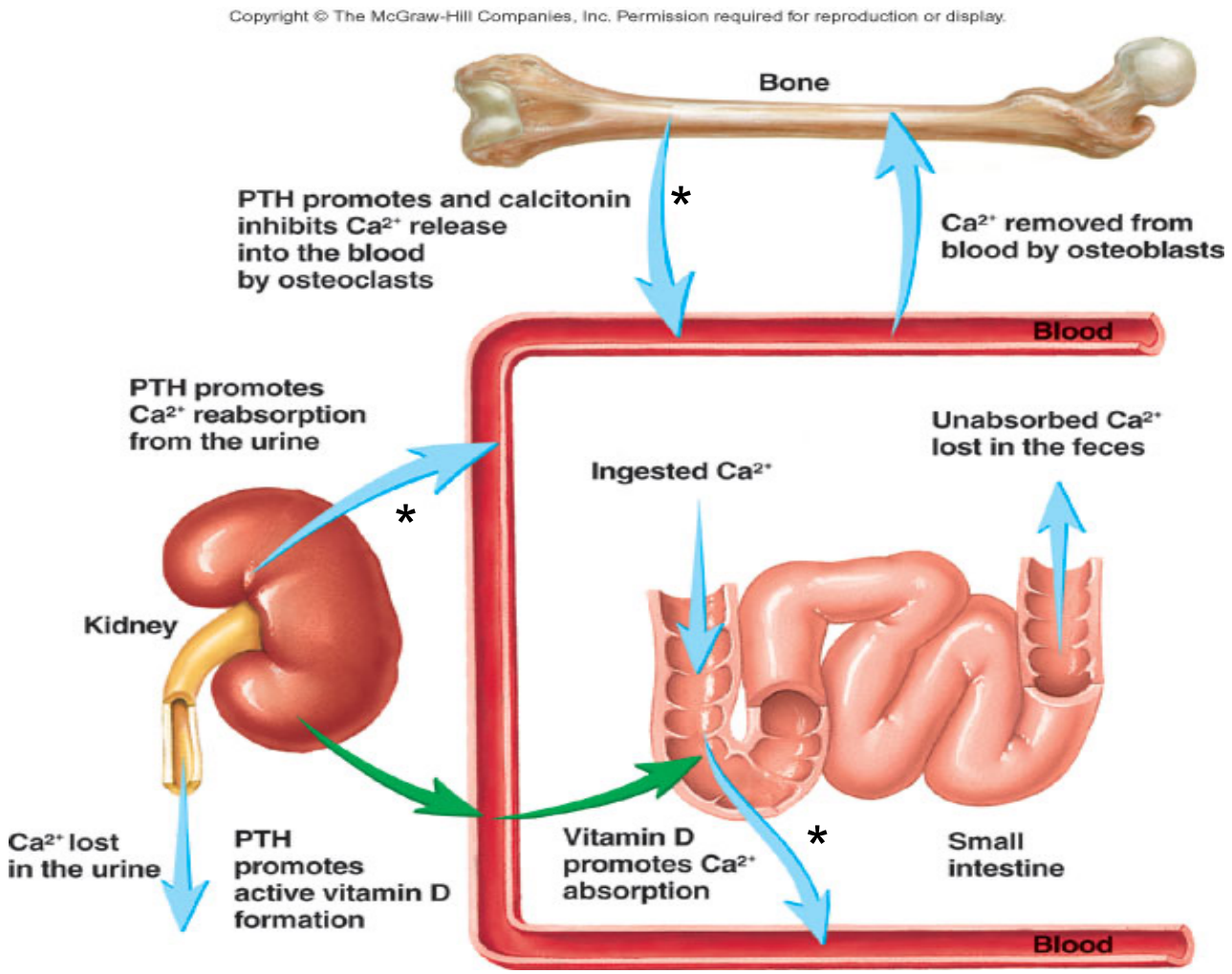
Parathyroid Glands

Four small glands located on the posterior / dorsal aspect of the thyroid

Parathyroid hormone

- Functions
  - Regulates blood calcium and blood phosphate
    - Regulates release of calcium from bones
    - Regulates reabsorption of calcium by kidneys
    - Regulates conversion of vitamin D_3 in the kidneys
      - Calcidiol into calcitriol
        - Aids in absorption of calcium in the gut
    - Regulates release of phosphate from bones
    - Regulates secretion of phosphate by kidneys
- What stimulates PTH
  - Low blood calcium
  - High blood phosphate
- What inhibits PTH
  - High blood calcium
  - Low blood phosphate

PTH Effects on Blood Calcium



Decreases blood phosphate levels

Stimulates secretion of phosphate by kidneys

PTH

Effects of PTH hypersecretion
- Osteoporosis
- High blood calcium (**hypercalcemia**)
  - Muscle weakness
  - Lethargy
  - High blood pressure
- Caused by
  - **Primary hyperparathyroidism**
    - Approximately 90% caused by tumor
    - Approximately 10% are idiopathic
  - ~~**Secondary hyperparathyroidism**~~
    - ~~Caused by conditions that reduce blood calcium~~
      - ~~Leads to release of PTH~~
    - ~~e.g. inadequate dietary intake of calcium~~
    - ~~e.g. inadequate levels of vitamin D~~

Effects of PTH hyposecretion
- Low blood calcium (**hypocalcemia**)
  - Seizures
  - Muscle spasms
- Caused by
  - Accidental removal during thyroidectomy
  - Autoimmune
  - Tumor
  - Congenital
  - Idiopathic

Endocrine Glands and Specialized Structures

Hypothalamus

Pituitary Glands (Anterior Pituitary and Posterior Pituitary)

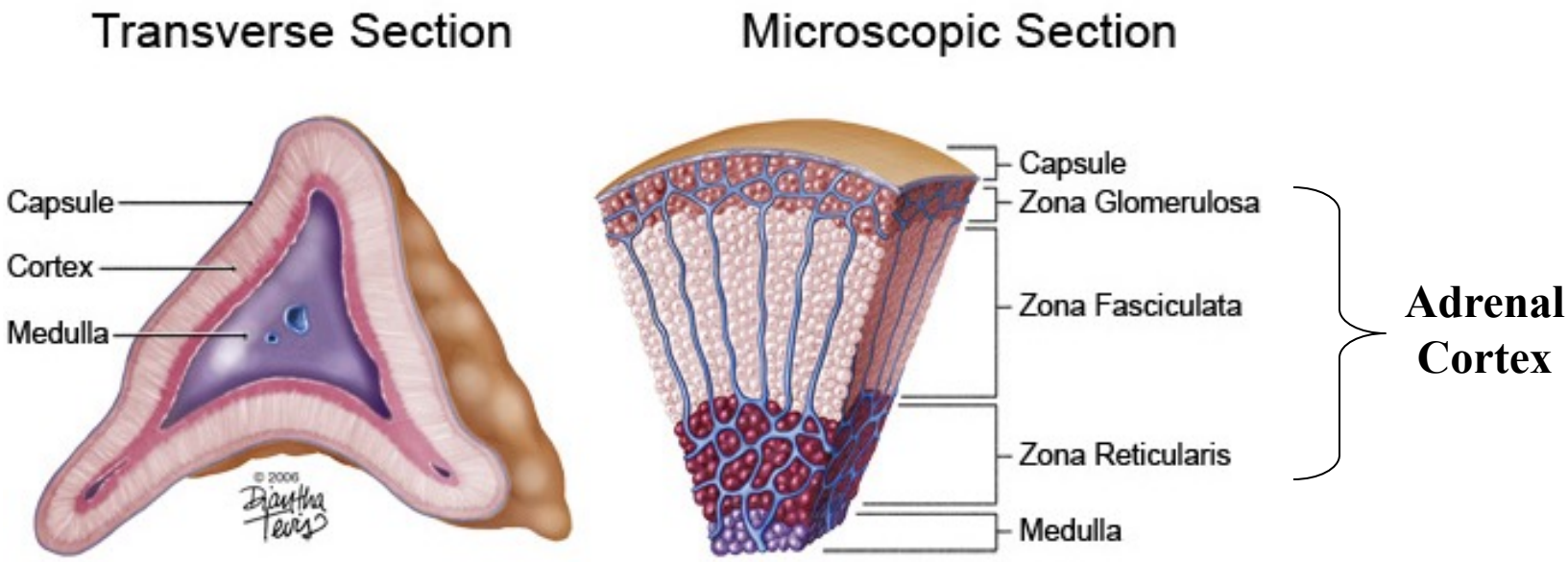
Thyroid Gland

Parathyroid Glands

Adrenal Gland

Pancreas

Adrenal Gland



Adrenal Gland

Adrenal Medulla

- Epinephrine and norepinephrine

Adrenal Cortex

- Mineralocorticoids (via zona glomerulosa)
  - e.g. Aldosterone
- Glucocorticoids (zona fasciculata)
  - e.g. Cortisol
- Androgens (zona reticularis)
  - e.g. DHEA and Androstenedione

Adrenal Medulla

Hormones released by the adrenal medulla

Epinephrine (adrenaline)

80% of secretion

Norepinephrine (noradrenaline)

20% of secretion

Adrenal Medulla

Functions of epinephrine and norepinephrine

- Increase metabolism (especially of skeletal and cardiac muscles)
- Increase heart rate
- Increase blood pressure
- Increase blood glucose via glycogenolysis
- Increase blood fatty acids via lipolysis
- Dilate airways

Norepinephrine and Epinephrine

What stimulates epinephrine and norepinephrine
- Sympathetic nervous system:
  - Stress
  - Physical activity
  - Low blood pressure (only when blood pressure is very low)

What inhibits epinephrine and norepinephrine
- No inhibitory input (low basal levels)

Effects of epinephrine and norepinephrine hypersecretion
- High blood pressure
- Increased heart rate
- Hyperglycemia
- Nervousness
- Excess sweating

Effects of epinephrine and norepinephrine hyposecretion
- None (low basal levels)

Mineralocorticoids (from zona glomerulosa)

- **Aldosterone**
  - Functions
    - Regulates blood pressure
      - Regulates Na^+ transport into blood by kidneys
        - Water follows Na^+ osmotically
      - Regulates transport of K^+ out of the blood by kidneys
      - Regulates transport of H^+ out of the blood by kidneys
  - What stimulates aldosterone (manifested via stimulation of renin)
    - **Renin** (an enzyme) is released from the kidneys
      - ~~Renin converts angiotensinogen into angiotensin I~~
        - ~~Angiotensin I converted into angiotensin II~~
          - ~~*Angiotensin II stimulates aldosterone*~~
      - Renin released in response to:
        - Decreased blood pressure / blood volume
        - Low blood sodium (hyponatremia)
        - High blood potassium (hyperkalemia)
    - Stress (solely psychological)
    - ACTH (minor role)
  - What inhibits aldosterone (manifested via inhibition of renin)
    - Increased blood pressure / blood volume
    - High blood sodium (hypernatremia)

Aldosterone

Effects of aldosterone hypersecretion
- High blood pressure / blood volume
- Hypernatremia
- Low blood potassium (hypokalemia)
- High blood pH (alkalosis)
- Caused by
  - **Primary aldosteronism**
    - **Conn's syndrome**
      - Caused by adrenal cortex tumor
      - Low renin due to negative feedback
  - **Secondary aldosteronism**
    - Caused by increased renin production
    - Can also be caused by increased ACTH
      - Can lead to darkened skin

Effects of aldosterone hyposecretion
- Low blood pressure / blood volume
- Hyponatremia
- Hyperkalemia
- Low blood pH (acidosis)
- Caused by
  - **Addison's disease**
    - Autoimmune destruction of adrenal cortex

Glucocorticoids (from zona fasciculata)

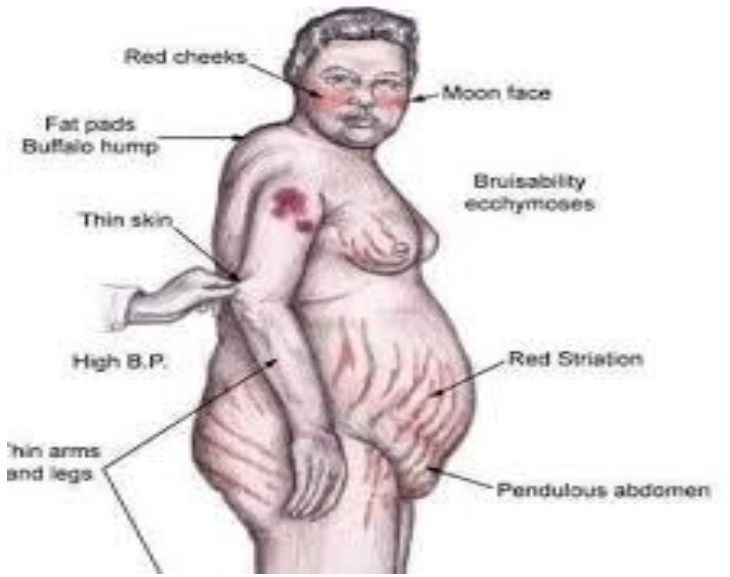
- **Glucocorticoids (Cortisol)**
  - Functions
    - Anti-inflammatory
      - Inhibits series 2 prostaglandins
      - ~~Decreases number of white blood cells~~
      - ~~Inhibits release of histamine from mast cells~~
    - Maintenance of blood vessel tone
    - Increases blood glucose via gluconeogenesis
      - Glucose made from amino acids
        - Cortisol stimulates protein catabolism
    - Helps the body deal with stress
  - What stimulates cortisol
    - Stress
    - Inflammation
    - Low blood glucose
    - Sleep-wake transition
    - ACTH
  - Inhibition of release
    - Deep sleep
    - Low ACTH

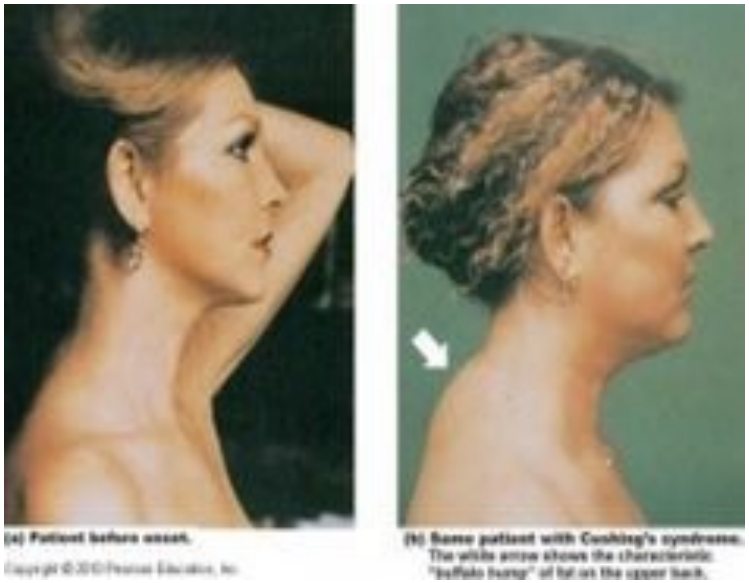
Cortisol

Effects of cortisol hypersecretion

- Hyperglycemia (**adrenal diabetes: a Type 2 diabetes**)
- Increased production of fat from excess glucose
  - Mainly in face ("moon face") and trunk (buffalo hump")
- Muscle wasting (from loss of protein)
- Edema (from loss of protein . . . collagen)
  - Blood vessels become more prominent in appearance
    - Face appears red and striae seen mainly on abdomen
- High blood pressure
- Frequent infection (immune system depressed)
  - Caused by
  - **Cushing's syndrome**
    - Due to adrenal tumor
    - Due to prolonged corticosteroid use
    - Results in low blood ACTH
      - Negative feedback from high cortisol
  - **Cushing's disease**
    - Caused by elevated ACTH from pituitary tumor
      - Could have darkened skin

Cushing's


Red cheeks
Moon face
Fat pads
Buffalo hump
Bruisability
ecchymoses
Thin skin
High B.P.
Red Striation
hin arms
nd legs
Pendulous abdomen




2008
2010
2015
2016

Cortisol

Effects of cortisol hyposecretion

- Inability to deal with stress (life threatening)
- Caused by
  - **Primary adrenal insufficiency**
    - Destruction of zona fasciculata due to Addison's
    - Results in high blood ACTH
      - Negative feedback from low cortisol
      - Could have darkened skin
  - **Secondary adrenal insufficiency**
    - Inadequate secretion of ACTH

Androgens (released by zona reticularis)

Androgens

- Released in very small amounts
- **Dehydroepiandrosterone (DHEA)**
  - Main androgen released
  - Converted to testosterone in males and females
  - Converted to estrogen in females
- **Androstenedione**
  - Converted to testosterone
- Effects masked in males by high testosterone levels
- Effects are only significant in females
  - Plays a large role in libido

What stimulates adrenal androgens

- ACTH

What inhibits adrenal androgens

- Low blood ACTH

Androgens

Effects of adrenal androgen hypersecretion
- Masculinizing effects in females
- Increased libido in women
- Early secondary sex characteristics in males
- Short stature if it occurs prior to puberty
  - Premature ossification of growth plate
- Ambiguous genitalia if it occurs prior to birth in females
- Caused by
  - **Adrenogenital syndrome**
    - Caused by inability to produce cortisol and aldosterone
      - Increased ACTH release via negative feedback
        - Leads to increased release of androgens

Effects of adrenal androgen hyposecretion
- Decreased libido in females

Endocrine Glands and Specialized Structures

Hypothalamus

Pituitary Glands (Posterior Pituitary and Anterior Pituitary)

Thyroid Gland

Parathyroid Glands

Adrenal Gland

Pancreas

Pancreas

Production and secretion of hormones by the Islets of Langerhans

Insulin (beta cells)

Glucagon (alpha cells)

Insulin

Functions
- Regulates blood glucose (main function)
  - Regulates cellular glucose transport from the blood
    - Decreases blood glucose
  - Regulates cellular transport of fatty acids from the blood
    - Increases lipogenesis
  - Regulates cellular transport of amino acids from the blood
    - Increases protein synthesis

Released by **beta islets of Langerhan cells** (~70% of cells)

What stimulates insulin
- High blood glucose (main stimulus)
- High blood fatty acids
- High blood amino acids
- ~~Glucose-dependent insulinotropic peptide~~

What inhibits insulin
- Low blood glucose
- Low blood fatty acids
- Low blood amino acids
- Stress

Insulin

Effects of insulin hypersecretion
- Low blood glucose (hypoglycemia)
  - Can lead to coma ("diabetic coma)
- Excess fat production and storage
- Increase in protein production
- Can lead to **type 2 diabetes mellitus** (~ 95% of cases)
  - Poor lifestyle is usually (but not always) to blame
  - Constant insulin hypersecretion leads to insulin resistance
  - **Insulin resistance**
    - Blood glucose regulation is lost
      - Leads to elevated blood glucose
    - Insulin production is usually fine (can decrease in later stages)
    - Type 2 diabetes is manageable with positive change in lifestyle
      - Meds (e.g. metformin, insulin) might also be needed

Effects of insulin hyposecretion
- Due to **type 1 diabetes mellitus** (~ 5% of cases)
  - Autoimmune destruction of beta cells (insulin not produced)
  - Also known as insulin-dependent diabetes mellitus
    - *Must take insulin daily (treatment)*

Insulin

Problems with failing to treat diabetes mellitus

Hyperglycemia (glucose cannot be transported into cells from blood)

Causes inflammation of blood vessels

Leads to cardiovascular disease

All tissues in danger of damage / death

Diabetic ketoacidosis

Combination of hyperglycemia and excess blood ketones

Events leading to diabetic ketoacidosis starts with a lack of insulin

Type 1 diabetic who fails to take insulin

Extreme stress (inhibits insulin)

Lack of insulin

Cells cannot transport glucose into cells from the blood

Causes hyperglycemia

Cells do not have glucose to make ATP

Use fatty acids exclusively to make ATP

Excess ketones are produced

Ketones are acidic

Can lead to coma *("diabetic coma")*

Pancreas

Production and secretion of hormones by the Islets of Langerhans

Insulin (beta cells)

Glucagon (alpha cells)

Glucagon

Functions

- Raises blood glucose via gluconeogenesis and glycogenolysis
- Raises blood fatty acids via lipolysis

Released by **alpha islets of Langerhan cells** (~30% of cells)

What stimulates glucagon

- Low blood glucose (main stimulus)
- Increased blood amino acids
  - Used for gluconeogenesis
- Sympathetics

What inhibits glucagon

- High blood glucose

Effects of glucagon hypersecretion

- High blood glucose

Effects of glucagon hyposecretion

- Low blood glucose (however, other hormones can raise blood glucose)

Too Much Chronic Stress and Its Effects on the Body

Stress Hormone	Increase Blood Pressure	Increase Blood Glucose
ADH	Yes	No
GH	No	Yes
ACTH	Yes (indirectly)	Yes (indirectly)
Epi & NE	Yes	Yes
Aldosterone	Yes	No
Cortisol	Yes	Yes

Too much chronic stress can contribute to the development of hypertension and type 2 diabetes

Male Reproductive System

Functions

Spermatogenesis
(production of male gametes - spermatozoa / sperm)

Delivery of sperm into the female reproductive tract

Produce male sex hormones

Spermatogenesis

Occurs in walls of seminiferous tubules

Starts during gestation

- However, stops during gestation and then starts again at puberty
  - Continuous once puberty begins

Mitosis of **spermatogonium** (sperm stem cell)

- Creates a single spermatogonium
- Creates a single **primary spermatocyte** (46 chromosomes)
  - Stops at this stage until puberty
    - Testosterone levels are too low up until puberty
    - Occurs continually at puberty and thereafter

Meiosis of primary spermatocyte (**meiosis I**) occurs at puberty and thereafter

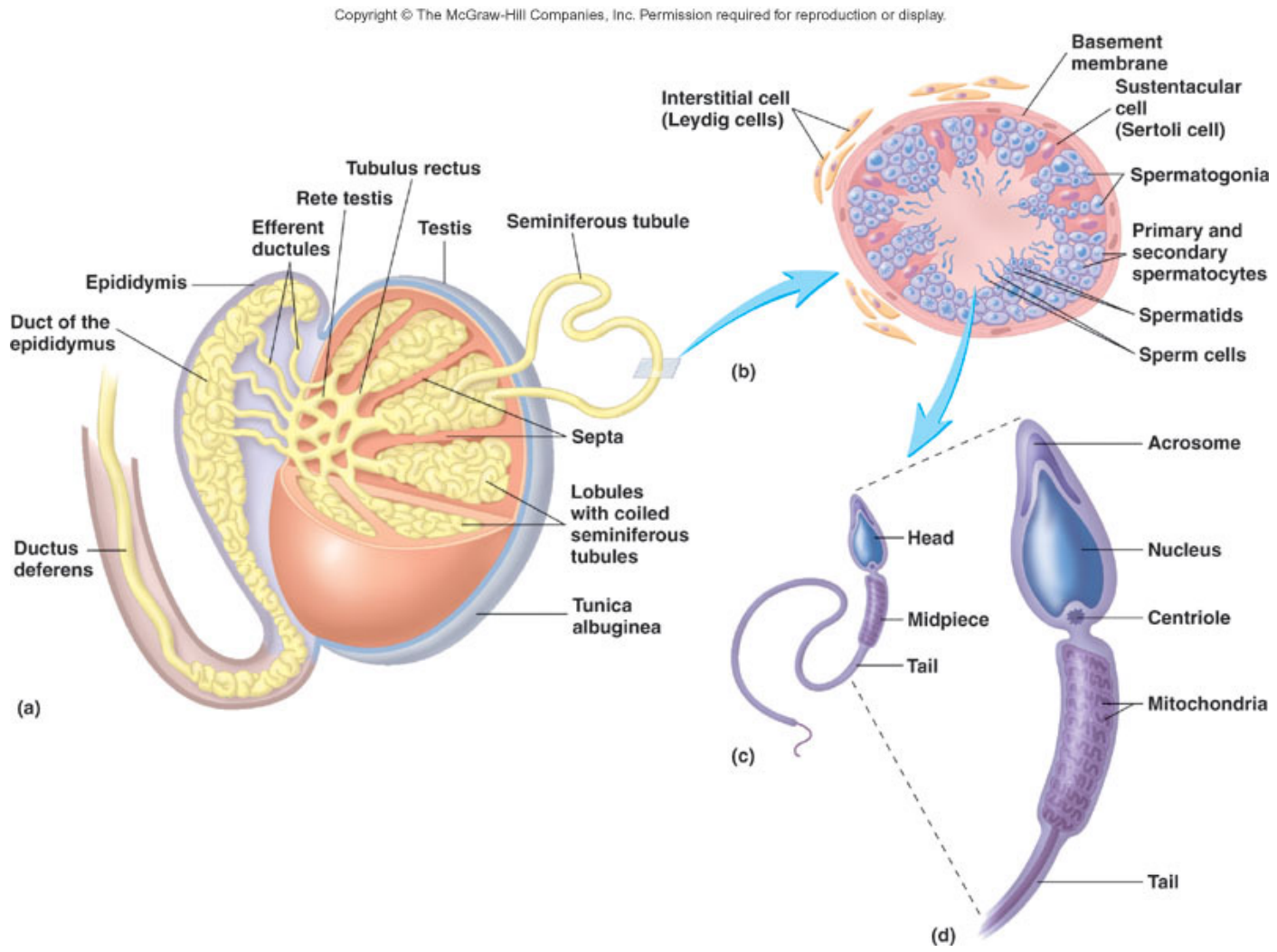
- Testosterone levels increase at puberty and thus initiate meiosis I
- Two **secondary spermatocytes** (23 chromosomes)

Meiosis of both secondary spermatocytes (**meiosis II**)

- Four **spermatids** (23 chromosomes)
  - Develop into mature sperm
    - Specialized **haploid** (23 chromosome) cells

Sperm stored in tail of epididymis and proximal end of vas deferens

Spermatogenesis Occurs in Walls of Seminiferous Tubule

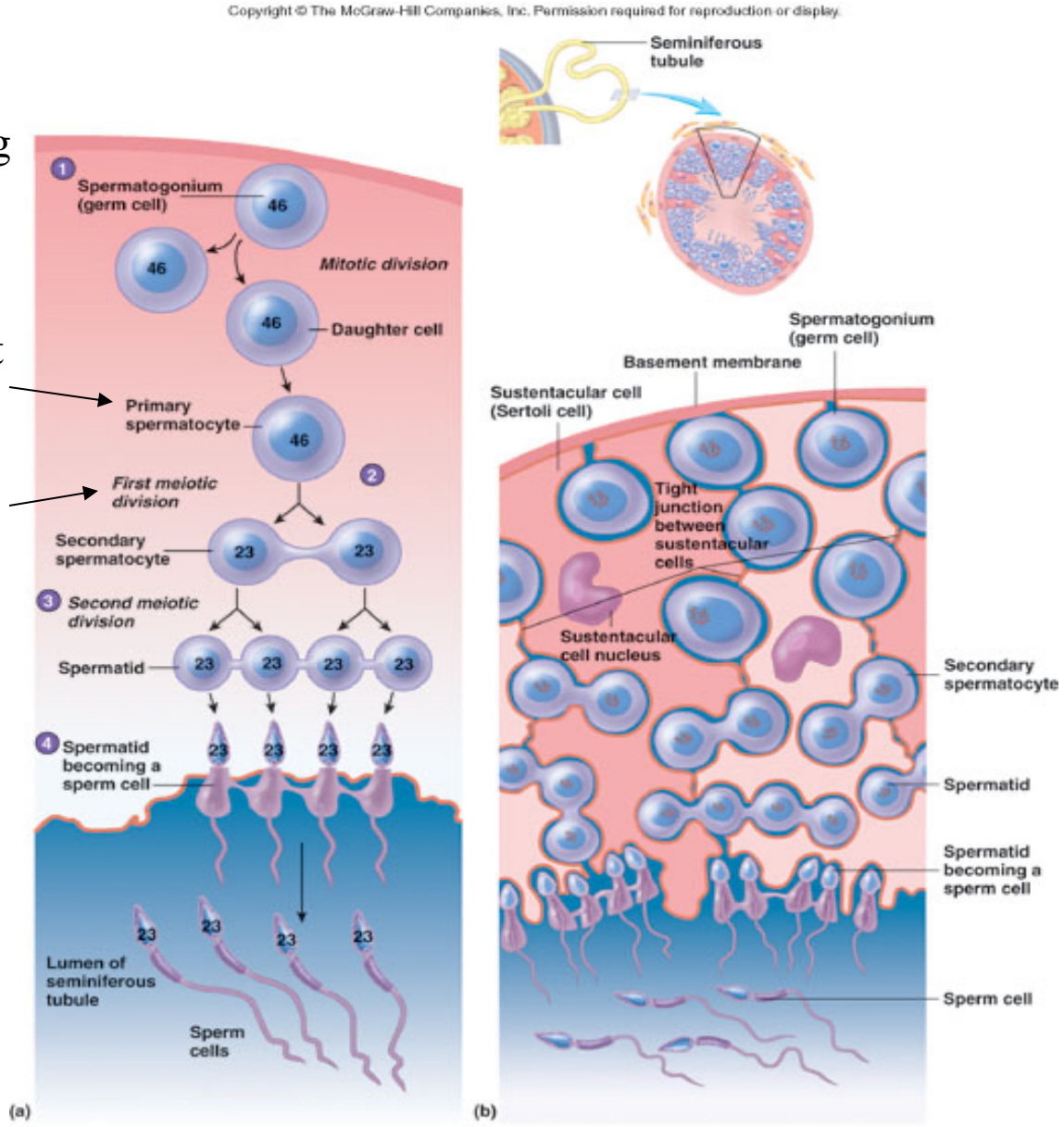


Spermatogenesis

starts during gestation

arrested at this stage

resumes at puberty



Delivery of Sperm into the Female Reproductive Tract

Semen is the vehicle of sperm delivery

Erection of penis for delivery of semen

Ejaculation of semen into female reproductive tract

Erection

With stimulation, *parasympathetics* release two neurotransmitters

Acetylcholine and *nitric oxide*

Causes an increase in blood flow to erectile tissue of penis

Sinusoids of erectile tissue fill with blood

Compress veins of erectile tissue

Blood trapped within erectile tissue

Ejaculation

With stimulation, *sympathetics* cause **emission**

Mixing of sperm with semen and discharge into prostatic urethra

Urethra fills and sends action potentials to sacral nerves

Parasympathetics relax smooth muscle of the urethra, urogenital diaphragm and external urethra

Somatics

Causes rhythmic contractions

Forces semen out of penile urethra

Hormonal Control of Male Reproductive System

Hypothalamus

Releases **gonadotropin-releasing hormone (GnRH)**

Stimulates anterior pituitary to release gonadotropins

Anterior pituitary

Releases gonadotropins

Luteinizing hormone (LH)

Stimulates Leydig cells of testes

Stimulates production of testosterone

Follicle stimulating hormone (FSH)

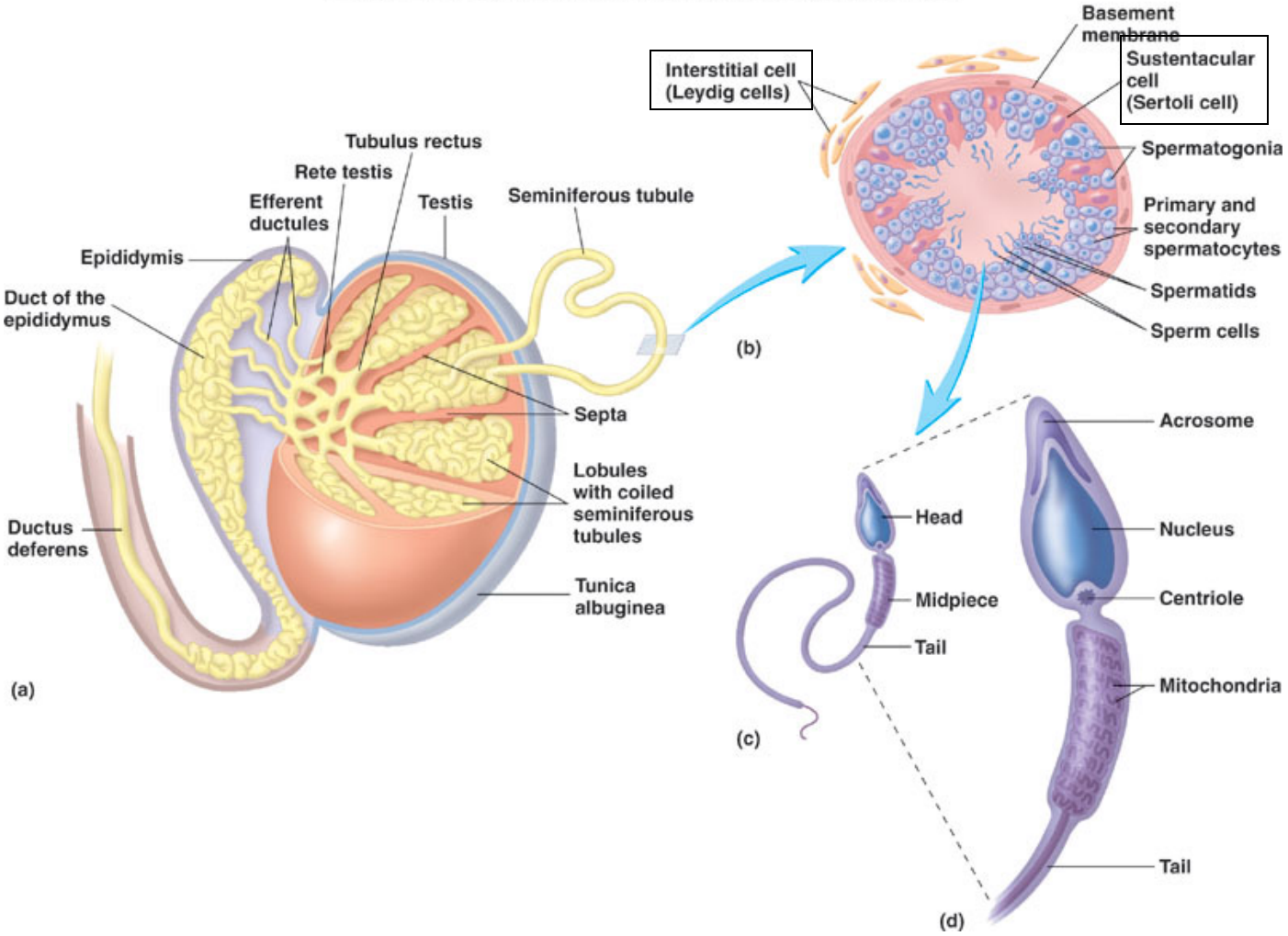
Stimulates Sertoli cells of testes

Stimulates spermatogenesis

Stimulates release of **inhibin**

Inhibits release of FSH

Feedback mechanism on FSH

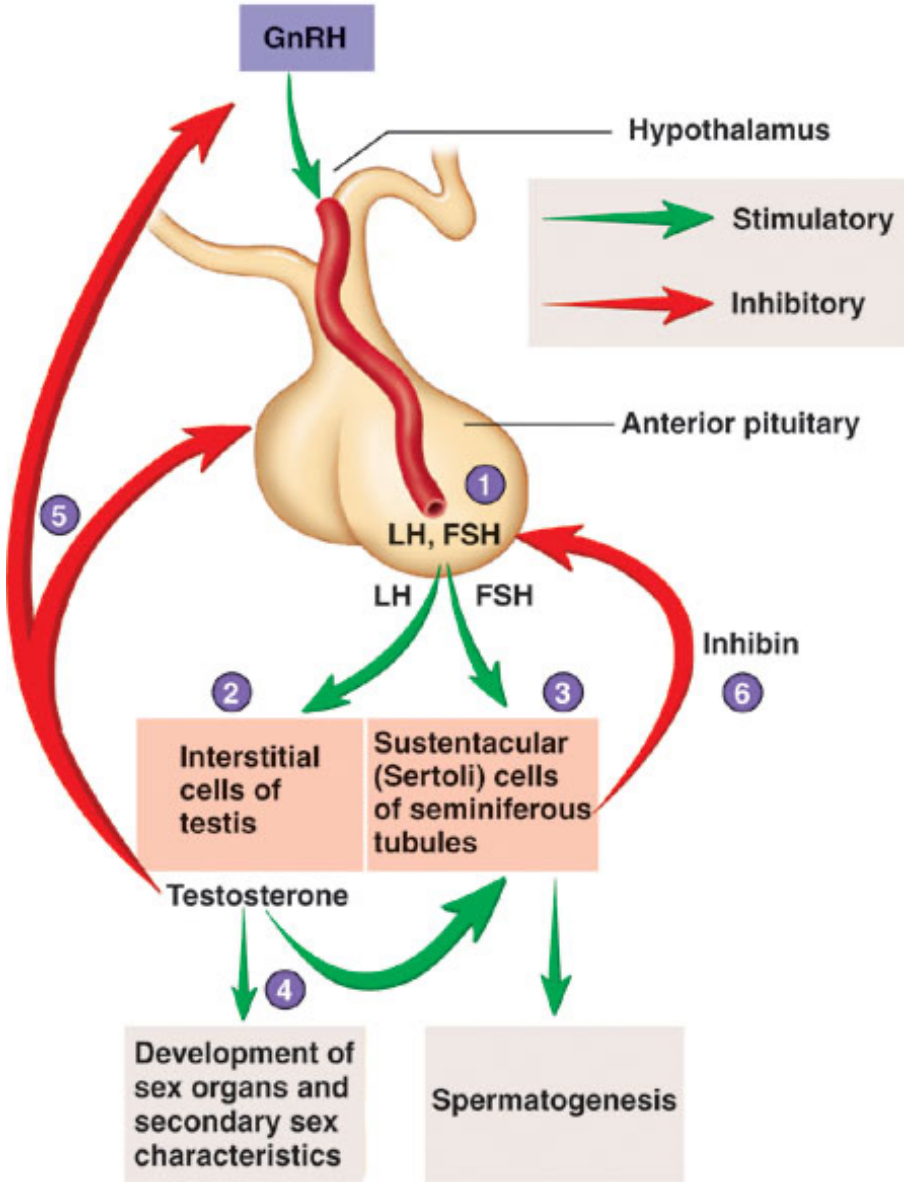


Hormonal Control of Male Reproductive System

Testes

- **Leydig cells** release **testosterone** (main androgen of the body)
  - Secretion during gestation
    - Causes development of primary sex characteristics
      - Development of male reproductive organs
    - Causes descent of testes
  - Secretion ceases during childhood
  - Secretion resumes during puberty
    - Allows production of sperm
    - Causes development of secondary sex characteristics
      - Muscle growth
      - Thickening of vocal cords (deep voice)
      - Increased bone density
      - Increased growth of body hair
      - Increased activity of sebaceous glands
- **Sertoli cells** release inhibin

Hormonal Control of Male Reproductive System



Female Reproductive System

Functions

Oogenesis
(production of female gamete)

Produce female sex hormones

Transport of female gamete to site of fertilization

Incubate fertilized egg / Gestation
(becomes embryo and finally fetus)

Chapter 29

Oogenesis

At birth, each ovary contains ~ 1,000,000 **primordial follicles**

Each follicle contains an **oocyte**

Each oocyte has already begun meiosis I but then stops

At puberty, further development reduces them to ~ 400,000

Primary follicles

Each contains a **primary oocyte**

With the onset of puberty, a dozen or so primary follicles develop each month

Only one of these follicles outgrows the others and continues to mature

The other primary follicles disintegrate

Meiosis I of oocyte is completed as is primary follicle maturation

Produces:

Primary follicle matures into a **Graafian follicle**

Primary oocyte divides to produce:

Secondary oocyte

Contained within Graafian follicle

Polar body

Disintegrates

Meiosis II begins but then stops

Secondary oocyte ruptures from Graafian follicle at ~ 2 weeks

Ovulation

Remains viable for approximately 24 hours

If fertilized by sperm, completes meiosis II

Secondary oocyte divides to produce:

Zygote

Polar body

Disintegrates

Remaining Graafian follicle becomes the **corpus luteum**

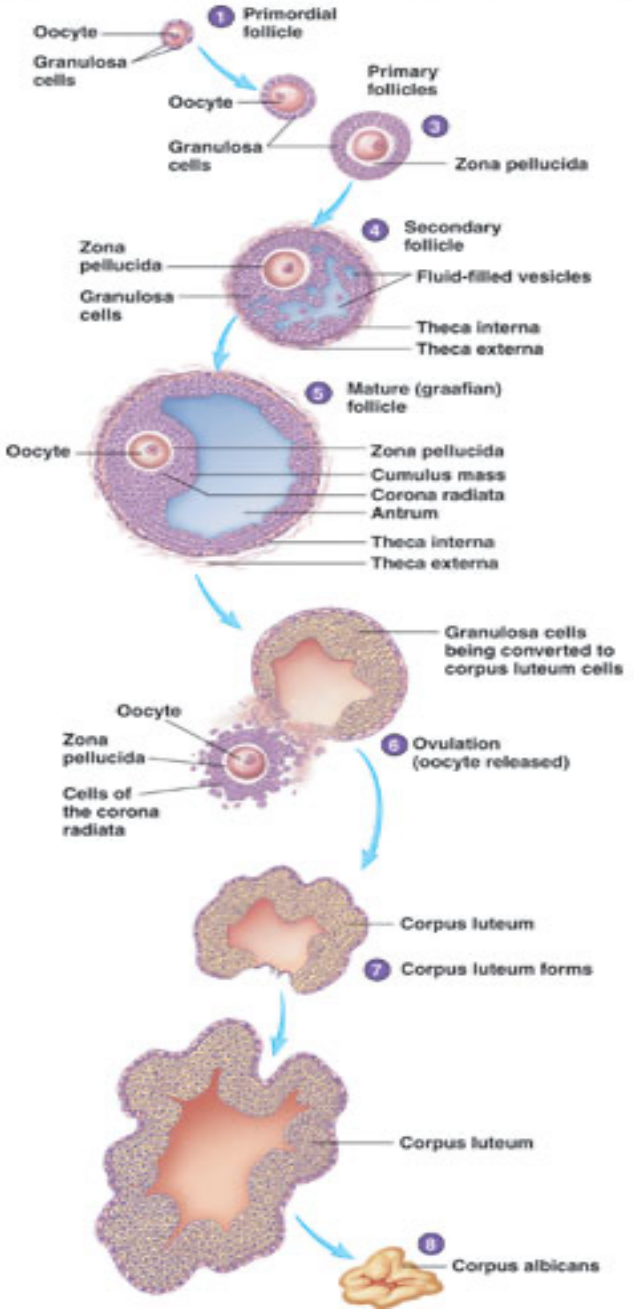
"Yellow body"

Produces estrogen and progesterone

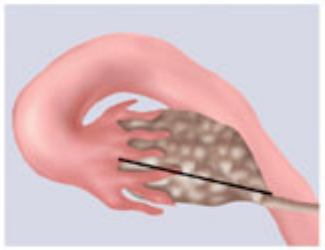
If not fertilized

Secondary oocyte disintegrates

Corpus luteum degenerates



Ovary



Hormonal Control of Female Reproductive Functions

Hypothalamus

- Releases **gonadotropin-releasing hormone (GnRH)**
  - Stimulates gonadotropins to be released by the anterior pituitary

Anterior pituitary

- Releases gonadotropins
  - **Luteinizing hormone**
    - Helps regulate the menstrual cycle
  - **Follicle stimulating hormone**
    - Aids in maturation of the follicles
    - Stimulates release of estrogen and progesterone from ovaries
    - Helps regulate the menstrual cycle

Hormonal Control of Female Reproductive Functions

Ovaries
- Release progesterone
  - Helps regulate the menstrual cycle
  - Affects mammary glands during puberty
- Release estrogens (**estradiol** is the most abundant)
  - Helps regulate the menstrual cycle
  - Responsible for development of secondary sex characteristics
    - Increased deposition of fat in breasts, thighs and buttocks
    - Stimulates enlargement of accessory reproductive organs

Trophoblast cells of the embryo
- **Human chorionic gonadotropin (hCG)**
  - Supports corpus luteum
    - Estrogen and progesterone levels remain high
      - Prevents ovulation and menses
  - Pregnancy tests detect hCG in urine

Menstrual Cycle

Three Phases

Menstruation / Menses

Proliferative Phase

Secretory Phase / Luteal Phase

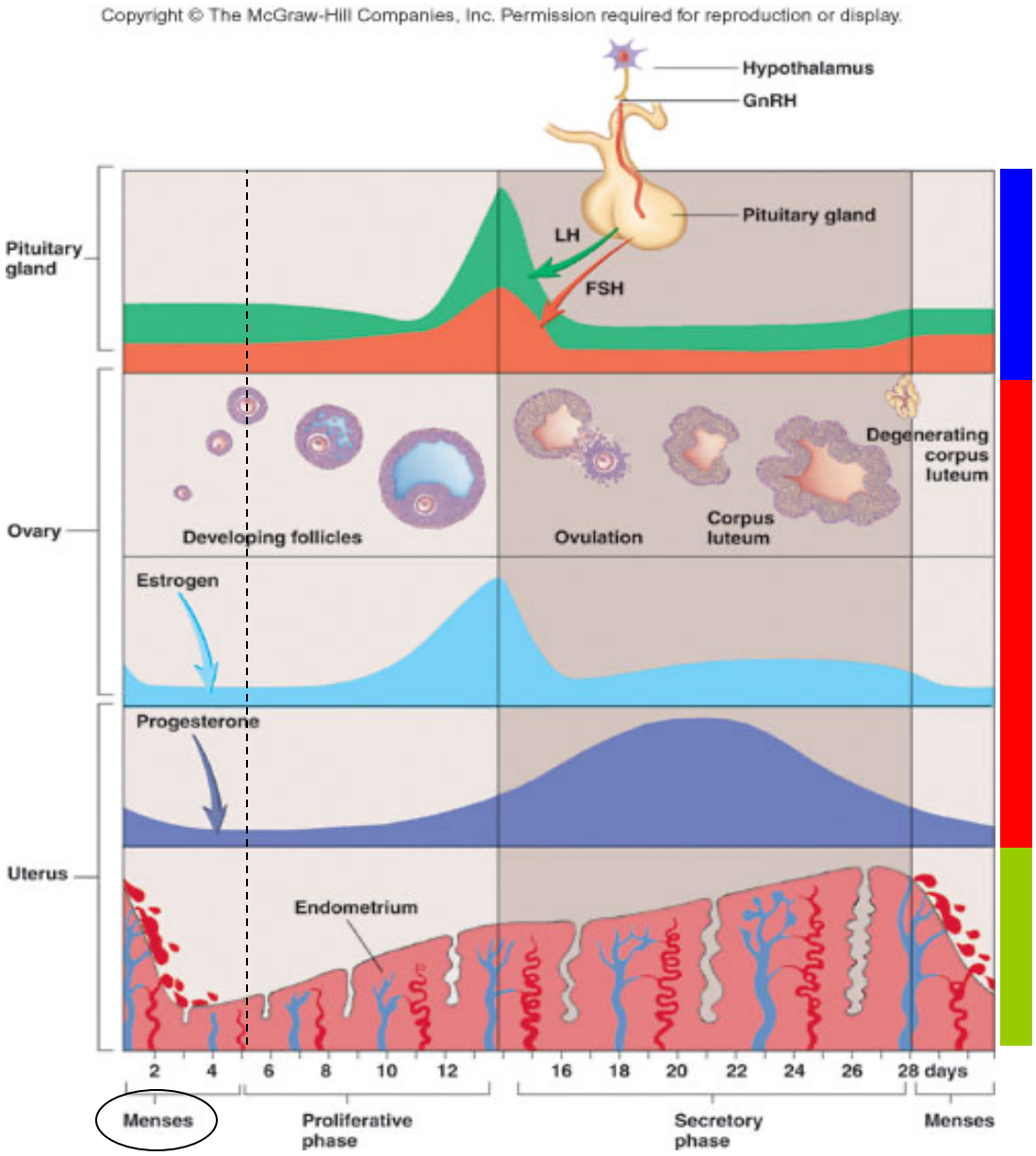
Menstruation / Menses

Day one is considered the start of the menstrual cycle

Decrease in estrogen and progesterone
- Brought about when corpus luteum degenerates
- Causes blood vessels of endometrium to constrict
  - Causes lack of blood supply to endometrium
    - Causes uterine lining to slough off
      - Blood vessels are torn (bleeding)

Release of GnRH from hypothalamus begins another cycle
- FSH slowly rises
  - Stimulate several primary follicles to start developing
- LH slowly rises

Menstruation / Menses



Anterior Pituitary

Ovary

Endometrium

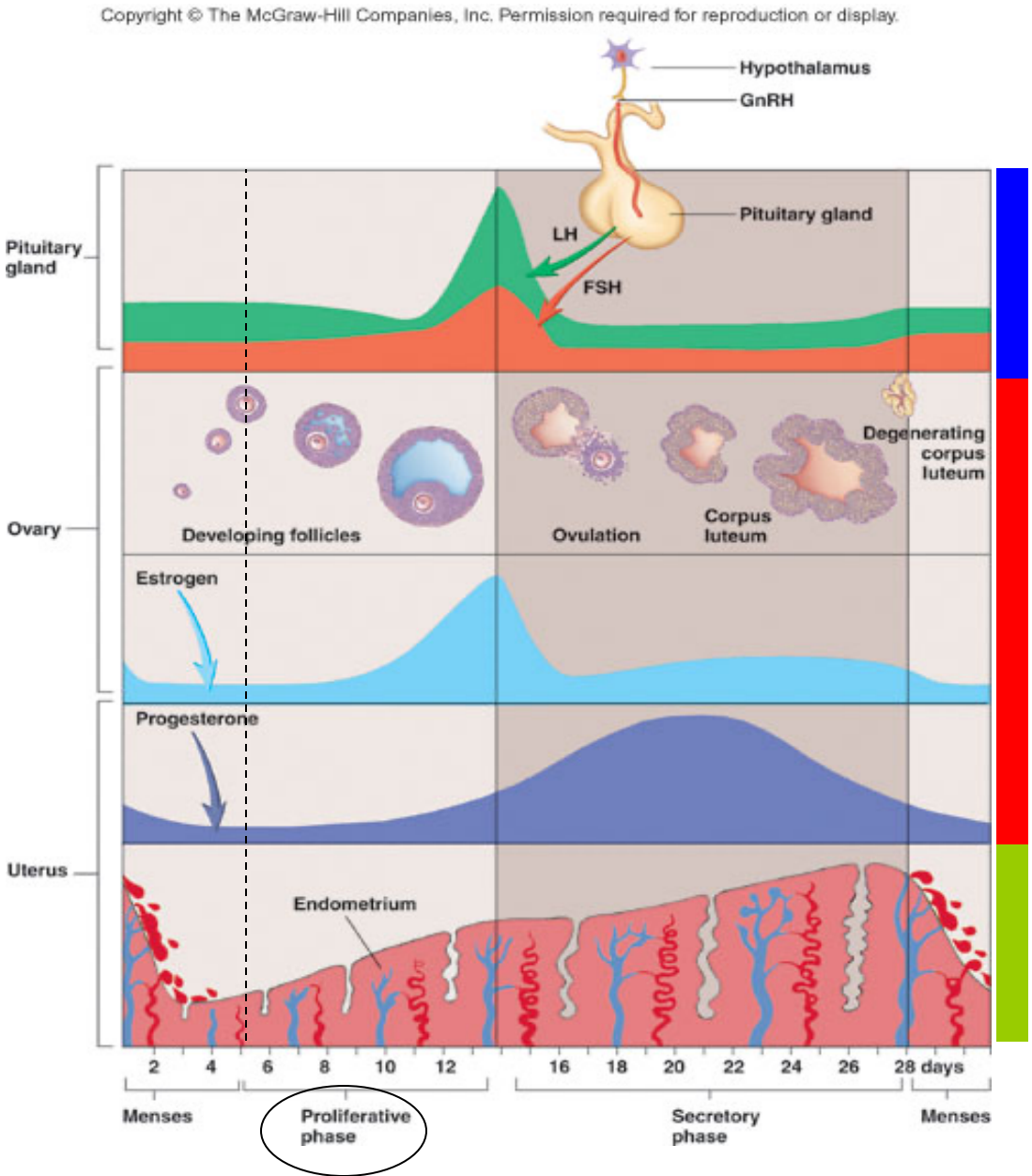
Proliferative Phase

End of menses to (but not including) ovulation

Primary follicles mature
- Single secondary follicle will emerge (others degenerate)
  - Develops into Graafian follicle

Increase in estrogen secretion from developing follicles
- Begins to thicken uterine wall
- Increase in estrogen is aided by LH and FSH
- Estrogen surges
  - Occurs via positive feedback
  - Causes LH surge and FSH surge

Proliferative Phase



Anterior Pituitary

Ovary

Endometrium

Secretory Phase / Luteal Phase

Begins at **ovulation**

- Secondary oocyte ruptures from the Graafian follicle
  - Caused by **LH surge** and to a lesser extent **FSH surge**

Estrogen levels rapidly decline

- Due to FSH surge

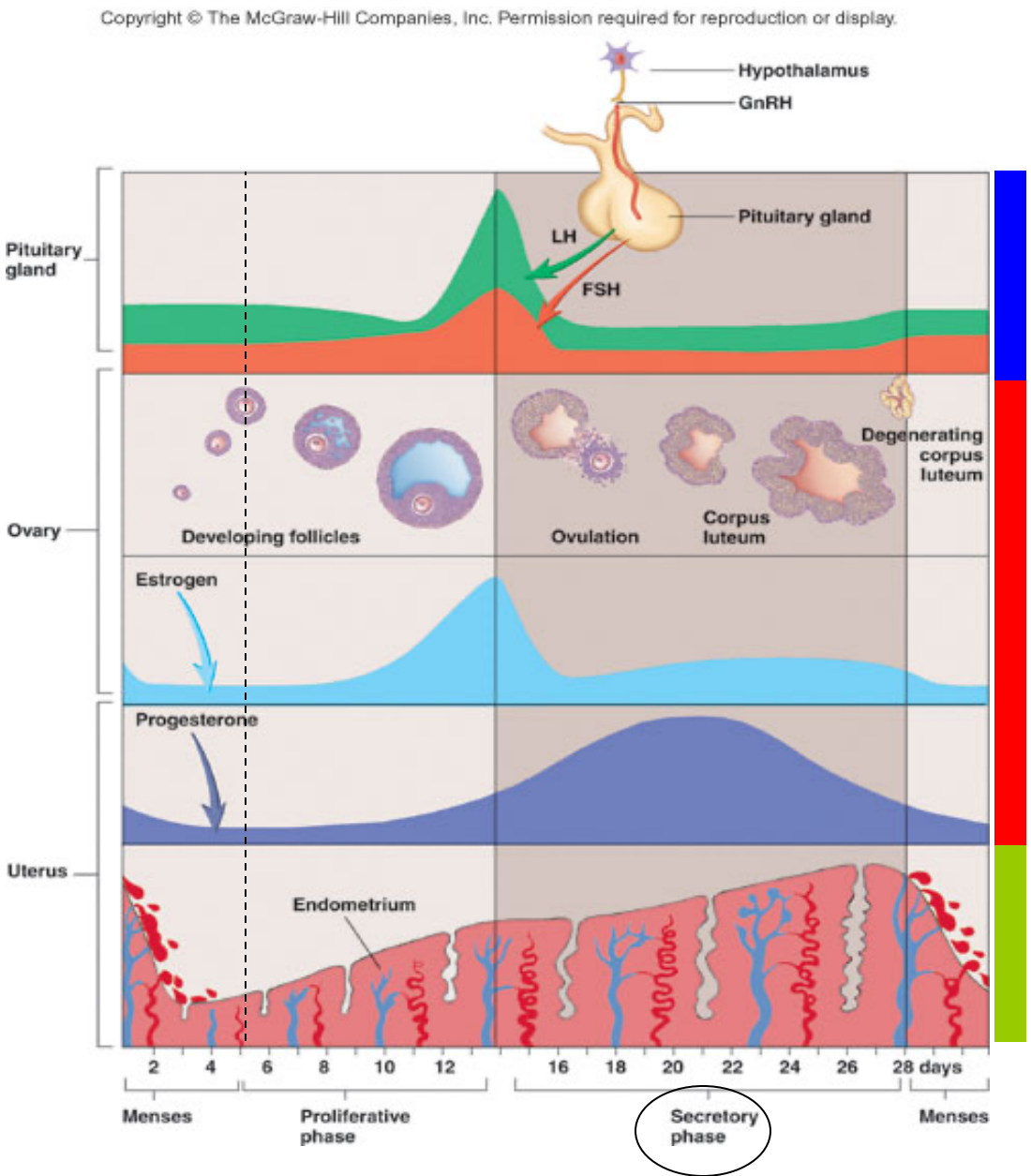
Corpus luteum secretes estrogen and progesterone

- Inhibits LH and FSH
- Estrogen levels begin to increase again
- Progesterone reaches its highest level
  - Thickens uterine wall
  - Increase vascularity of uterine wall

Corpus luteum degenerates

- Due to failure of fertilization
- Progesterone and estrogen then begin to decline

Secretory Phase / Luteal Phase



Anterior Pituitary

Ovary

Endometrium

Menarche

- First menstrual cycle (typically begins at ~ 12 years of age)
- Ovaries mature and are able to respond to hormones of menstrual cycle

Amenorrhea

- Failure to menstruate
- Occurs for any number of reasons
  - Women with very low body fat
    - Need fat to make estrogen and progesterone
  - Some change in lifestyle
  - Stress and anxiety

Menopause

- Cessation of the menstrual cycle
- Typically occurs between the ages of 45 and 55
- Due to degeneration of the ovaries
  - Decrease in levels of progesterone and estrogen
- Symptoms (revolve around hypothalamic dysfunction)
  - "Hot flashes"
  - Irritability
  - Fatigue
  - Emotional disturbance

Birth control pills

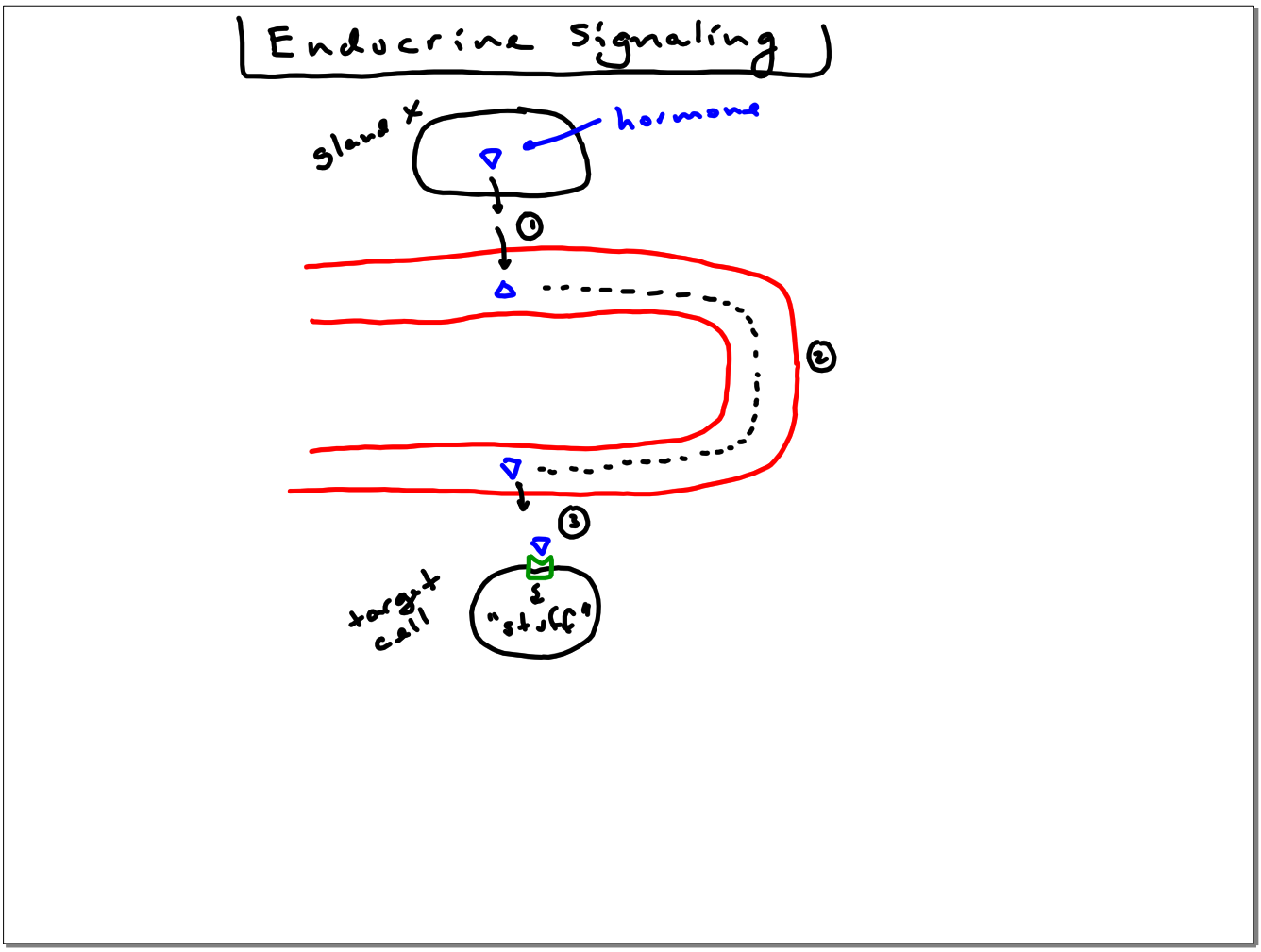
- "Traditional" birth control pills
  - Contains high levels of estrogen and progesterone
    - Prevents ovulation (especially the high levels of estrogen)
      - Inhibits LH / FSH surge
    - Thickens cervical mucus (progesterone)
      - Makes it difficult for sperm to pass through the uterus
- "Minipill" birth control
  - Contains solely progesterone
  - Thickens cervical mucus
- Approximately 99% effective when taken as directed

Emergency Contraception (e.g. Plan B One Step – very high dose of progesterone)

- Mechanism of action dictated by when during the menstrual cycle it is taken
  - Can prevent ovulation
  - Can prevent fertilization
    - Thickens cervical mucus
  - Can block progesterone receptors of the endometrium
    - Causes thinning of endometrium and thus prevents implantation
- Needs to be taken within 72 hours of unprotected sex (~ 90 to 95% effective)

RU486 (Mifepristone . . . Mifeprex® / Early Option®)

- Abortion pill
- Blocks progesterone receptors of the endometrium
  - Causes endometrium to slough off
- Used in conjunction with **misoprostol**
  - Synthetic prostaglandin (PGE_1) that causes uterine contractions
- ~ 90% effective up through the 7th week of pregnancy



Jan 11-4:53 PM

Production of Prostaglandins

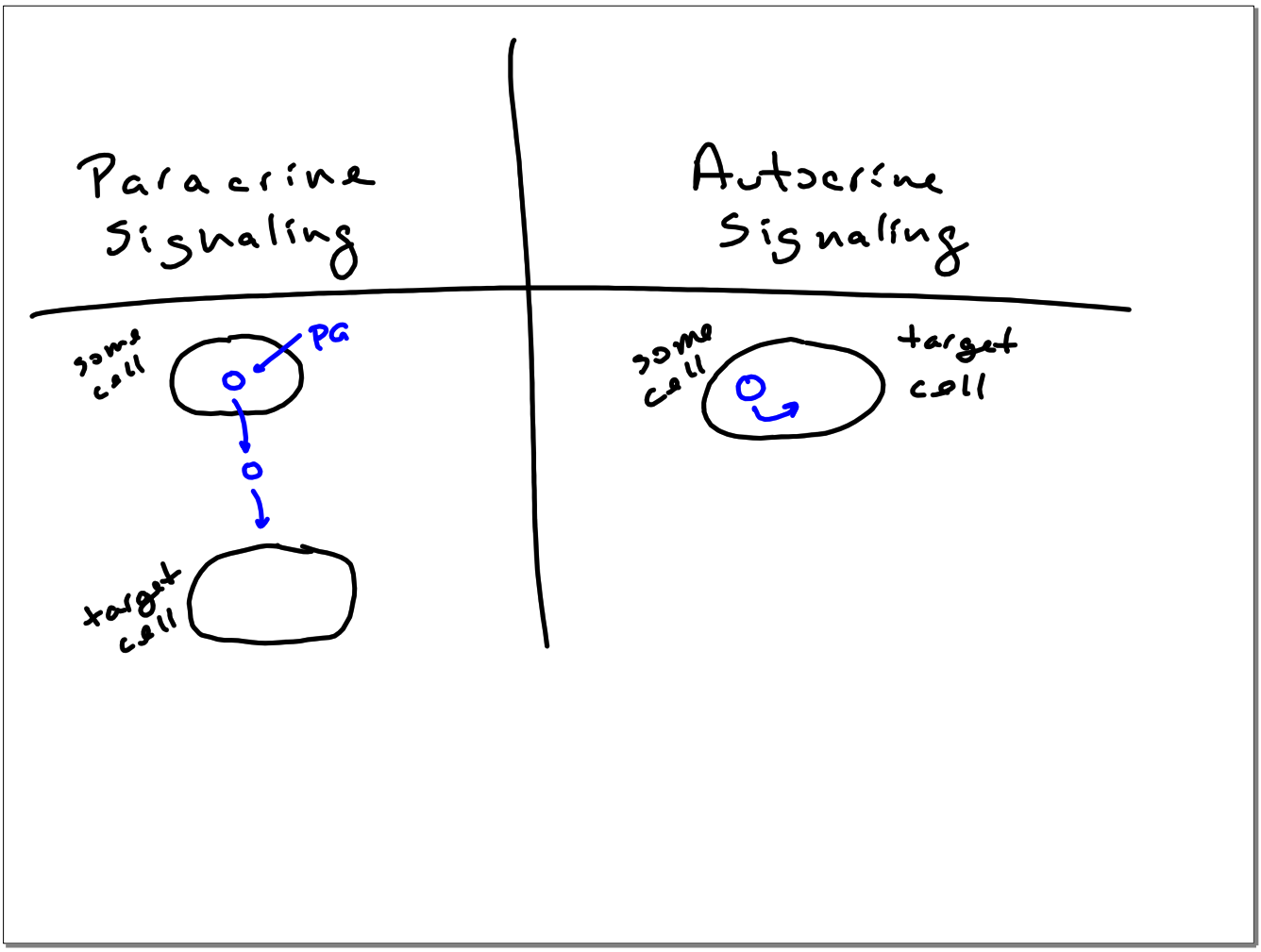
fatty acids	Chem Rxn's	prostaglandins
GLA	cox-1 cox-2 →	PG-1
AA	cox-1 cox-2 →	PG-2
EPA	cox-1 cox-2 →	PG-3

cox inhibitors: block production of PG's

e.g. ibuprofen (Motrin, Advil), naproxen (Aleve), aspirin

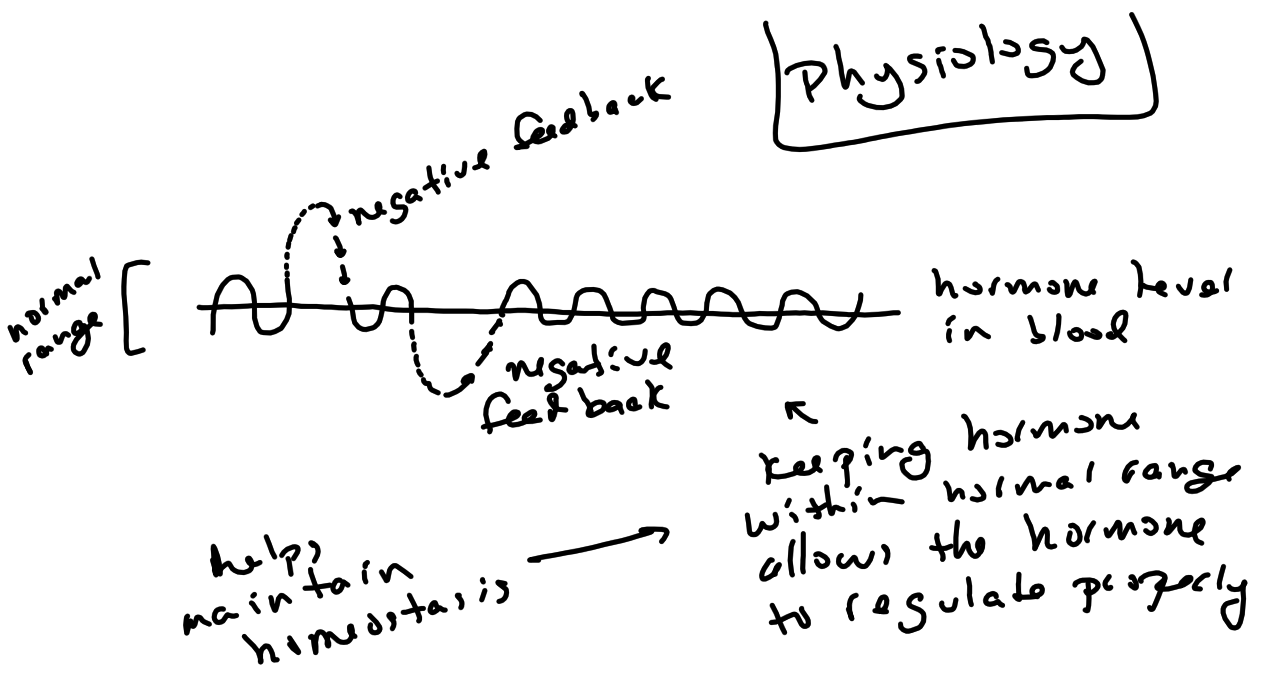
NSAIDs

← specifically PG-2
↳ ↓ pain
↳ ↓ inflammation
↳ ↓ fever


Paracrine Signaling
Autocrine Signaling
some cell
PG
target cell
some cell
target cell

Jan 11-5:23 PM

Physiology



keeping hormone within normal range allows the hormone to regulate properly

help maintain homeostasis

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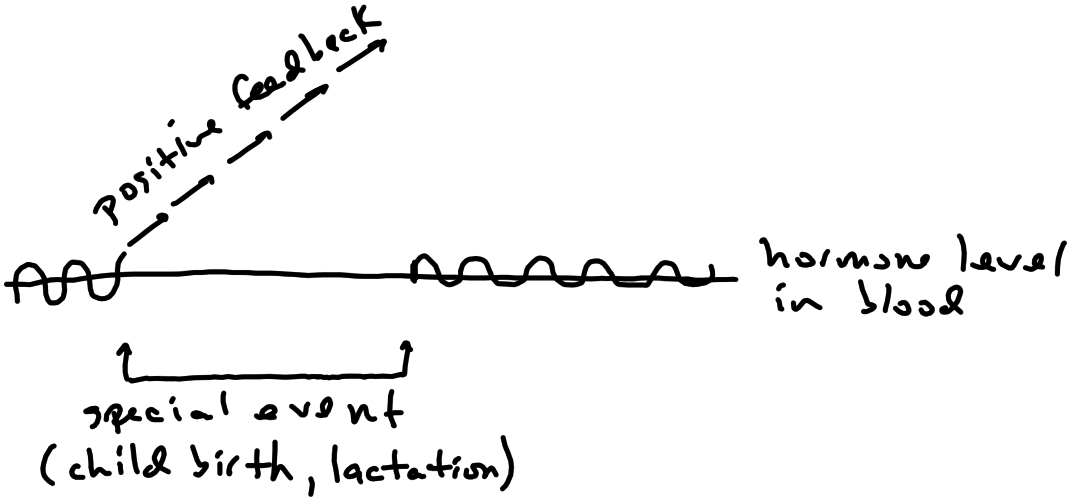
positive feedback
hormone level in blood
special event
(child birth, lactation)

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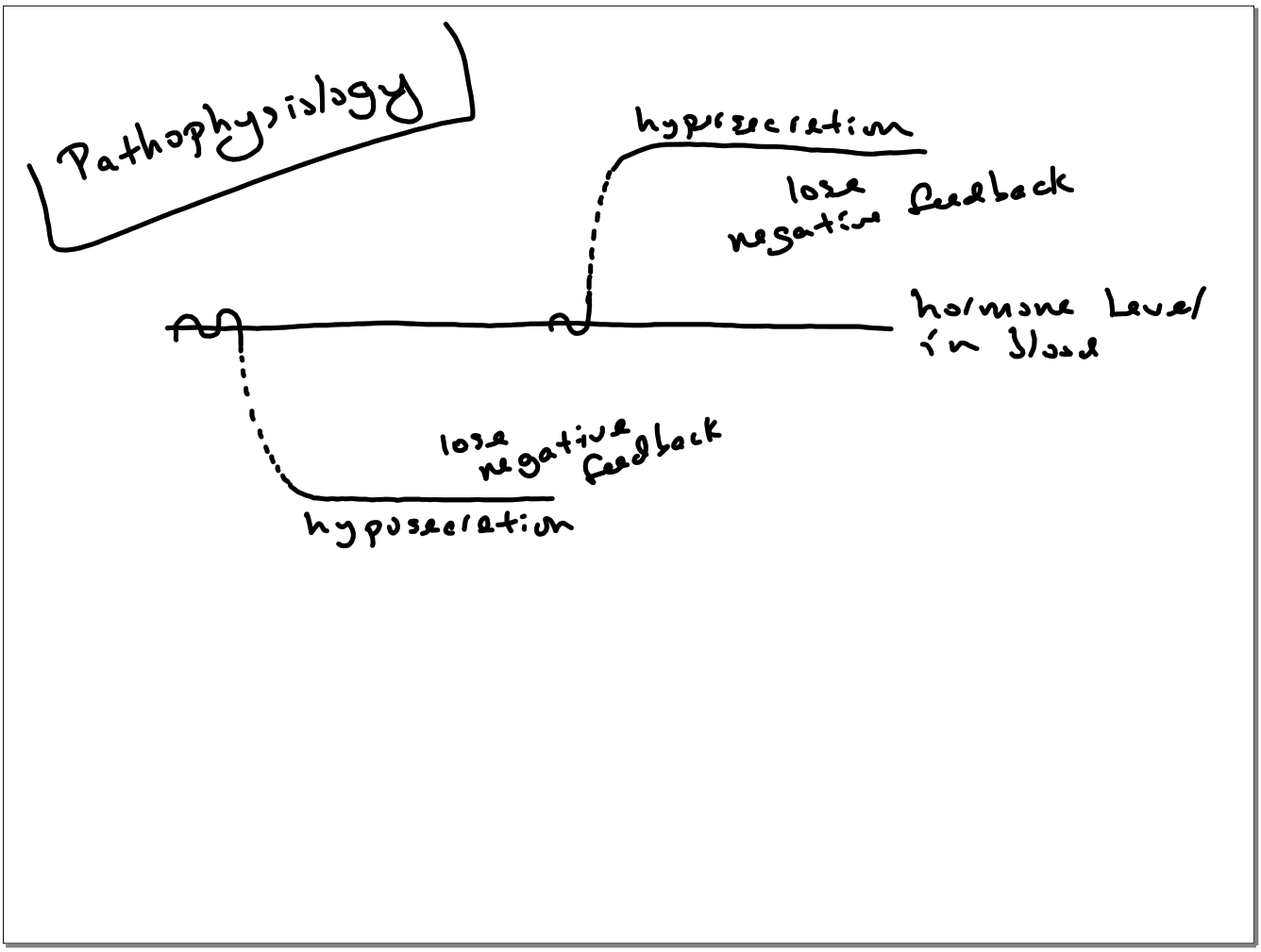
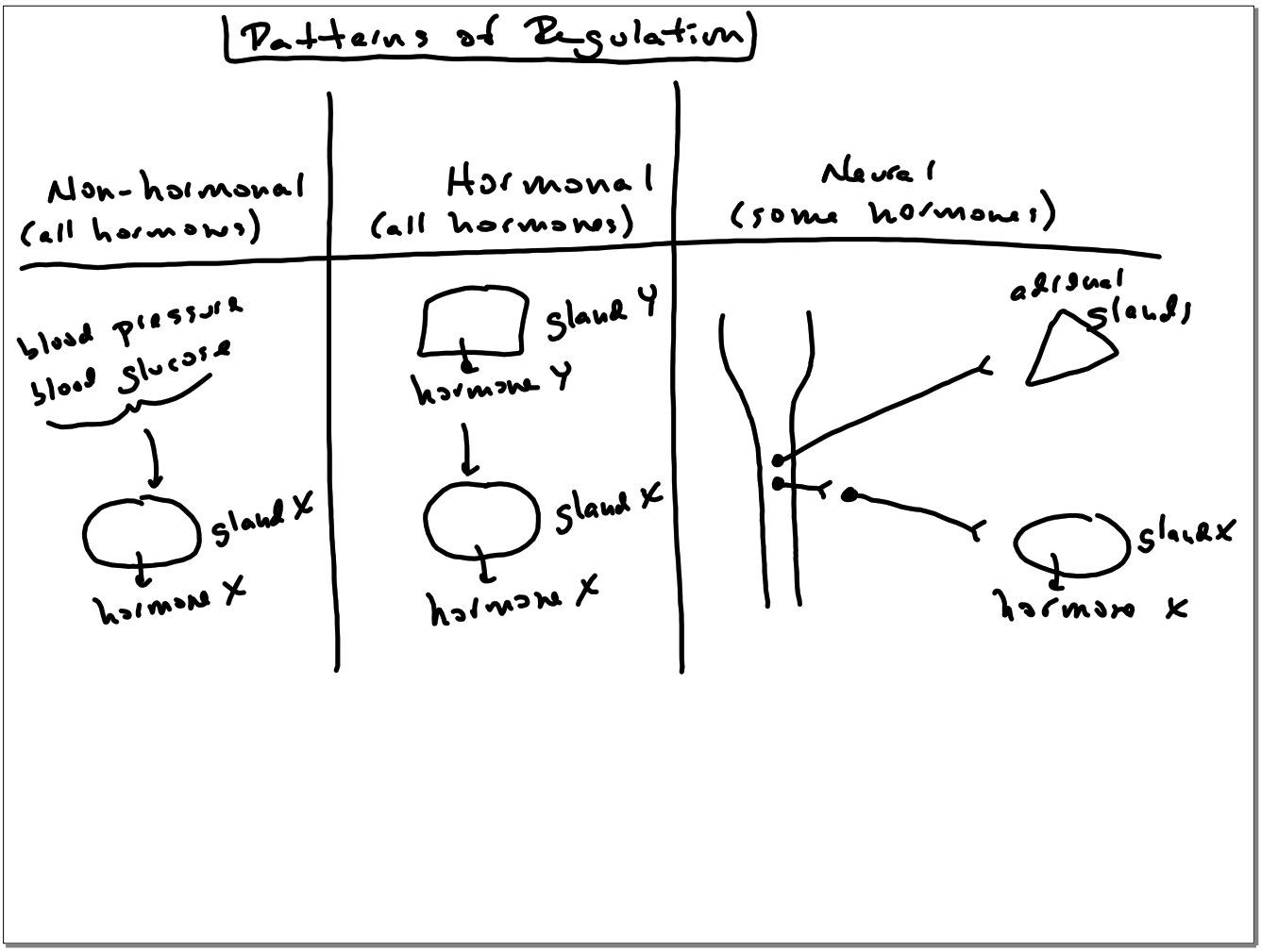

Pathophysiology
hypersecretion
lose negative feedback
hormone Level in blood
lose negative feedback
hyposecretion

Jan 11-5:33 PM


Patterns of Regulation
Non-hormonal (all hormones)
Hormonal (all hormones)
Neural (some hormones)
blood pressure
blood glucose
gland X
hormone X
gland Y
hormone Y
gland X
hormone X
adrenal gland
gland X
hormone X

Jan 11-5:36 PM

Patterns of hormone release

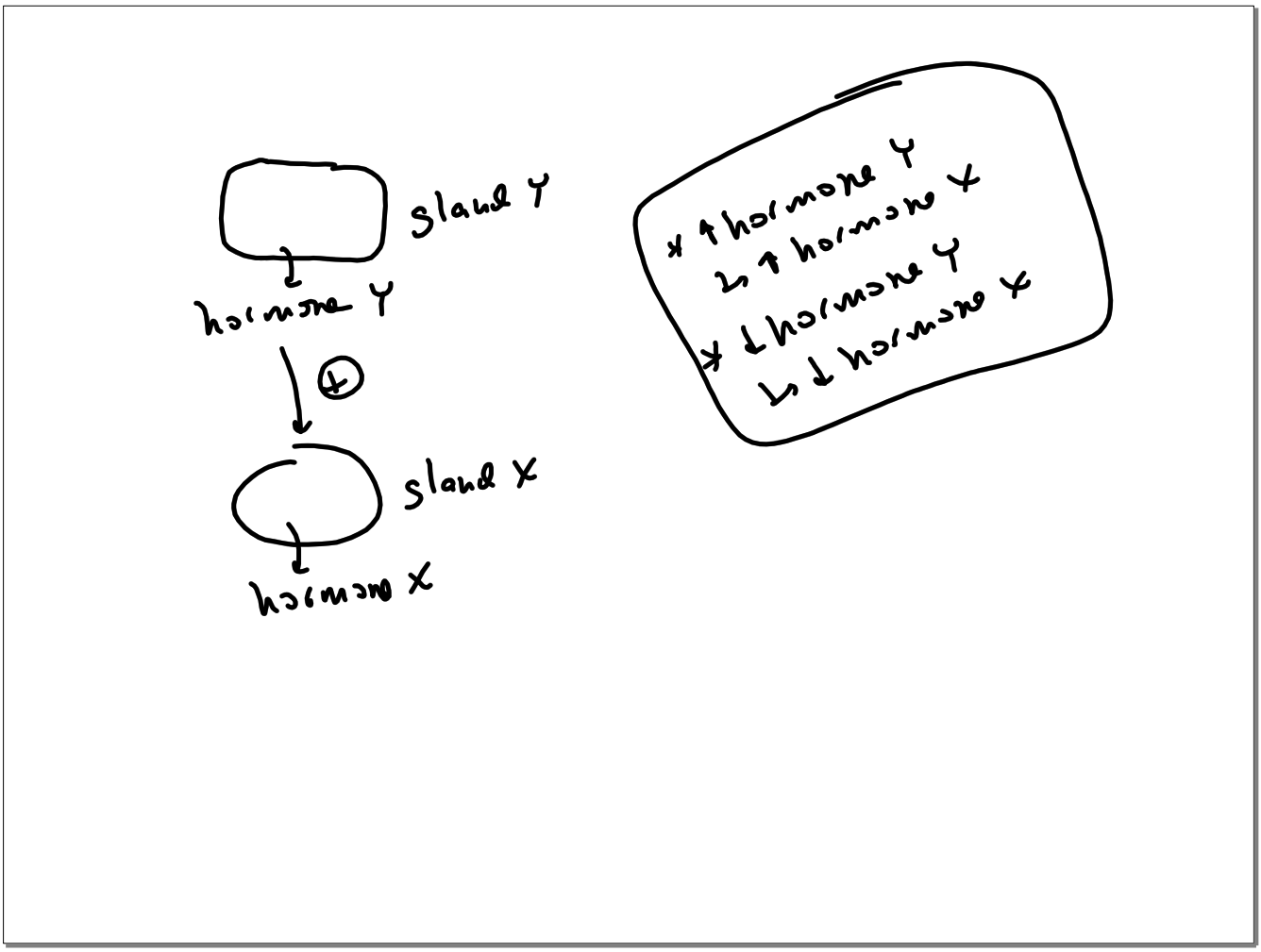
chronic release of hormone — all hormones

stimulus ("unplanned")

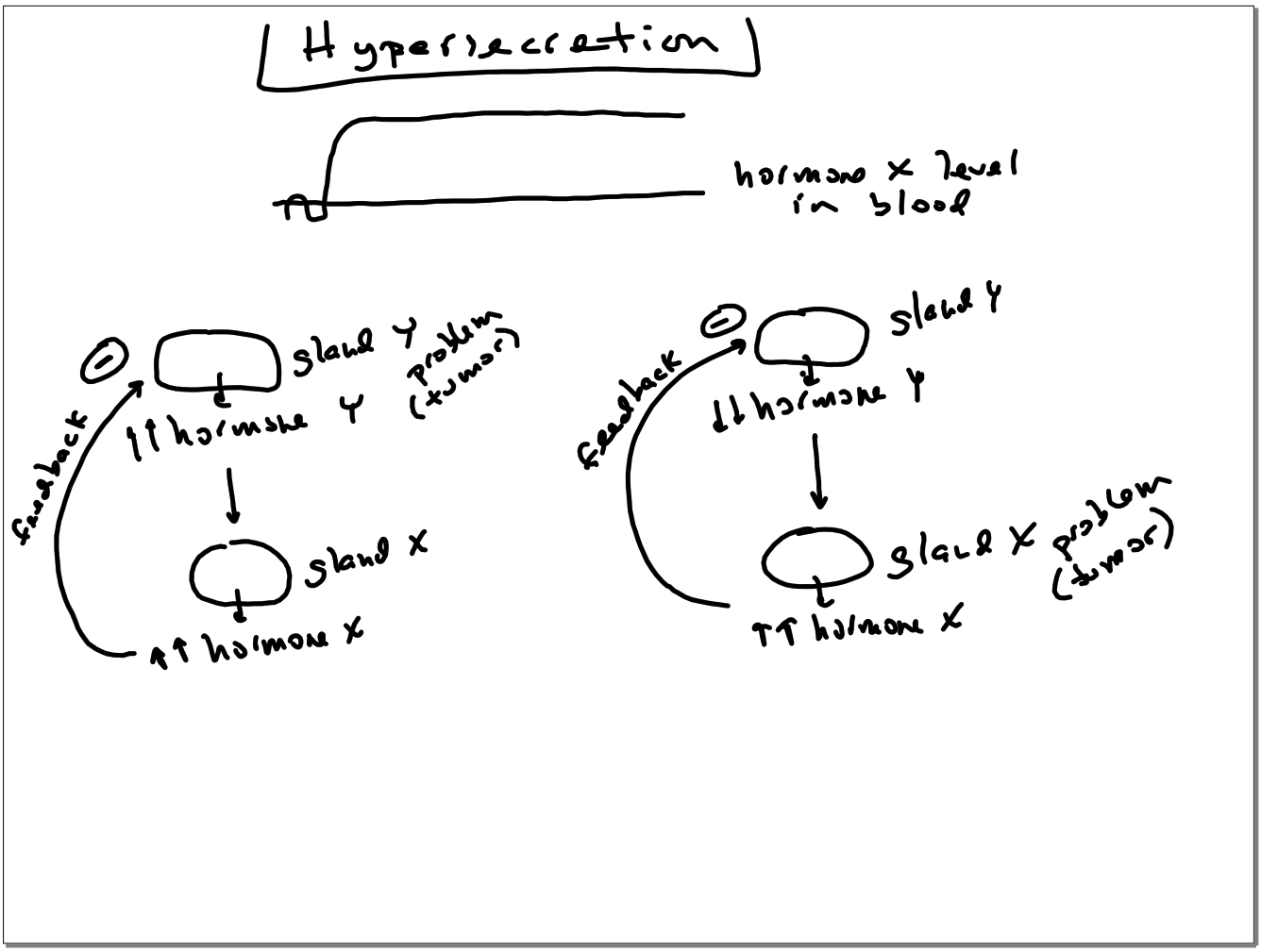
acute release of hormone — all hormones

stimulus ("planned"/"programmed")

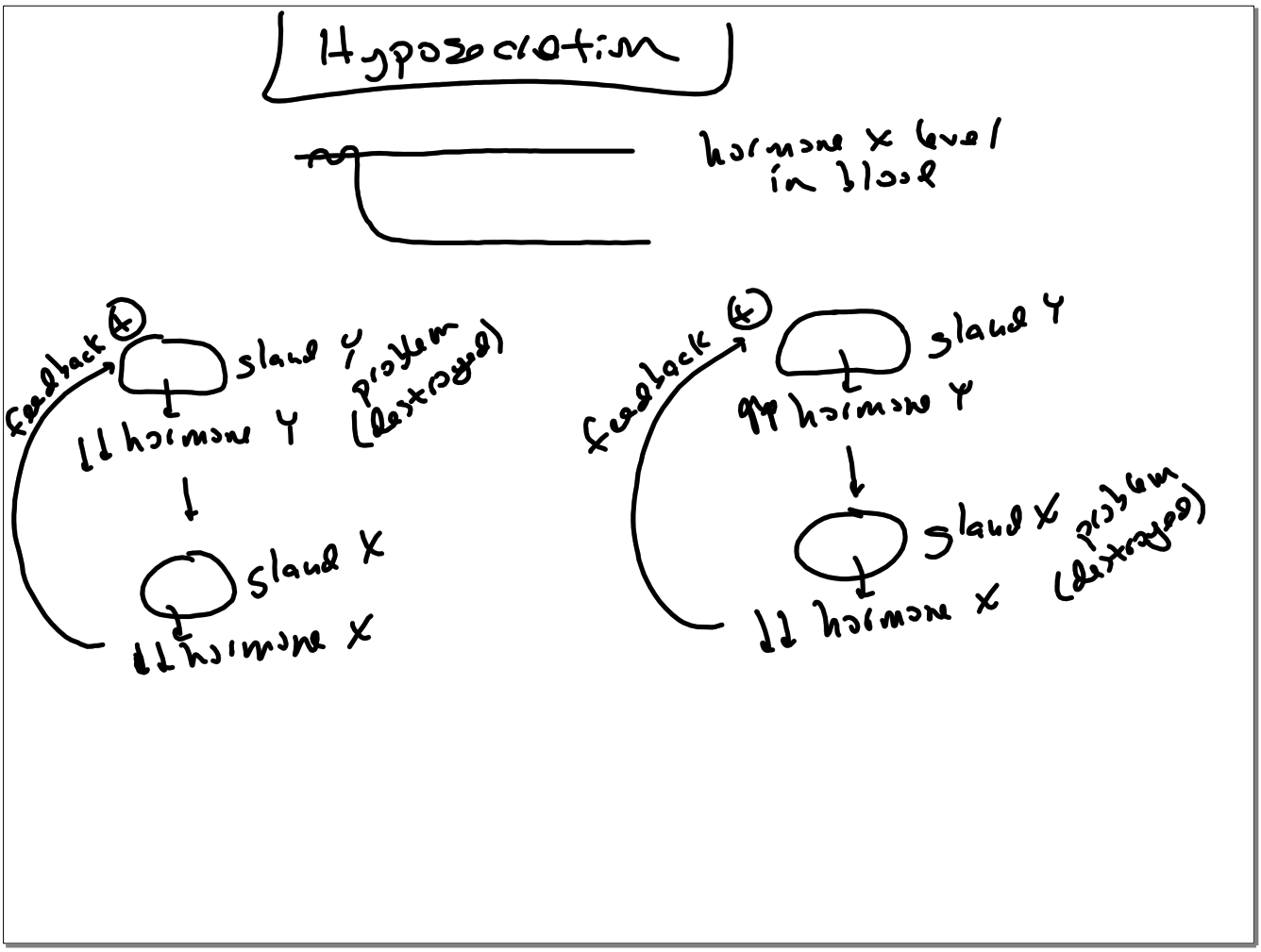
cyclic release of hormone — some hormones


gland Y
hormone Y
+
gland X
hormone X
* ↑ hormone Y
↳ ↑ hormone X
* ↓ hormone Y
↳ ↓ hormone X

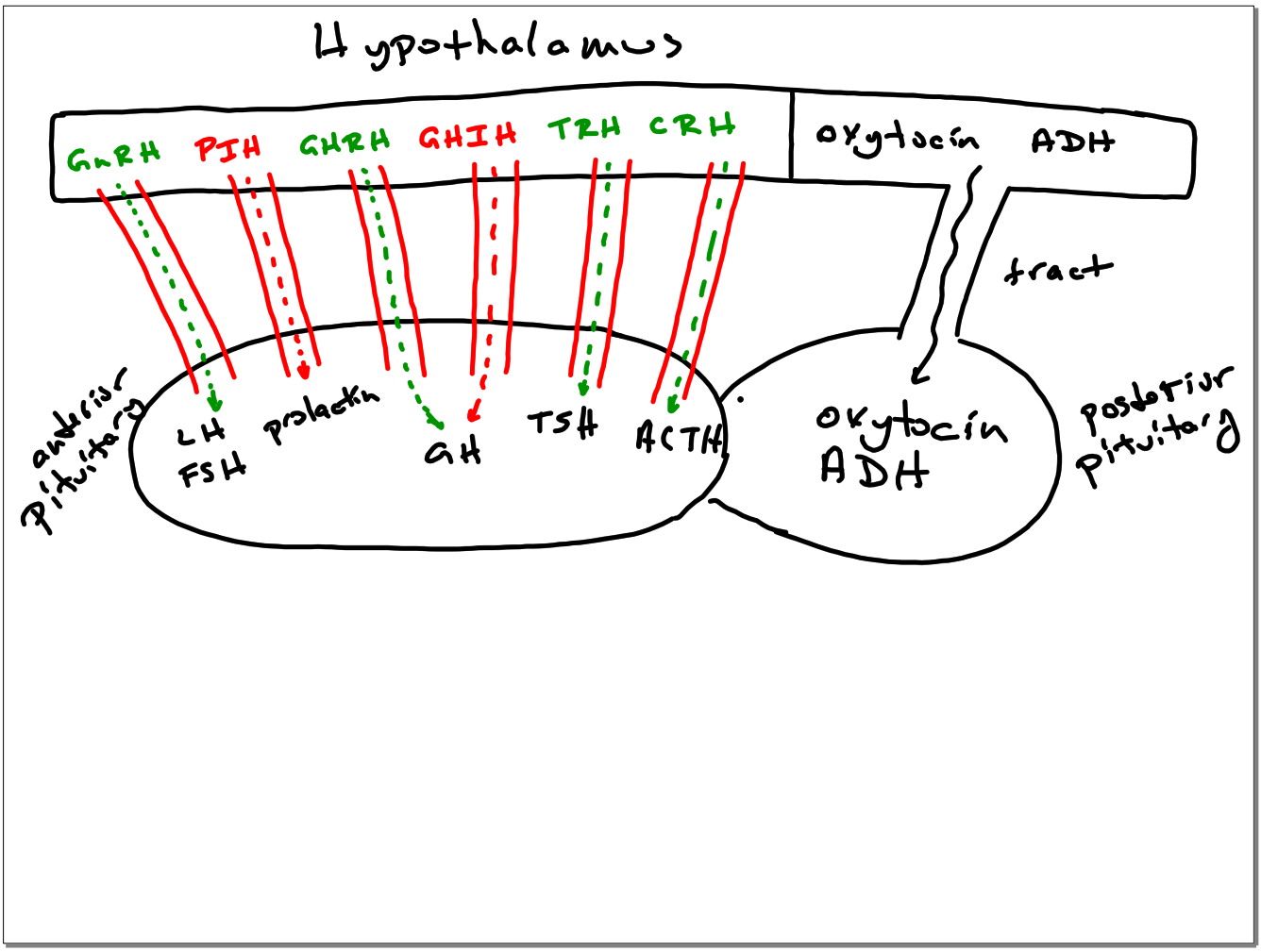
Jan 11-5:59 PM


Hypersecretion
hormone x level in blood
gland Y problem (tumor)
↑↑ hormone Y
gland X
↑↑ hormone X
feedback
gland Y
↑↑ hormone Y
gland X problem (tumor)
↑↑ hormone X
feedback

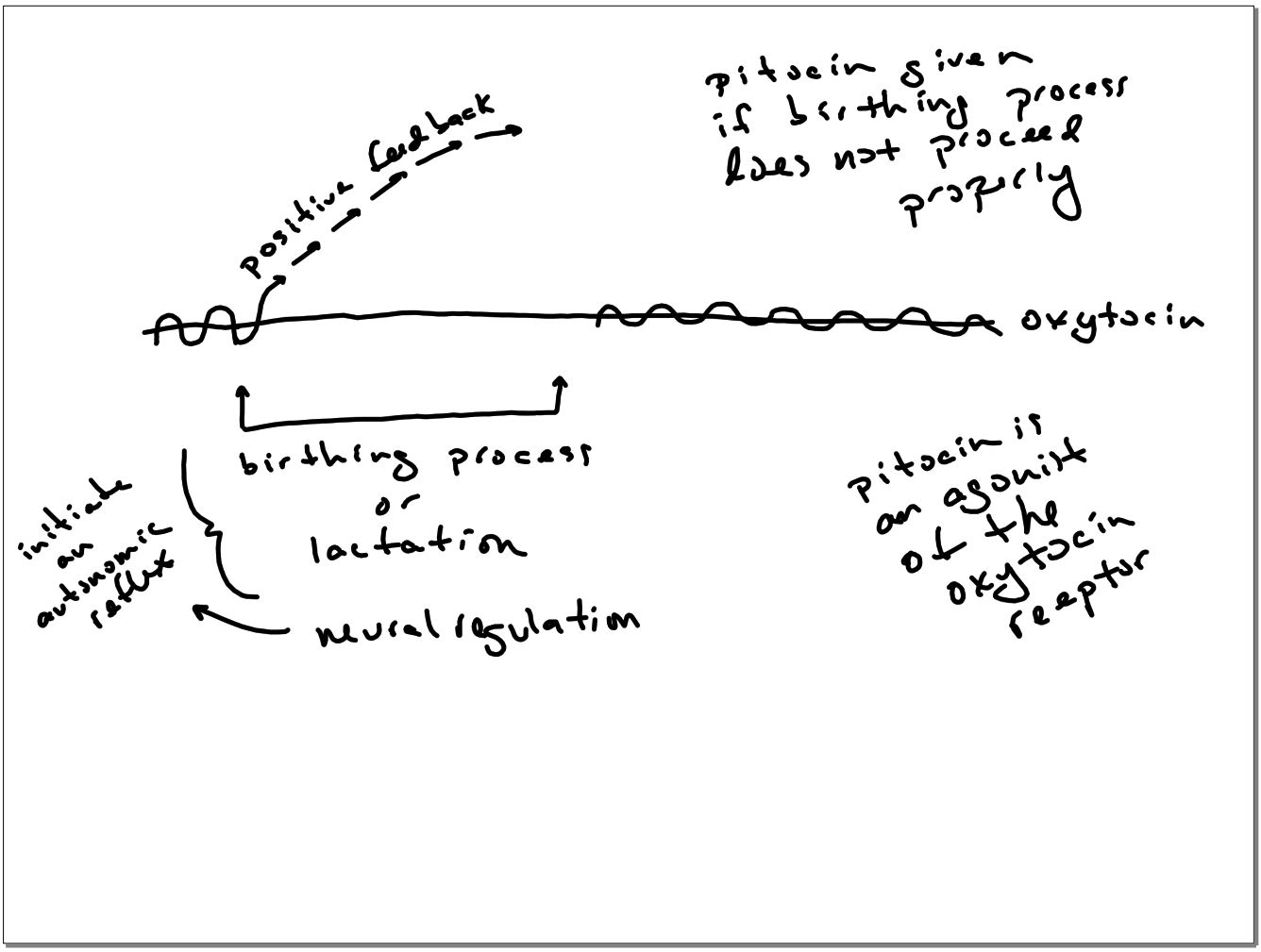
Jan 11-6:01 PM


Hypothalamus
hormone X level in blood
feedback
gland Y
problem (destroyed)
hormone Y
gland X
hormone X
feedback
gland Y
hormone Y
gland X
problem (destroyed)
hormone X

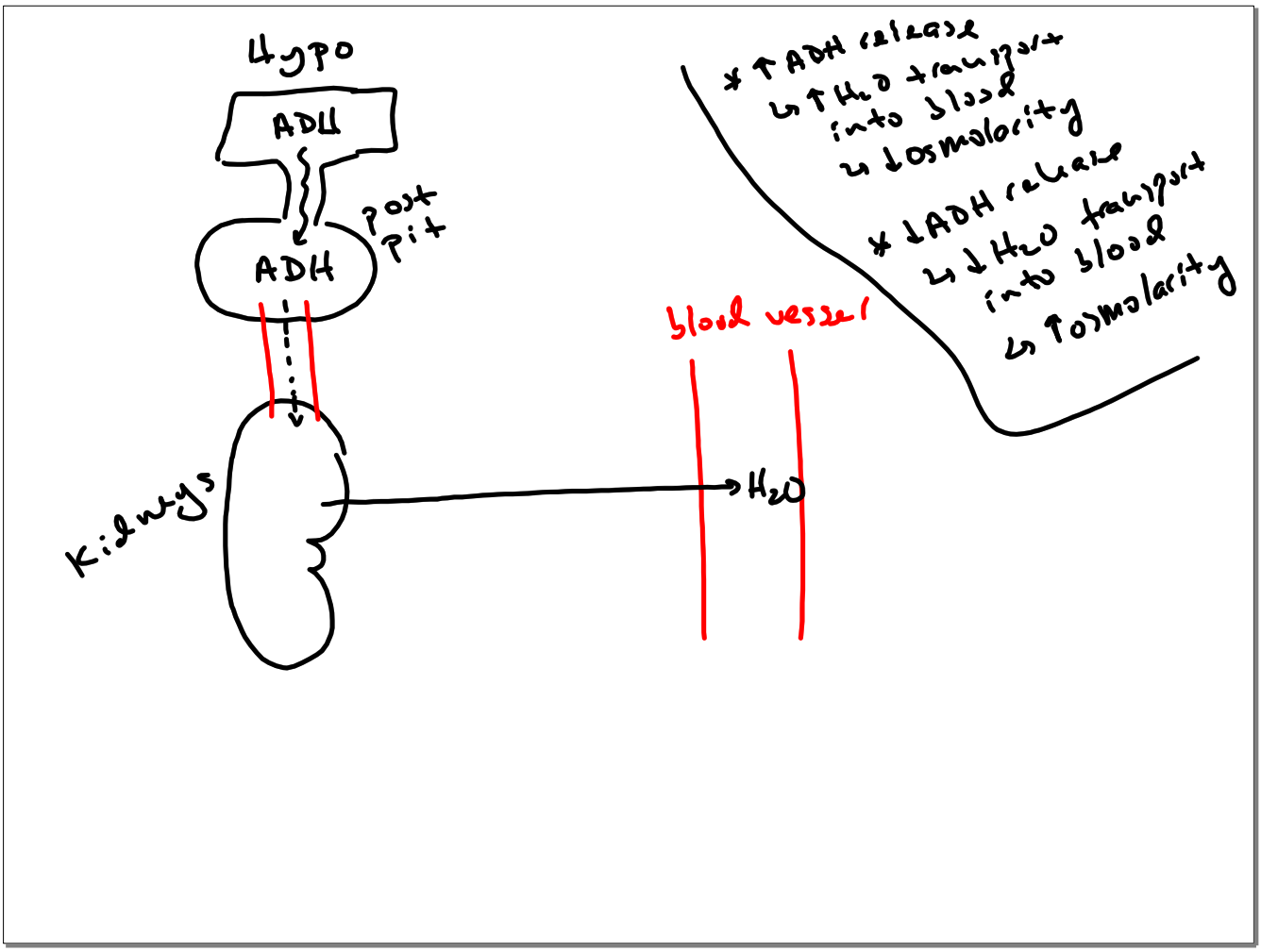
Jan 11-6:09 PM

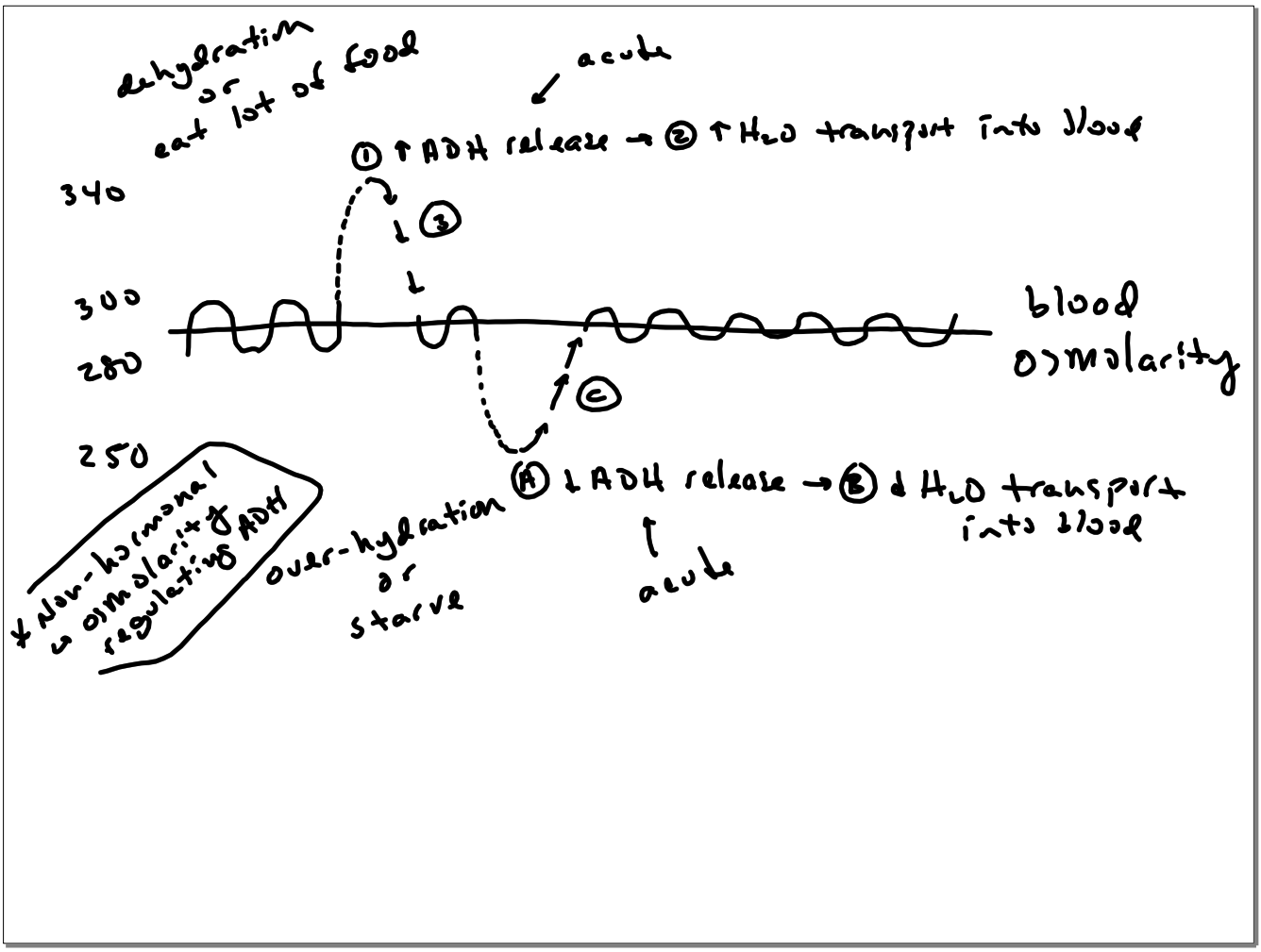

Hypothalamus
GnRH
PIH
GHRH
GHIH
TRH
CRH
oxytocin
ADH
tract
anterior pituitary
LH
FSH
prolactin
GH
TSH
ACTH
oxytocin
ADH
posterior pituitary

Jan 11-6:14 PM


pitocin given if birthing process does not proceed properly
positive feedback
oxytocin
birthing process or lactation
initiate an autonomic reflex
neural regulation
pitocin is an agonist of the oxytocin receptor

Jan 13-4:56 PM

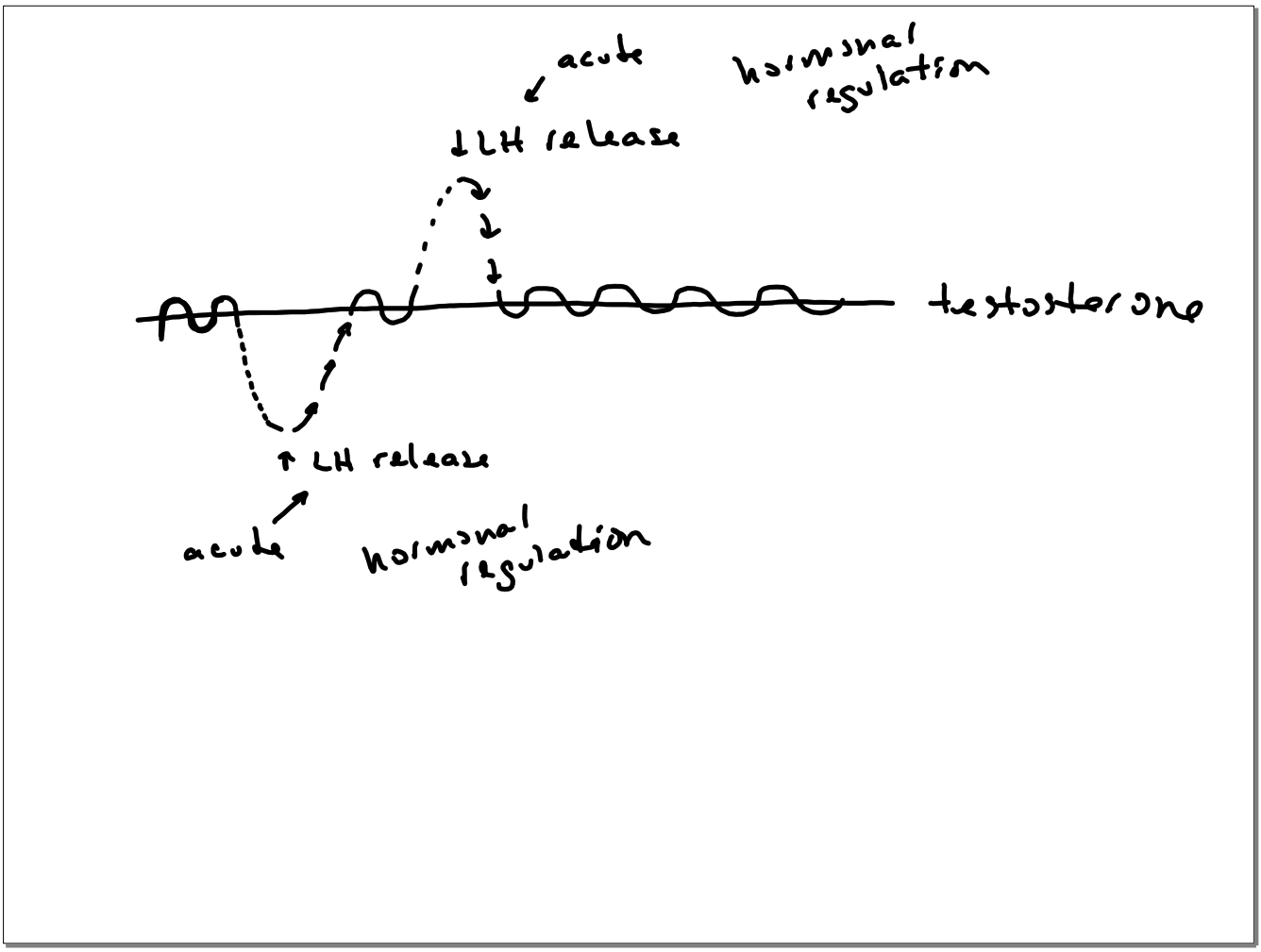

Hypo
ADH
Post Pit
ADH
Kidneys
H_2O
blood vessel
* ↑ ADH release
↳ ↑ H_2O transport into blood
↳ ↓ osmolarity
* ↓ ADH release
↳ ↓ H_2O transport into blood
↳ ↑ osmolarity


dehydration
or
eat lot of food
acute
① ↑ ADH release → ② ↑ H_2O transport into blood
340
300
280
250
③
blood
osmolarity
④ ↓ ADH release → ⑤ ↓ H_2O transport into blood
over-hydration
or
starve
acute
* Non-hormonal ↓ osmolarity regulating ADH

Jan 13-5:16 PM

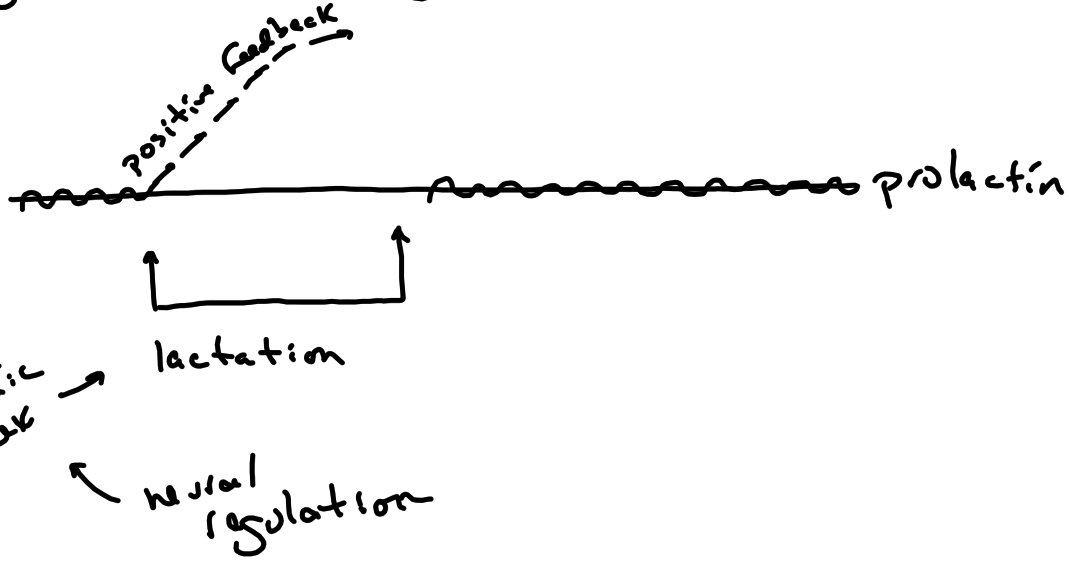
SIAD (hypersecretion)	Diabetes Insipidus (hyposecretion)
↑↑ ADH ↓ ↑↑ H_2O transport into blood ↓ ↓↓ osmolarity	↓↓ ADH ↓ ↓↓ H_2O transport into blood ↓ ↑↑ osmolarity

Jan 13-5:24 PM

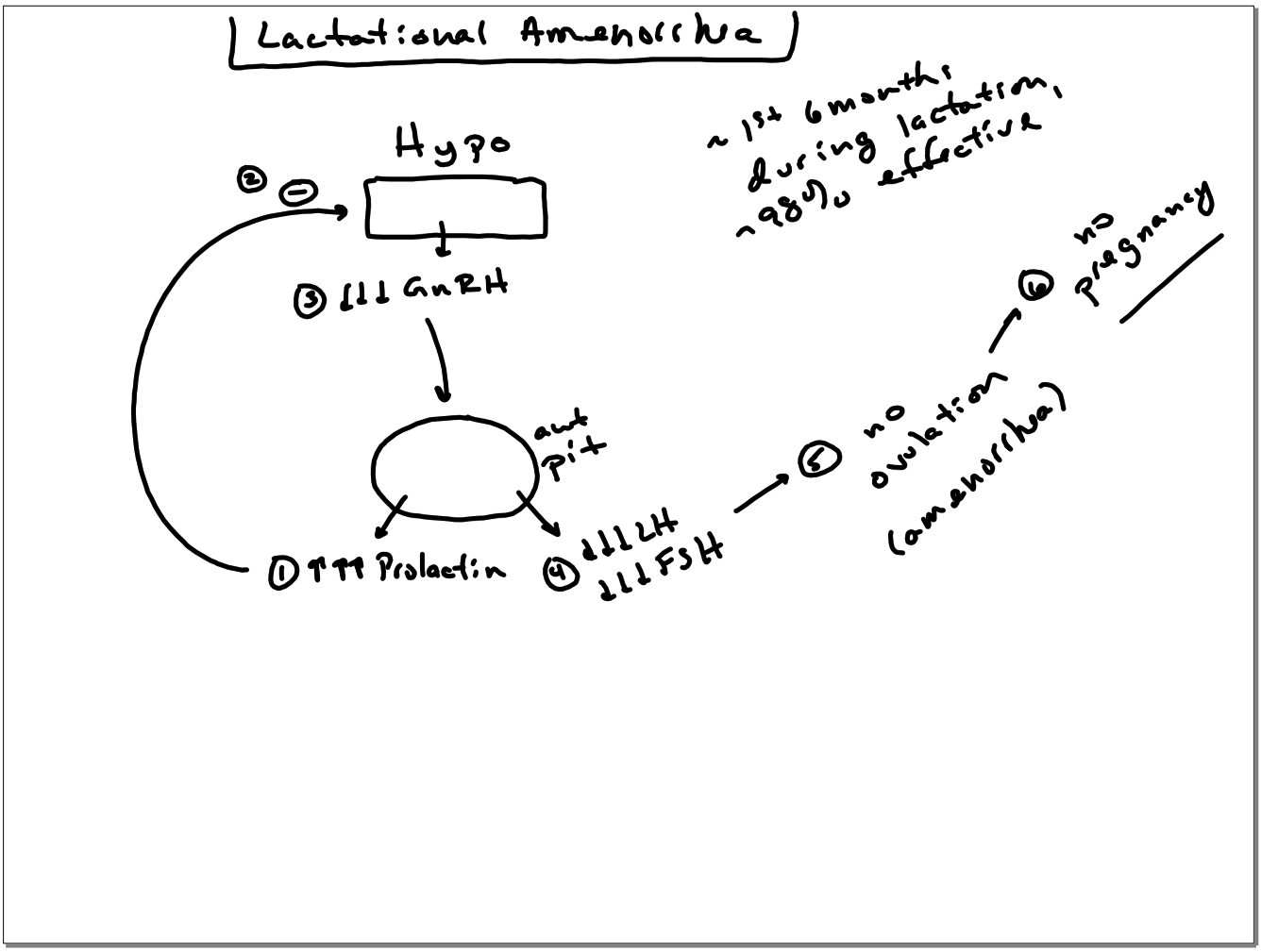

acute
hormonal regulation
↓ LH release
testosterone
↑ LH release
acute
hormonal regulation

Jan 13-5:28 PM

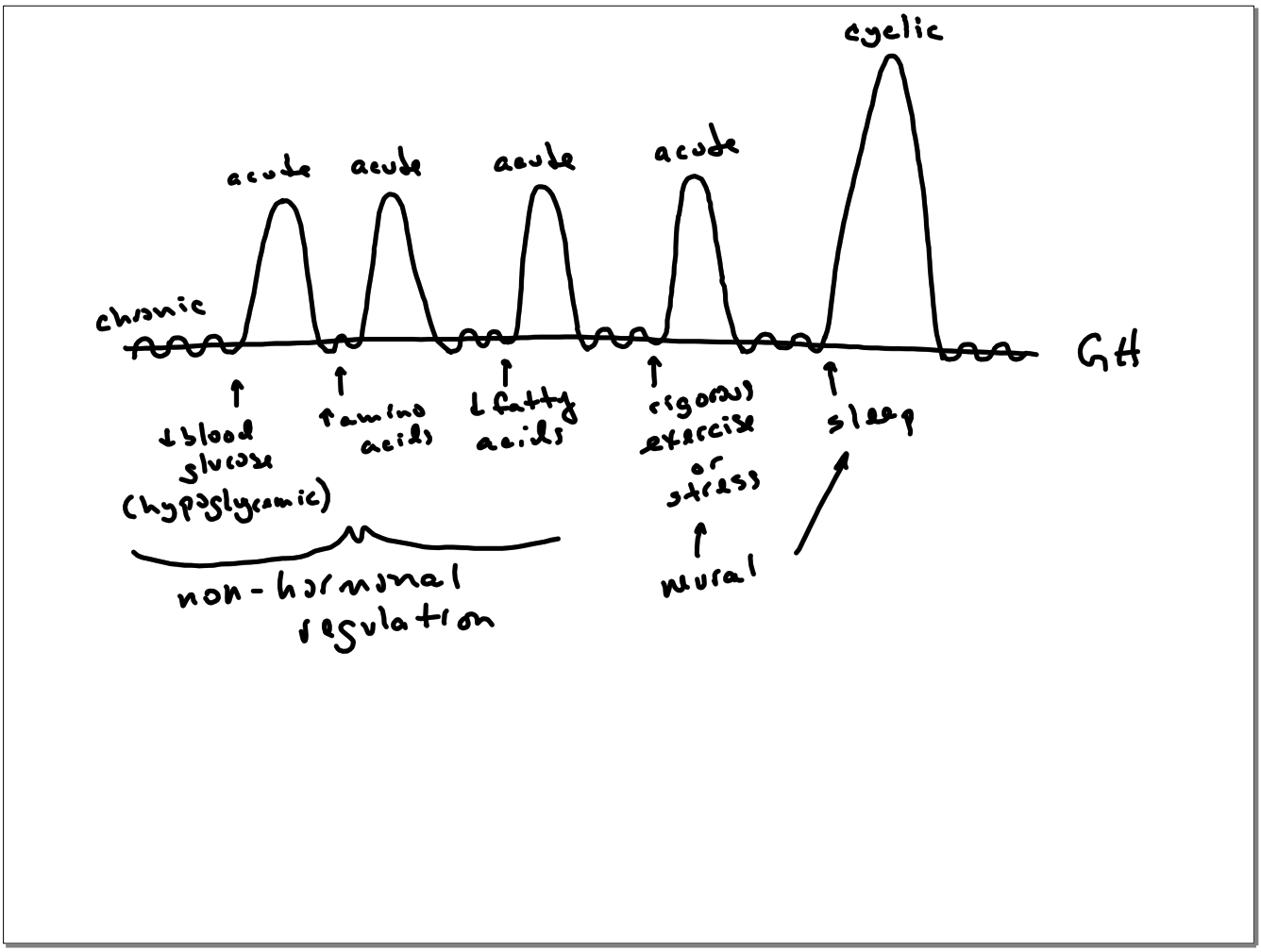
* Prolactin is low
  - why? ← continually inhibited by PIH



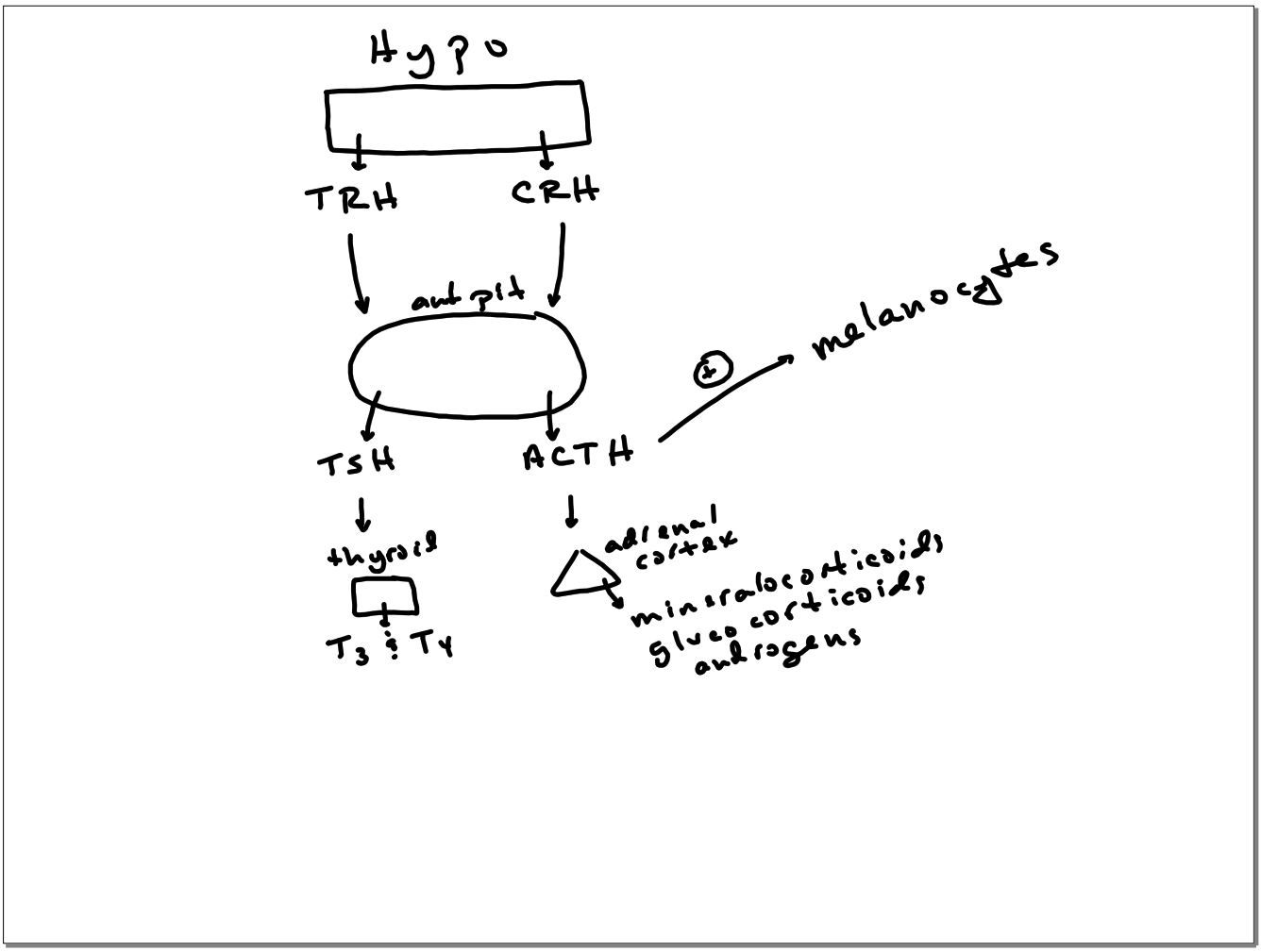
Jan 13-5:42 PM


Lactational Amenorrhea
~ 1st 6 months during lactation, ~98% effective
Hypo
② (-)
③ ↓↓↓ GnRH
ant Pit
① ↑↑↑ Prolactin
④ ↓↓↓ LH ↓↓↓ FSH
⑤ no ovulation (amenorrhea)
⑥ no pregnancy

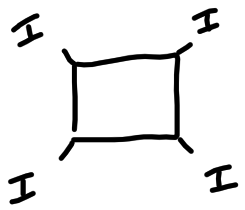
Jan 13-5:50 PM


cyclic
acute
acute
acute
acute
chronic
GH
↓blood glucose (hypoglycemic)
↑amino acids
↓fatty acids
rigorous exercise or stress
sleep
neural
non-hormonal regulation

Jan 13-5:58 PM

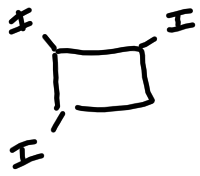

Hypo
TRH
CRH
ant pit
TSH
ACTH
+
melanocytes
thyroid
T3 & T4
adrenal cortex
mineralocorticoids
gluco corticoids
androgens

Jan 18-4:56 PM



thyroxine (T_4)

~ 90%



triiodothyronine (T_3)

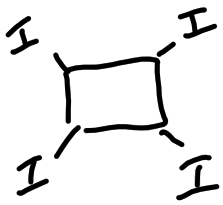
~ 10%

~ 5 x's more potent

T_3 & T_4 are stored in thyroid gland (~3 months worth)

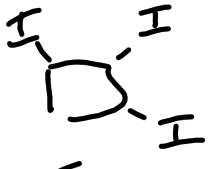
Jan 18-5:07 PM

occurs in thyroid and most tissues of body



T_4

deionization →



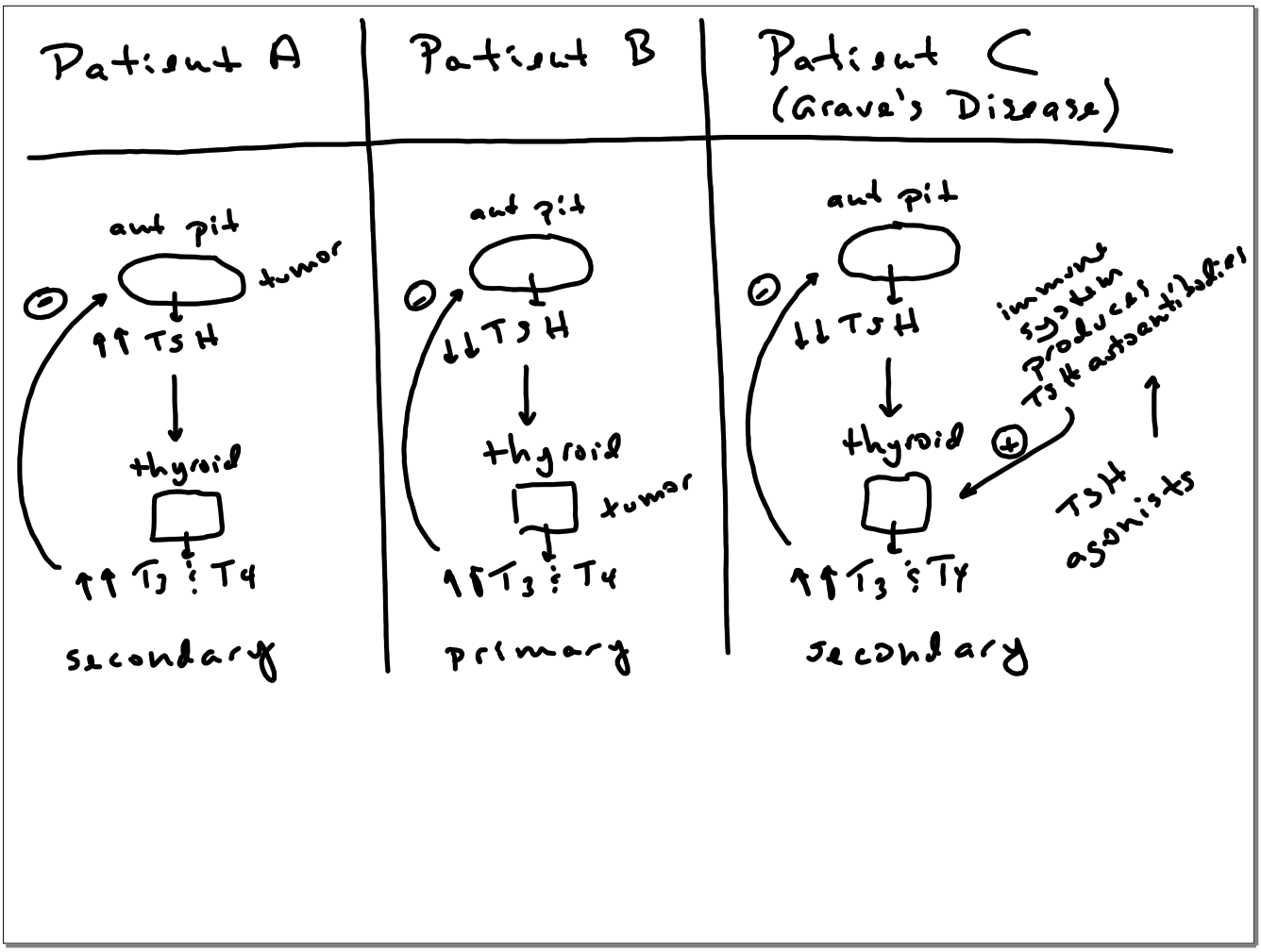
T_3

most T_3 exists in that manner

Patient A
Patient B
Patient C

Unexplained weight loss
Fidgety
High pulse
Goiter
Sweaty

↑ Hyperthyroidism


Patient A
Patient B
Patient C
(Grave's Disease)
ant pit
tumor
↑↑ TSH
thyroid
↑↑ T3 ; T4
secondary
ant pit
↓↓ TSH
thyroid
tumor
↑↑ T3 ; T4
primary
ant pit
↓↓ TSH
thyroid
immune system produces TSH autoantibodies
TSH agonists
↑↑ T3 ; T4
secondary

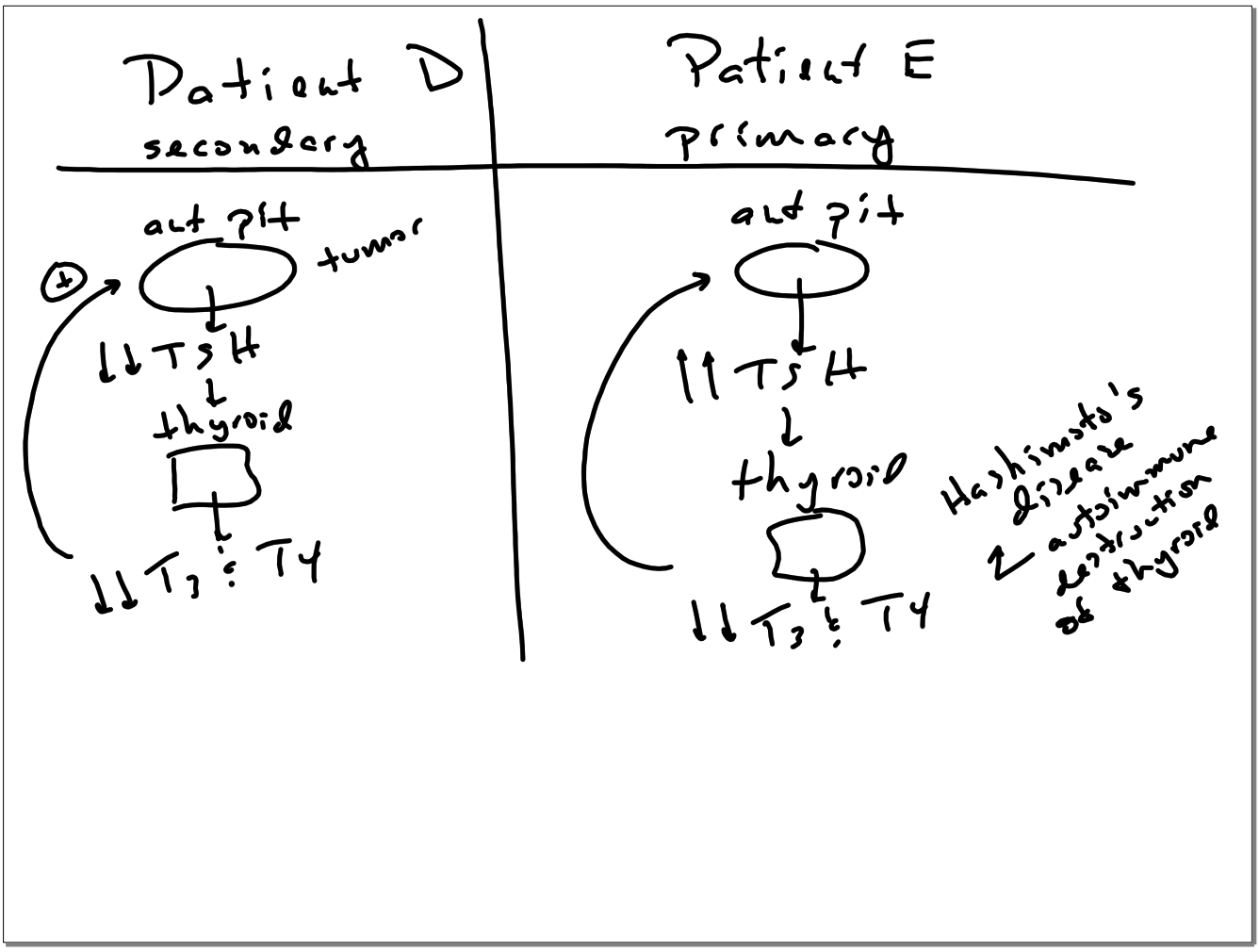
Jan 18-5:22 PM

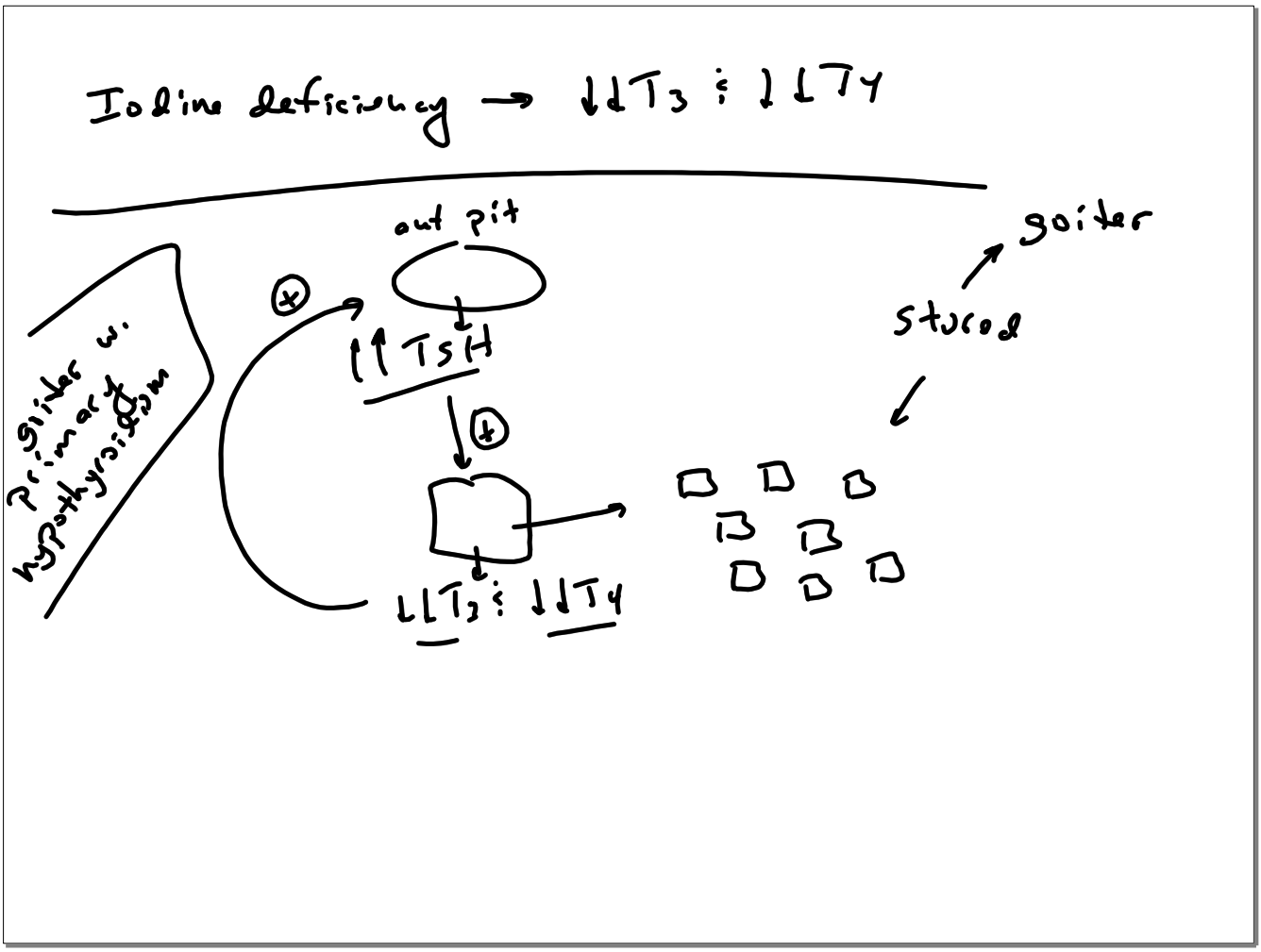
Patient D
Low pulse
Lethargic
Constipation

Patient E
Unexplained weight gain
High cholesterol

↑ Hypothyroidism

Jan 18-5:36 PM


Patient D
secondary
ant pit
tumor
+
↓↓TSH
thyroid
↓↓T3 : T4
Patient E
primary
ant pit
↑↑TSH
thyroid
↓↓T3 : T4
Hashimoto's disease
↳ autoimmune destruction of thyroid

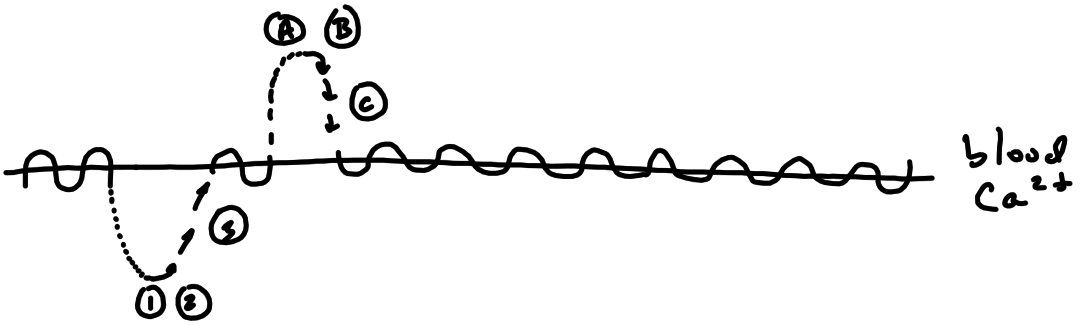

Iodine deficiency → ↓↓T3 : ↓↓T4
anterior pit
goiter
stored
TSH
LLT3 : ↓↓T4

Jan 18-5:49 PM

* Treatments
  - hyperthyroidism
    - ↳ anti-thyroid meds
    - ↳ thyroidectomy
  - hypothyroidism
    - ↳ synthroid (T_4)

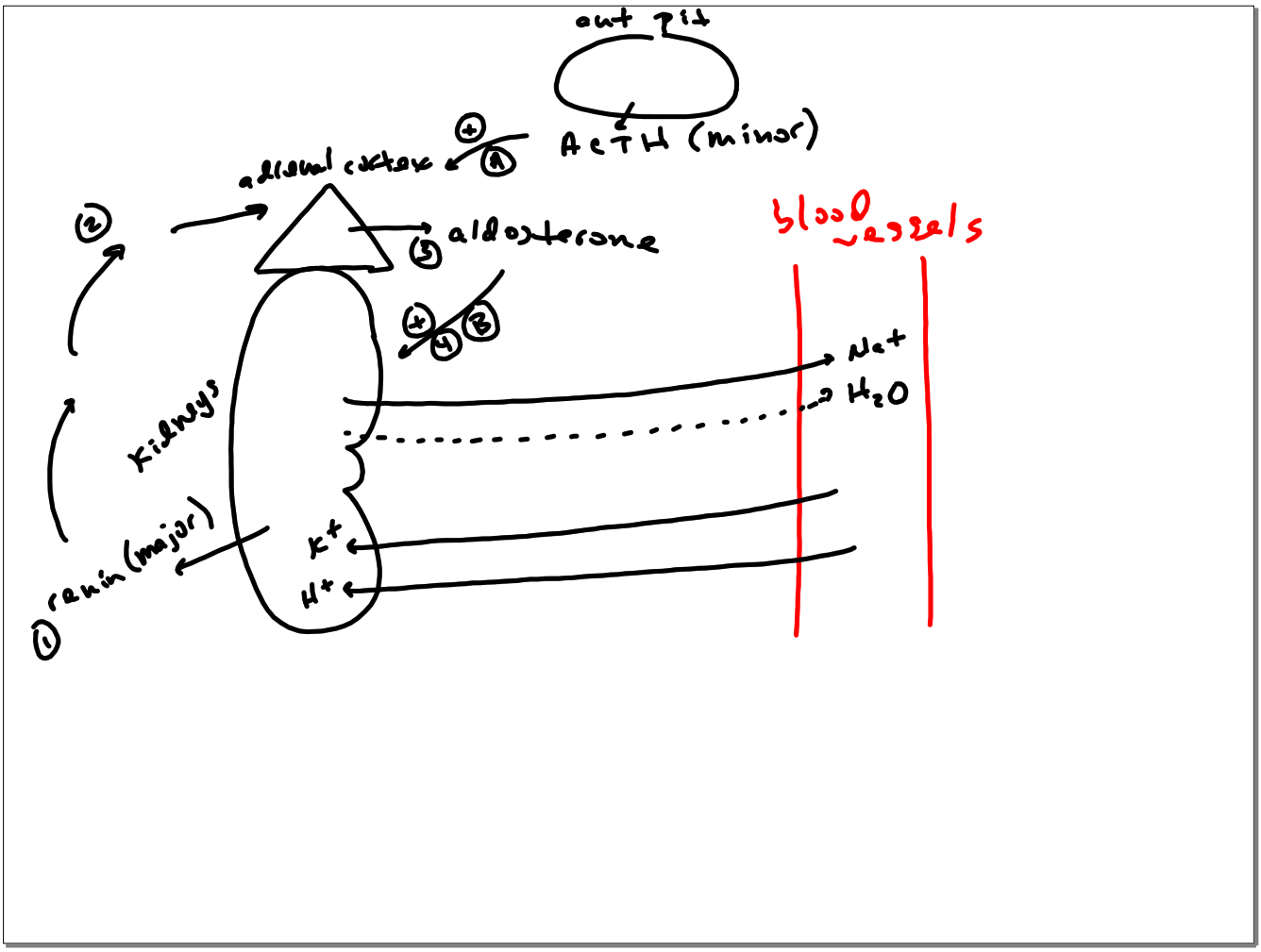
Jan 18-5:50 PM

Ⓐ ↑↑ blood Ca^{2+} → Ⓑ ↓↓ release of PTH → Ⓒ ↑ bone production
↓ Ca^{2+} reabsorption by kidneys
↓ Ca^{2+} absorption in gut

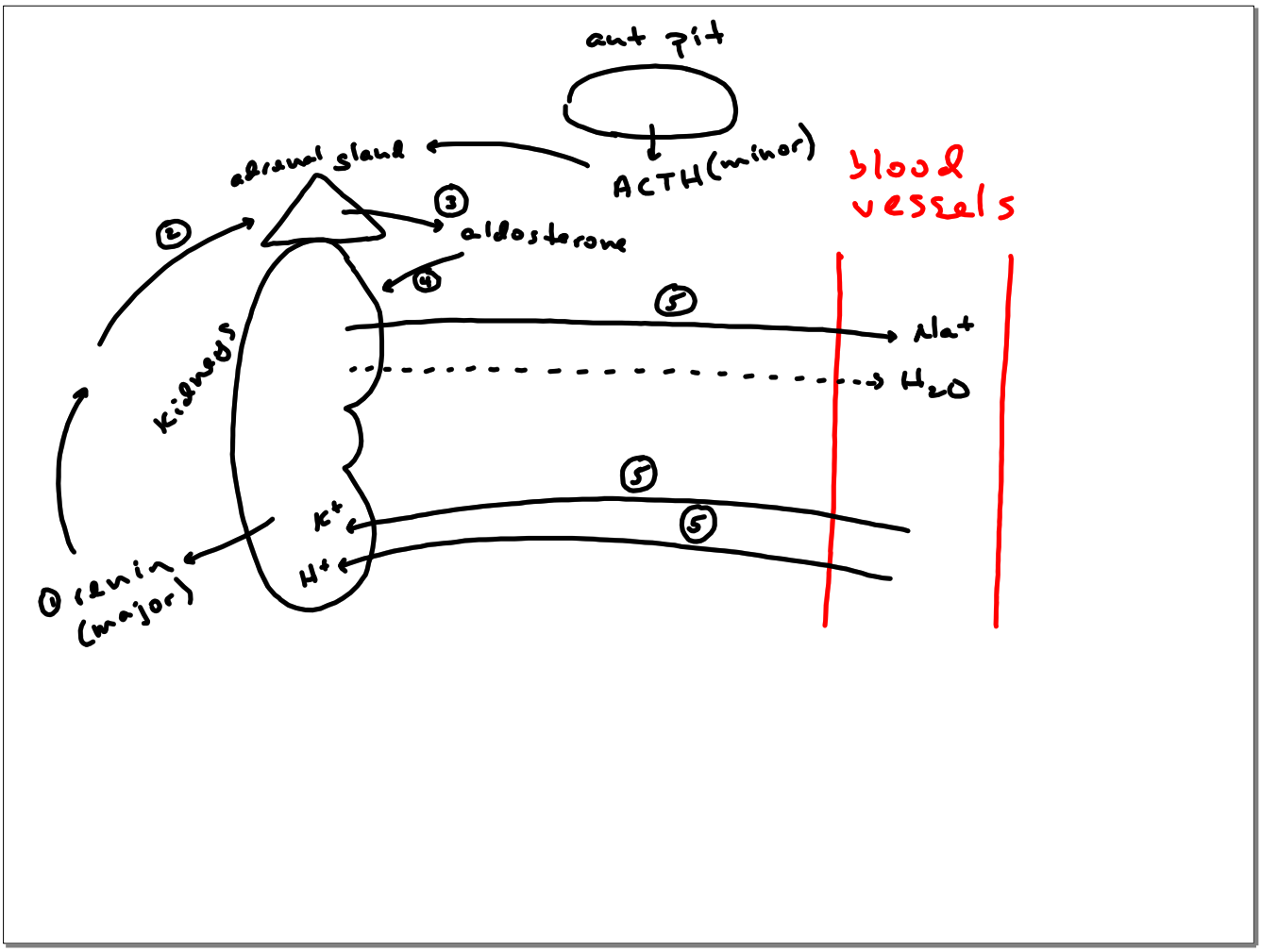


① ↓ blood Ca^{2+} → ② ↑↑ release of PTH → ③ ↑ bone resorption
↑ Ca^{2+} reabsorption by kidneys
↑ Ca^{2+} absorption in gut

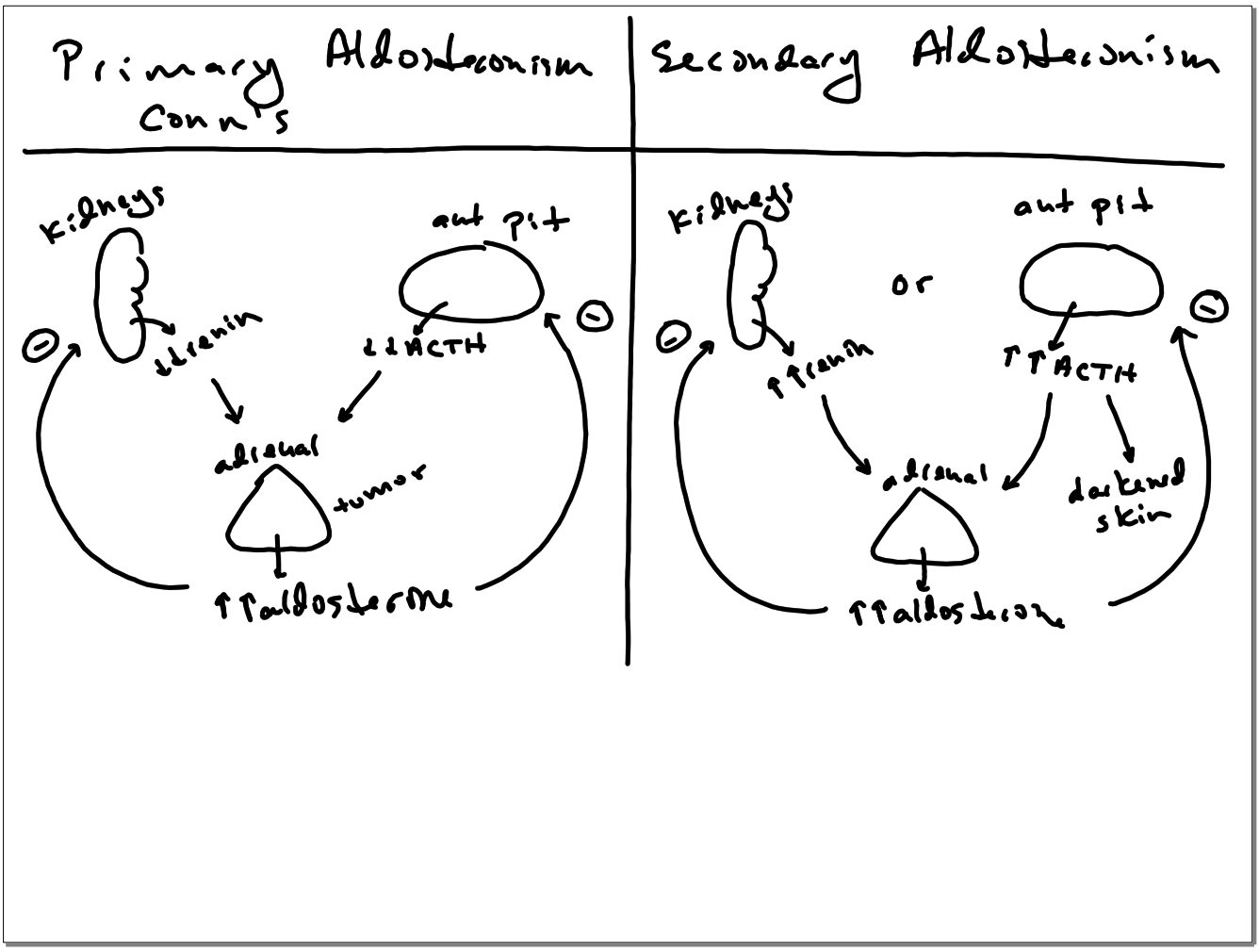
Jan 18-5:58 PM


ant Pit
ACTH (minor)
adrenal cortex
aldosterone
blood vessels
Na+
H2O
kidneys
renin (major)
K+
H+

Jan 18-6:04 PM

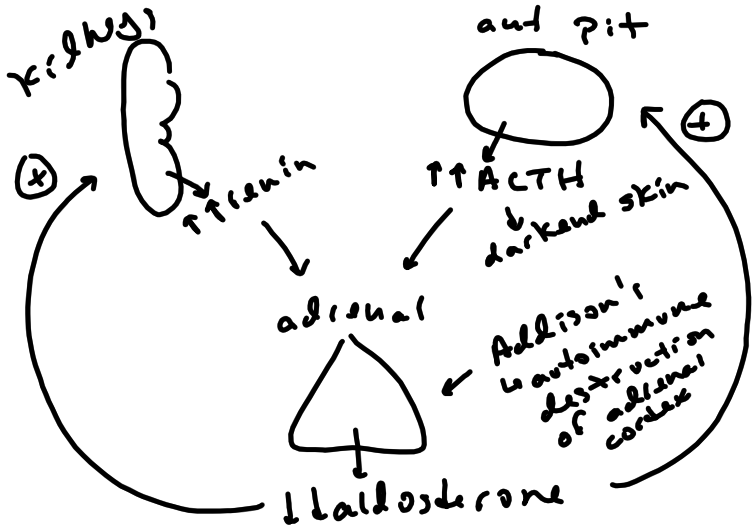

ant pit
ACTH(minor)
adrenal gland
aldosterone
blood vessels
kidneys
Na+
H2O
K+
H+
(major)

Jan 20-4:54 PM

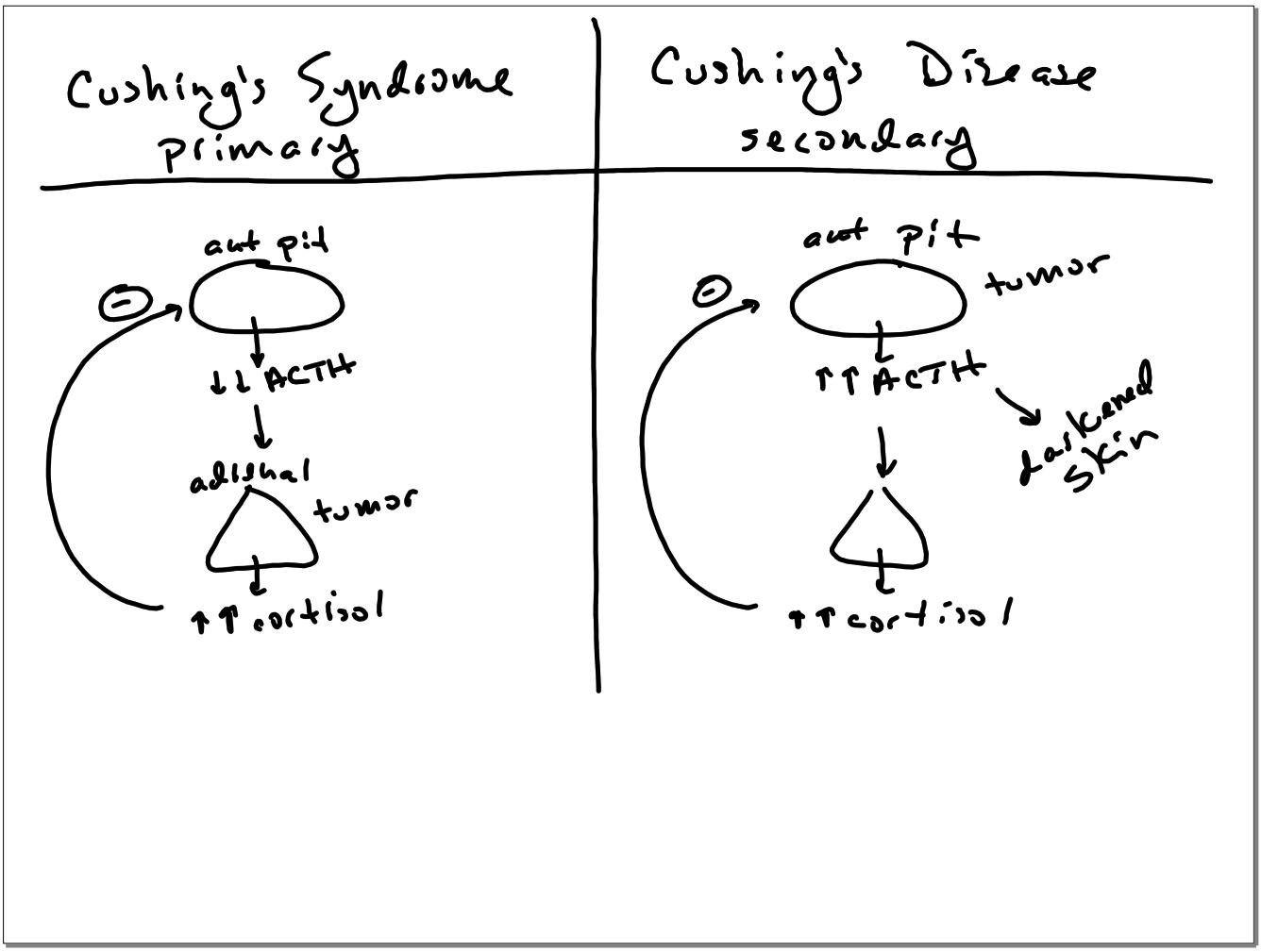

Primary Aldosteronism
Conn's
Secondary Aldosteronism
kidneys
renin
ant pit
ACTH
adrenal
tumor
aldosterone
kidneys
renin
or
ant pit
ACTH
adrenal
darkened skin
aldosterone

Jan 20-5:07 PM

Primary Adrenal Insufficiency



Jan 20-5:14 PM


Cushing's Syndrome
primary
ant pit
↓↓ ACTH
adrenal
tumor
↑↑ cortisol
Cushing's Disease
secondary
ant pit
tumor
↑↑ ACTH
darkened skin
↑↑ cortisol

Jan 20-5:30 PM

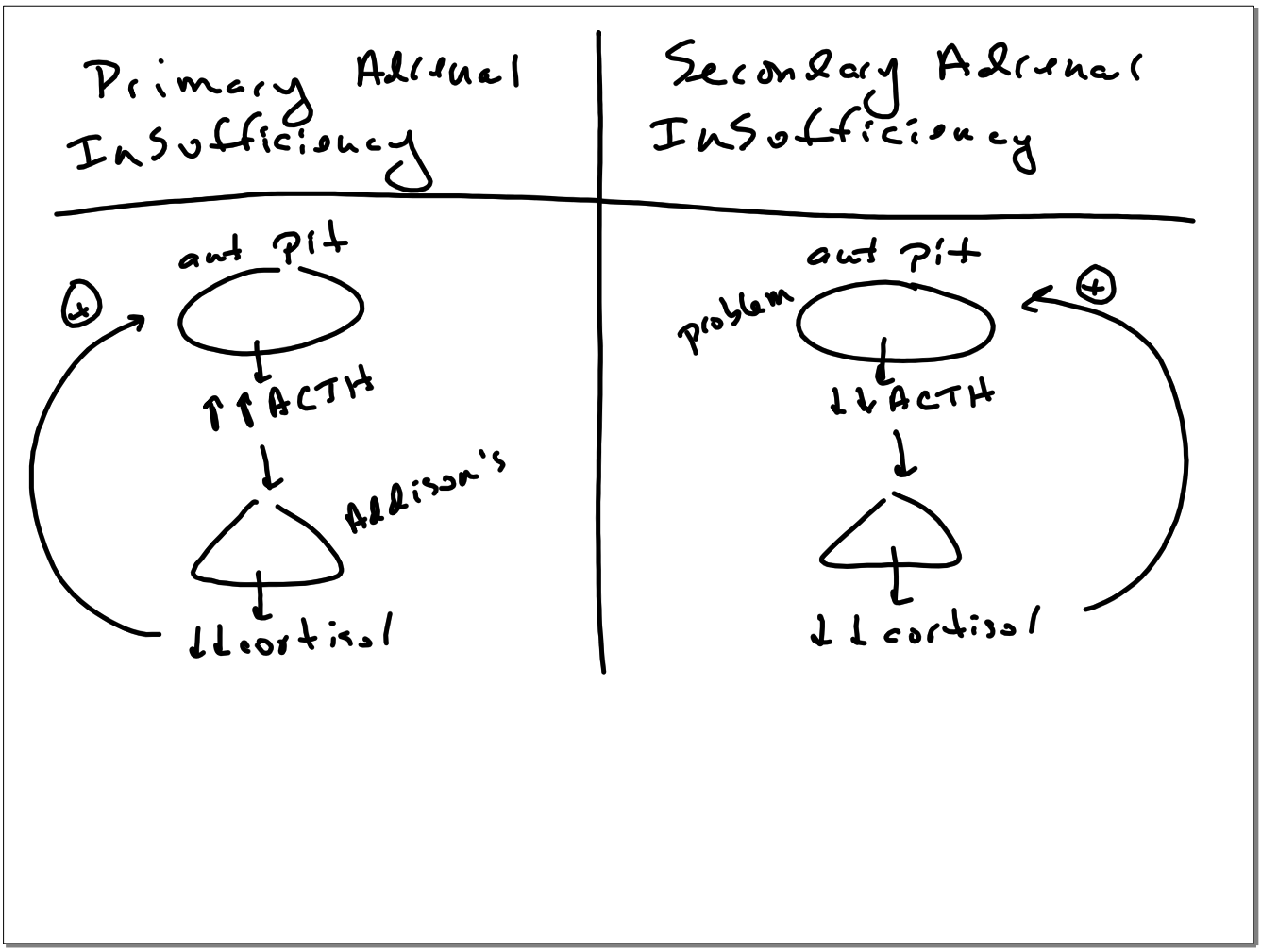
* Cushing's syndrome could be due to prolonged corticosteroid use at high doses

Prednisone
cortisone

* why prescribe?
  - autoimmune conditions
    - suppresses immune system
  - inflammation

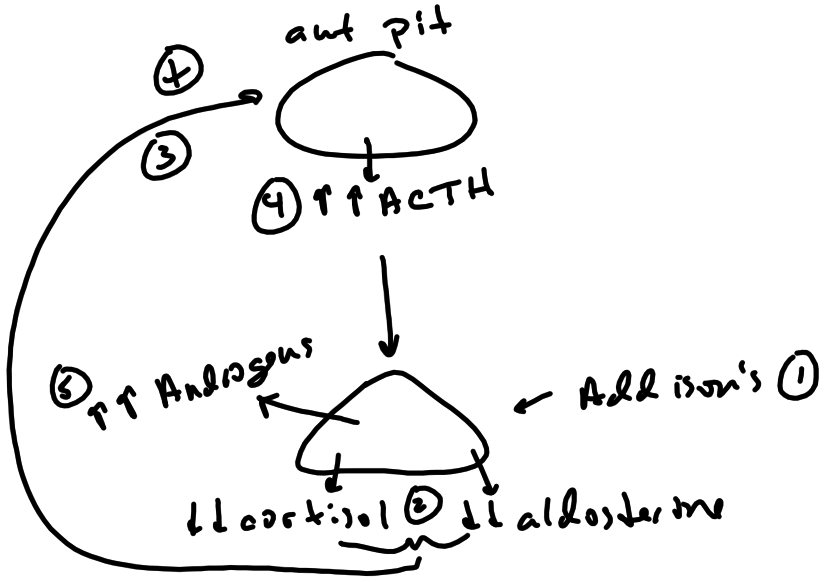
agonists of cortisol receptor

Jan 20-5:32 PM

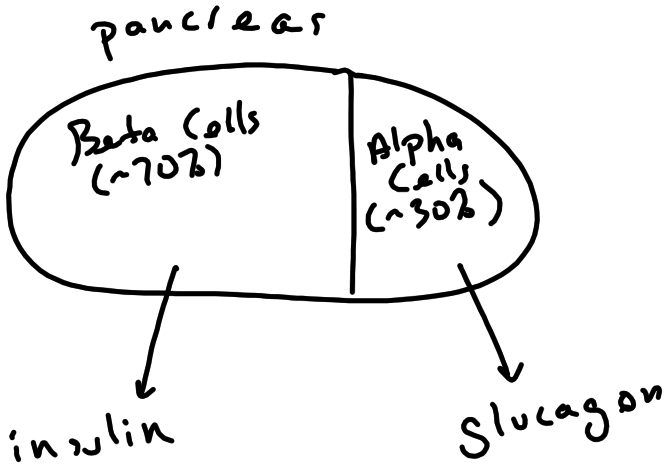

Primary Adrenal Insufficiency
ant pit
+
↑↑ACTH
Addison's
↓↓cortisol
Secondary Adrenal Insufficiency
ant pit
problem
+
↓↓ACTH
↓↓cortisol

Jan 20-5:39 PM

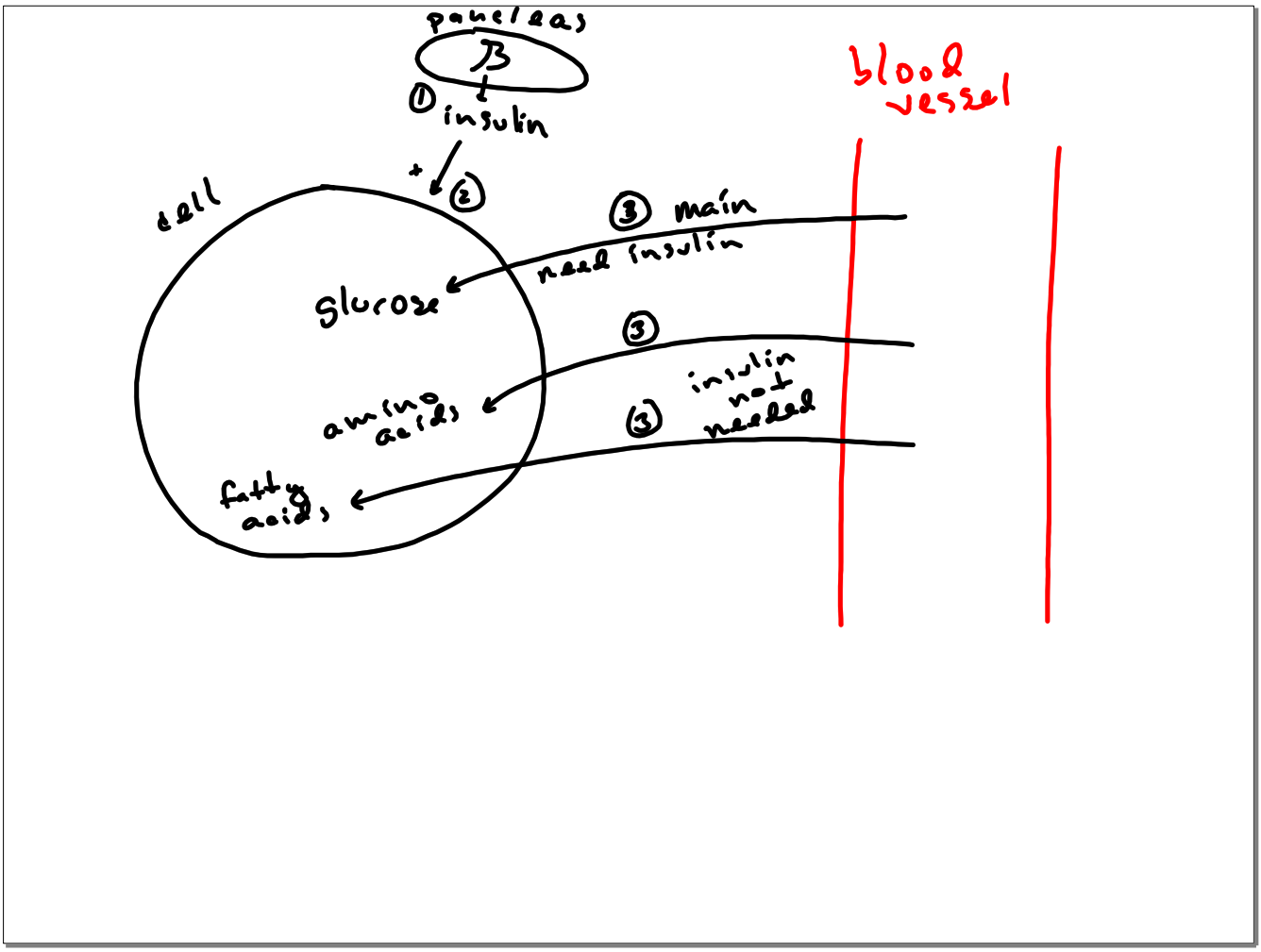
Adrenogenital Syndrome



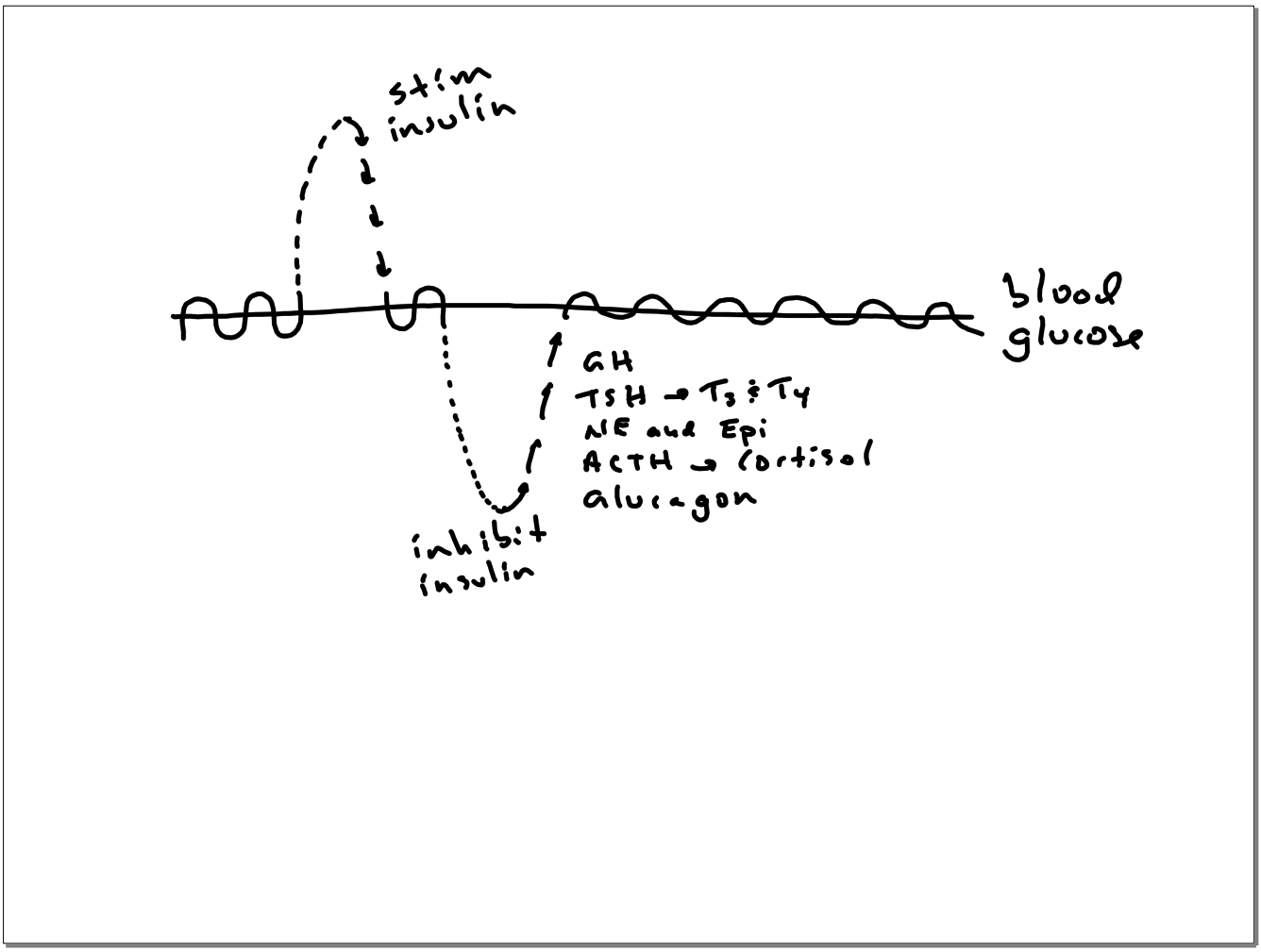
Jan 20-5:45 PM



Jan 20-5:52 PM


pancreas
β
① insulin
+
②
cell
③ main need insulin
glucose
③
amino acids
③ insulin not needed
fatty acids
blood vessel

Jan 20-5:53 PM


stim insulin
blood glucose
GH
TSH → T3 & T4
NE and Epi
ACTH → Cortisol
Glucagon
inhibit insulin

Jan 20-5:56 PM

Type 1 Diabetes ~5%	Type 2 Diabetes ~95%
* Cause: autoimmune destruction of B-cells ↳ no insulin * Treatment: insulin ↑ insulin-dependent	* Cause: insulin resistance ↳ typically due to poor lifestyle * Treatments: - better lifestyle - meds

* Problems w. untreated diabetes arise from ↑↑↑ blood sugar (hyperglycemia)

↳ inflammation/damage to blood vessel

"glass"

cardiovascular disease

↓↓ blood flow to tissues

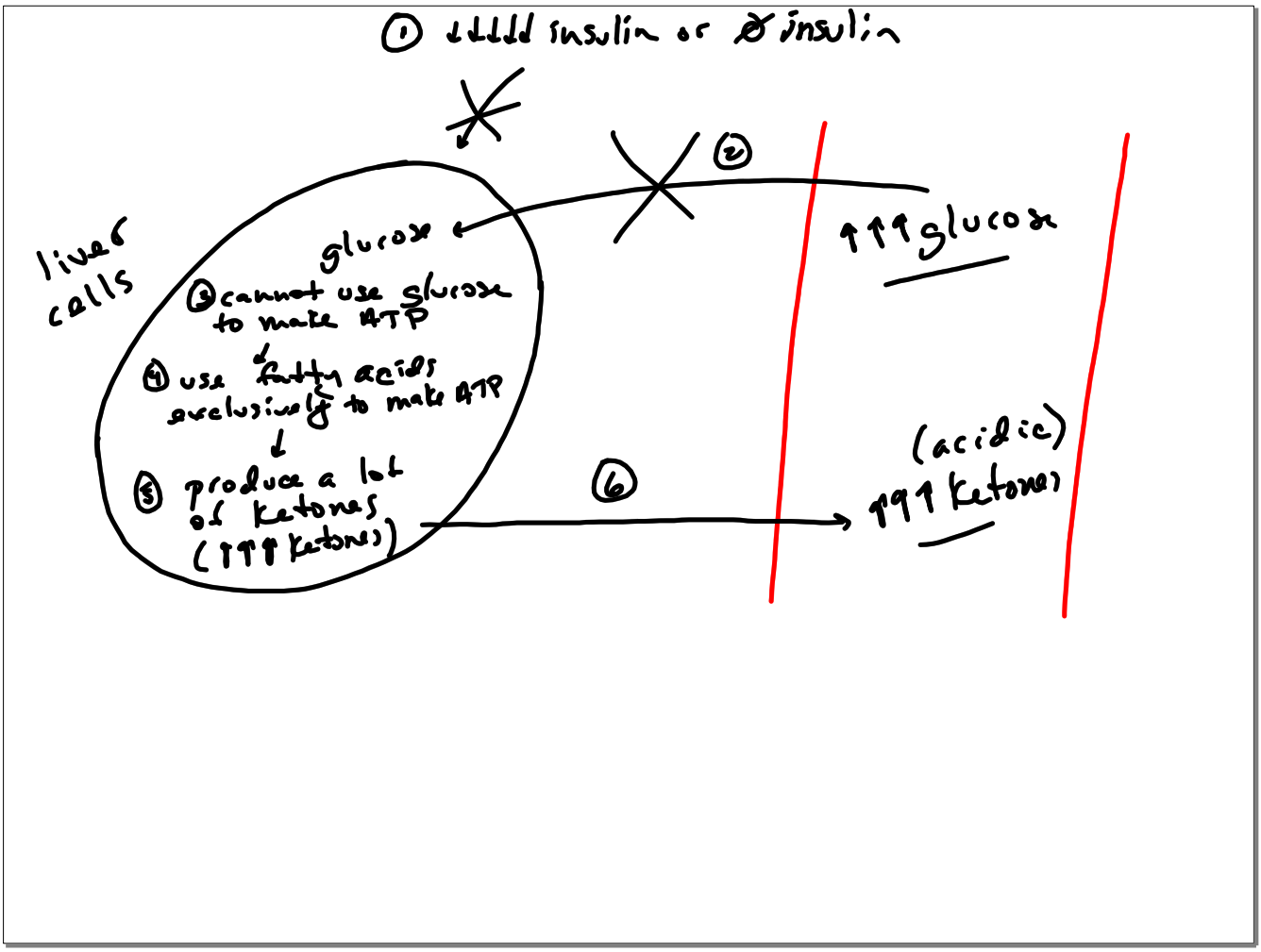
↓↓ damage/kill tissues (necrotic)

organ failure, blindness, amputation

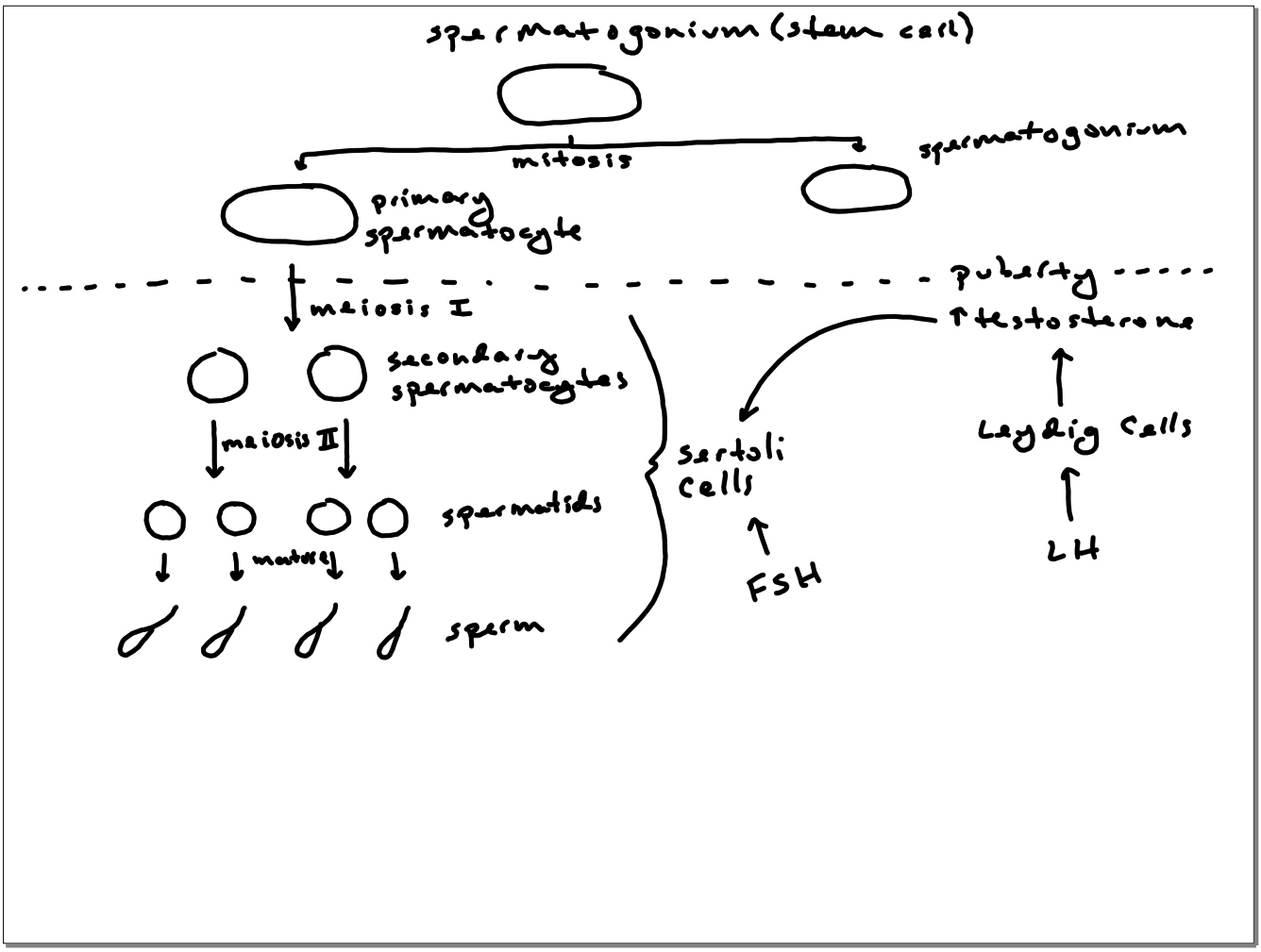
* Diabetic Ketoacidosis
  - 2 things occur:
    1. ↑↑↑ blood glucose
    2. ↑↑↑ blood ketones
  - how does it occur:
    - initiated by ↓↓↓↓ insulin or ∅ insulin

most often type 1 w/o insulin
or
↑↑↑↑ stress

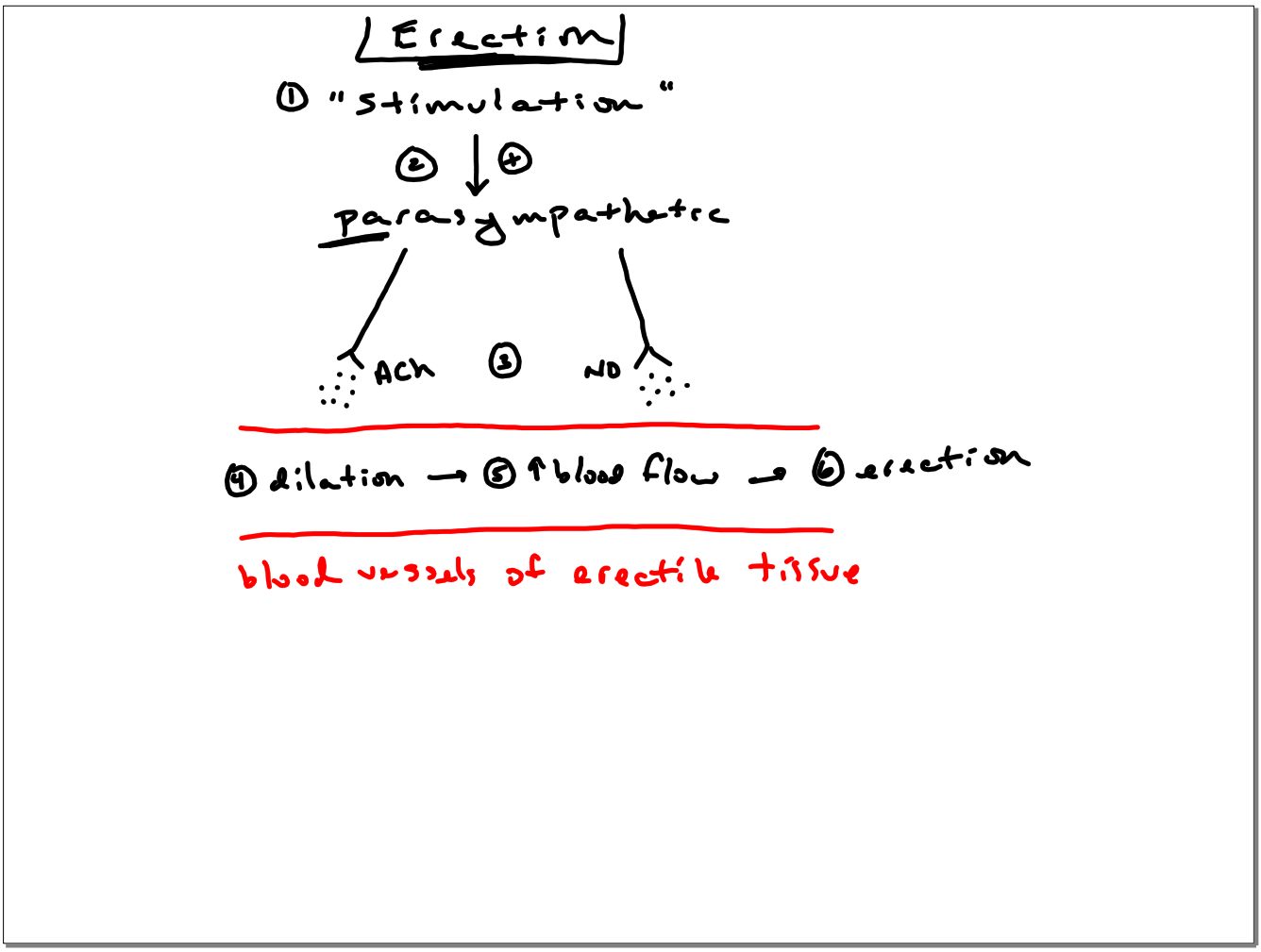
Jan 20-6:14 PM


① ↓↓↓↓ insulin or ⊘ insulin
②
liver cells
glucose
③ cannot use glucose to make ATP
④ use fatty acids exclusively to make ATP
⑤ produce a lot of ketones (↑↑↑ ketones)
⑥
↑↑↑ glucose
(acidic)
↑↑↑ ketones

Jan 20-6:15 PM


spermatogonium (stem cell)
mitosis
spermatogonium
primary spermatocyte
puberty
meiosis I
testosterone
secondary spermatocytes
Leydig cells
meiosis II
Sertoli cells
spermatids
maturation
FSH
LH
sperm

Jan 27-4:56 PM


Erection
① "stimulation"
② ↓ ③
parasympathetic
ACh
③
NO
④ dilation → ⑤ ↑blood flow → ⑥ erection
blood vessels of erectile tissue

Jan 27-5:07 PM

⑦ "stimulation"

↓ ⊕

⑧ sympathetic

↓

⑨ emission

↓

⑩ sensory nerves conduct AP's to ejaculatory reflex center of sacral spinal cord

↓

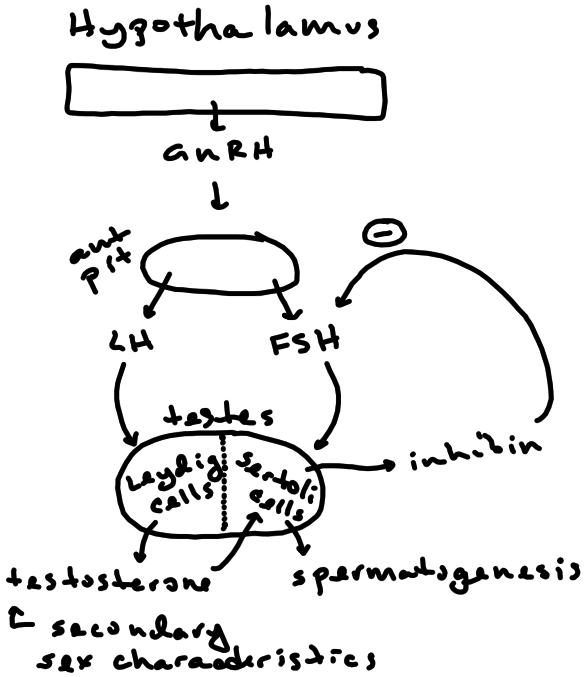
⑪ somatic motor nerves conduct AP's to skeletal muscle at base of penis

↓

⑫ skeletal muscles contract rhythmically

↓

⑬ ejaculation

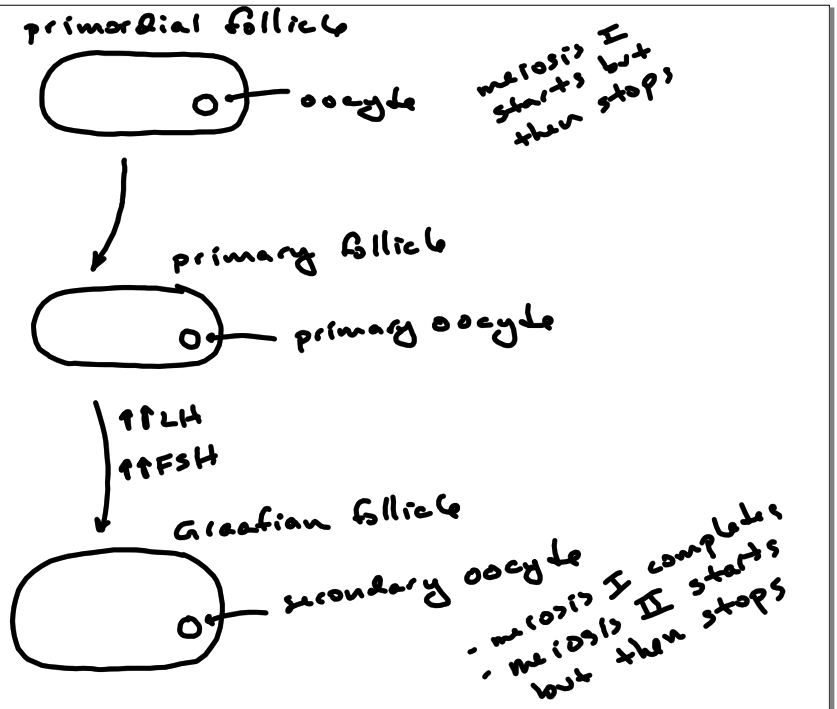

Hypothalamus
GnRH
ant pit
LH
FSH
testes
Leydig cells
Sertoli cells
inhibin
testosterone
spermatogenesis
secondary sex characteristics

Jan 27-5:18 PM

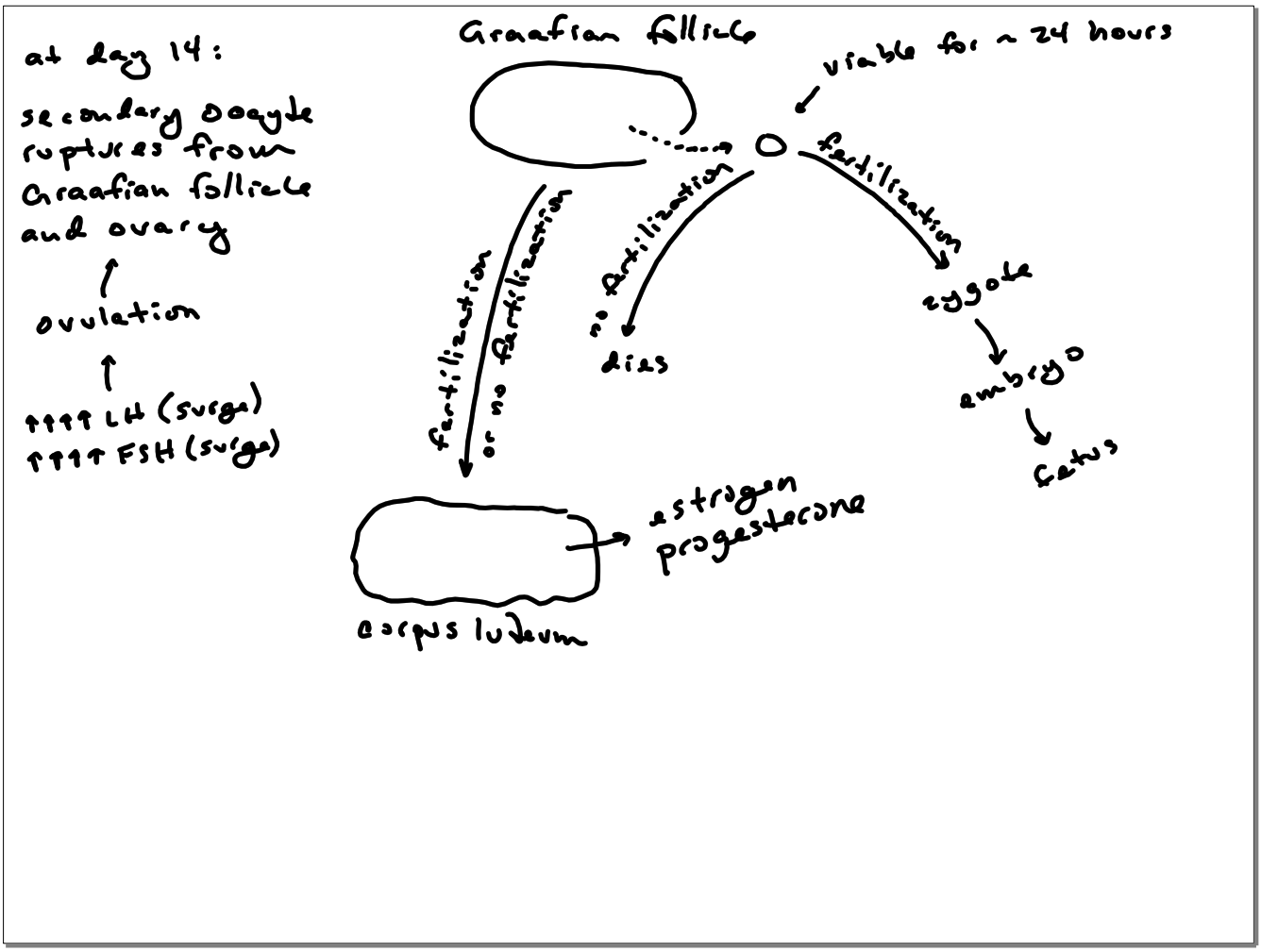
at birth:
~ 1 million oocytes per ovary

at puberty:
~ 400,000 oocytes per ovary

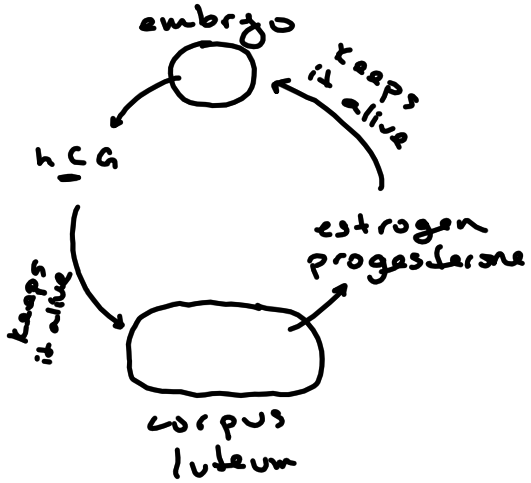
at beginning of each month
~ dozen follicles/oocytes mature but only one survives



Jan 27-5:24 PM


at day 14:
secondary oocyte ruptures from Graafian follicle and ovary
ovulation
↑↑↑↑ LH (surge)
↑↑↑↑ FSH (surge)
Graafian follicle
viable for ~ 24 hours
fertilization
no fertilization
fertilization or no fertilization
dies
zygote
embryo
fetus
estrogen
progesterone
corpus luteum

Jan 27-5:32 PM

fertilization	no fertilization
	no embryo ↓ no hCG ↓ corpus luteum dies

Jan 27-5:44 PM

Menses ← days 1-5

↓↓ estrogen ↓↓ progesterone
(corpus luteum disintegrated)

↓

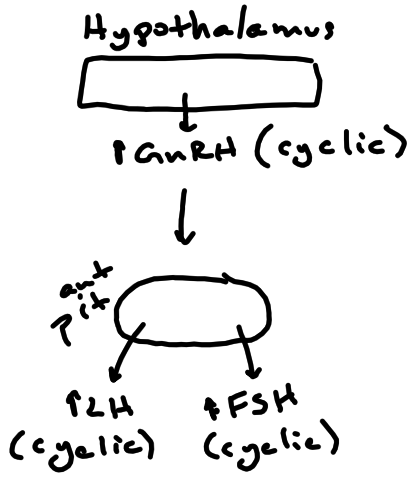
constriction of uterine wall blood vessels

↓

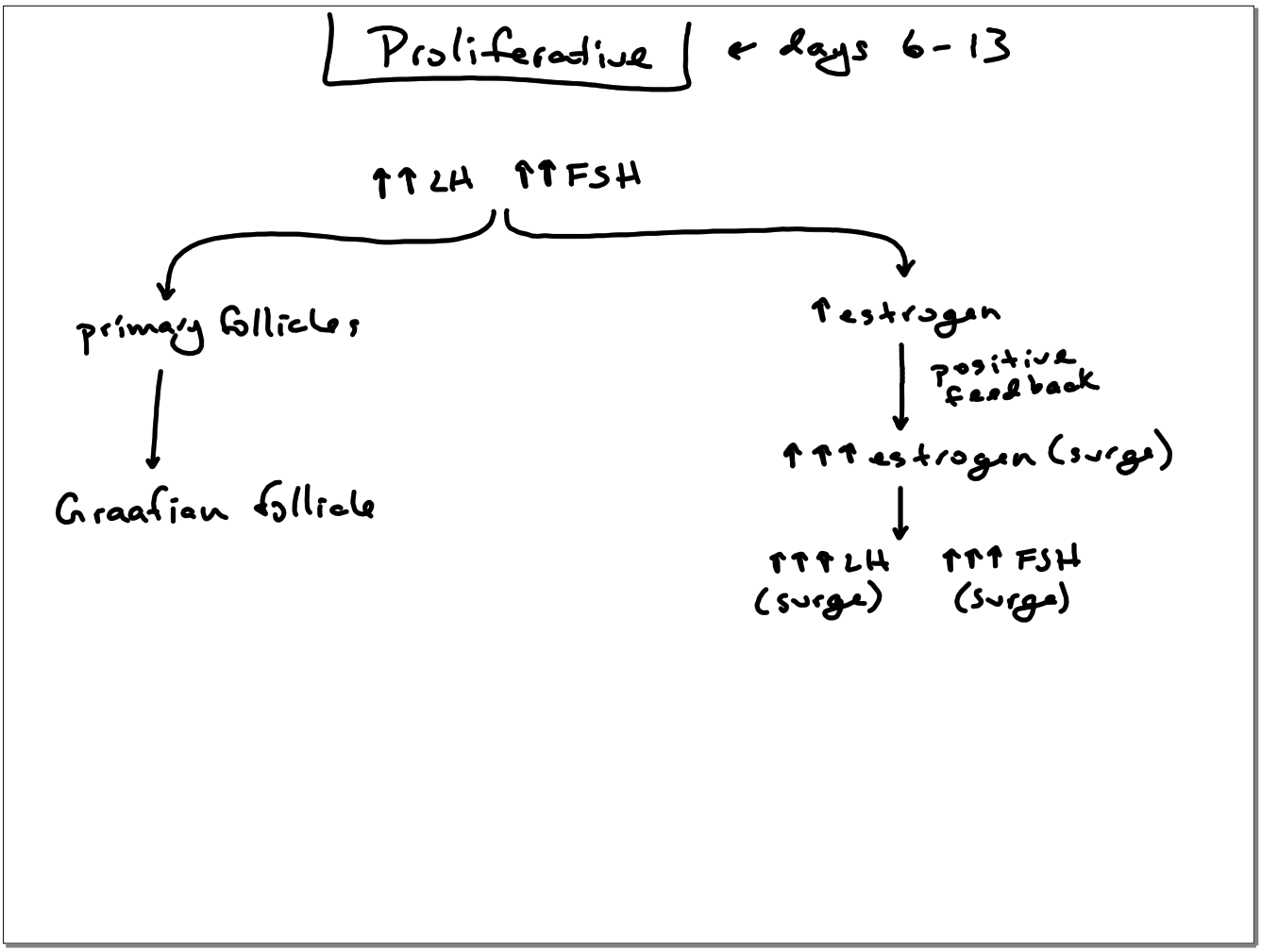
death of extra uterine wall tissue

↓

wall sloughs off and bleeding occurs



Jan 27-5:50 PM


Proliferative
← days 6-13
↑↑ LH ↑↑ FSH
primary follicles
Graafian follicle
↑estrogen
positive feedback
↑↑↑ estrogen (surge)
↑↑↑ LH (surge)
↑↑↑ FSH (surge)

Luteal ← days 14–28

Early	Middle	Late
↑↑↑↑ LH (surge) ↑↑↑↑ FSH (surge) ↓ ovulation	corpus luteum ↓ ↑↑ estrogen ↑↑ progesterone ① thicken uterine wall ② inhibit LH & FSH	corpus luteum dies ↓ ↓↓ estrogen ↓↓ progesterone

Jan 27-6:03 PM

* Menopause
  ↳ ↓↓↓ progesterone ? ↓↓↓ estrogen

causes dysfunction of hypothalamus

responsible in large part menopausal symptoms

can be treated with hormone therapy →

Jan 27-6:13 PM

Endo PM 1-11-21

Fri, 1/28 4:37PM 1:15:21

SUMMARY KEYWORDS

hormone, gland, prostaglandins, release, semester, regulating, draw, chapter, negative feedback, hormonal regulation, blood, fatty acids, fever, talk, hypothalamus, last semester, picture, cell, specific, notes

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00:02

Alright folks, here we go. Did you miss me? Okay, so what we're gonna do just for the first few few minutes today, just go over the syllabus very, very quickly. You know the deal, you know that we're very accessible, you know that we want you to come for help. This is how you get a hold of us. We have our office hours, of course, you can make appointments. And so please do now I'm not going to start my office hours until next week, it's just this week, it's just crazy hectic, and I'm just not going to be around too much. And so the exams now for some time, so it shouldn't be too big of a deal. I think like she receivables is around for her office hours if you need to see her. There's going to be SI sessions this semester. There is a new SI leader. His name is Shawn. He's awesome. I don't think he's here tonight. I don't see him. Soon he will have a room and time is going to be twice a week just like it was last semester an hour apiece for those particular sessions. There might be some online stuff going on too. I'm not sure he will let you know as soon as he knows. Of course, you're going to need the lab manual notes. If you want to book this is the one that we recommend any really a&p book will do as long as it's college level. If you want to book the needed know, if you want to save a few 100 bucks, don't have to buy one and foster some online sources. Although we know that sometimes online stuff is crap. If you want to throw something out, and he Dr. Rock saw this online, is this all right, I'll be more than happy to look at it. What else actually, really, let's just get right down to the nitty gritty. There's your life for the next 16 weeks. It's right there. What's our stand for? That's for Tucci so you notice there's a lot of our this semester, I have about 70% of the lectures. And it's just a systems that we're going to be covering, it's just a ton of physiology. Because there's so much more physiology, I'm going to have instead of four exams, five, my exams start right there, you're going to know exactly what's on my exams, because just like last year syllabus, I tell you, so that's what's gonna be on the first exam and oh and reproductive system, not until the beginning of February is that exam going to take place your first quiz is not going to take place until the very end of this particular month, that's going to be over the endocrine system and the reproductive system, just like last semester, six quizzes, two of which you can drop. What else midterm anatomy lab exam, just like it was last semester, we tell you exactly when it is your anatomy final will be a week before finals week. That way you can concentrate on just that anatomy material, and then you can concentrate on my physiology material for finals week, Tuesday in the evening is when that's going to be when it comes to your grades, it's exactly the same as it was last semester, it's based on the quizzes and exams that we give you. We don't give you busy work, we'd rather you study with that time, my exams are going to be 60 points apiece, except one of them's going to be 40 points. And so because there's a little bit extra when it comes to physiology this semester, there's going to be 60 more points this semester than there was last semester. And all of those 60 Extra points are going to be physiology points from my exams. That's it. Please go over that syllabus. If you have any questions

certainly ask me or lecture seabird about how things are going to be run, but it's going to be run exactly the same as it was last semester, it's going to be no difference whatsoever. Alright, that's all I have time for when it comes to the syllabus, because we have lots of stuff to cover this semester. So with that, let us start with the very first chapter. And that's the endocrine system, how's the endocrine system put together, that's how we're going to start. So as we always do, we are going to draw a picture and that picture is going to be that right there. So this is going to be an overview of the entire endocrine system. And we will refer to this picture a number of times as we go over the endocrine system. So let us call this picture endocrine signaling, and we're going to see what that means in just a second. So endocrine signaling. Now we're going to have up here on the screen and enter a gland. Now, hormones are made by glands certainly, but hormones don't necessarily have to be made by glands. And I'm actually going to give you an example, toward the end of the lecture today. But we're just going to call this a gland because most often that's what's going to produce a release a hormone. So this is going to be a gland and I'll just draw it down and I'll draw it like that. So that's a gland. Pick any gland you want doesn't matter. And that gland is going to make a hormone. I'm going to make that hormone a specific shape. I'm going to make it a triangle. So that's the hormone that that gland is producing.

05:00

And that hormone is going to regulate something, it's going to have a target. And that target is going to be far removed from where that gland is. So somehow, we need to get that hormone to what's called the target cell. And here at the bottom of the screen, I'm going to draw a single cell. And I'm going to call it a target cell. Now what could that target cell be, it could be whatever the heck you want it to be, that could be a muscle cell, that could be a liver cell, a kidney cell, it could be any cell in the body. That's what the target cell is. And it's the target of the hormone, the hormone is going to make its way to this particular cell, and it's going to tell that cell what to do, it's going to regulate that cell, how's it going to get there, it's going to get there, through these tubes. Blood vessels, let me give myself a little bit more room here. So the circulatory system is going to carry that hormone to where it needs to go. And so those are blood vessels, right? They're drawn very simply. And so let's get that hormone into the blood. So that hormone is going to be released into the interstitial compartment, right, and then into the blood. And so now we have the hormone. In the blood, we'll just call that step number one, the release of the hormone into the blood, and then that hormone is going to circulate in the blood, why we call it circulatory system. And off it goes. Again, far removed from where the gland is. And we'll just call that number two, it's circulating in the blood. And now our hormones over here. And then that hormone is going to leave the blood make its way to the target cell. And so here we go. Now this target cell is going to be affected by that hormone only if the hormone binds to something very specific on that target cell, what do we call it starts with an arc receptor. So let's put a receptor in this picture. Now we know from last semester that receptors can be located in the cell membrane, receptors can be located in the cytoplasm, receptors can be located in the nucleus, I'm just going to put it on the membrane, just to make it easy. And so there's our receptor. And I have to draw that receptor in a specific way to what mirror that particular hormone, that molecule in shape, we call that specificity, right? talked about that last semester. And so now our hormone is going to make its way to that cell and bind to that receptor, we'll just call that three. And then the receptor is going to be activated. And then what's going to happen is, as I called it last semester stuff, and we talked about some stuff last semester, we'll talk about more stuff this semester, especially in the second chapter, when we talk about specific hormones, and what they do when they bind to the receptor and tell the cells what to do and regulate the cells. That's endocrine signaling. That's a way that the cells of this gland are communicating with their target cells. Did we talk about communication last semester, yes or no? Yes. How do we have two cells communicate with each other starts with an S. synapses. Remember that this cell communicated with this cell through

synapses. Well, this is another way that we have communication within the body is to the circulatory system. So again, this is endocrine signaling. And we'll come back to this picture a number of times. Now, when it comes to hormones. There's a bunch of different kinds. And they're categorized into these three classes here. Some hormones, proteins, some hormones are amines, and some proteins are fat, are called steroid hormones. Now, I don't need you to specifically know that antidiuretic hormone growth hormone insulin, oxytocin, and a number of others are protein hormones, I'm not going to get into that kind of detail. I want you to know the first two lines, though, I want you to know that protein hormones are water soluble, we know what that means. And I want you to know that most are going to circulate freely within the blood, I want you to know that which is different than steroid hormones, which are fats, which are not water soluble, they are fat soluble, they need to be carried in the blood because well, about half of your blood is water. So there has soluble in water. And so they need to be carried by something but they need to be carried by well, we'll talk about it later on in the semester when we actually get to the blood. And then amines are also water soluble, like protein hormones most circulate freely within the blood. When I say most, that's not all. So there are exceptions to this. Really don't need to know what the exceptions are. So again, you do not need to know specific hormones and what kind of hormone they are. I just need you to know those first two lines. That's what you need to know when it comes to these. Now we're going to be talking about specific hormones in lots of detail in the next chapter, every single one of these hormones plus more

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before we move on with the end The current system though, I want to talk about some molecules that act like hormones that are almost hormones, but cannot be considered hormones, although many books will tell you their hormones, and many websites will tell you their hormones. They're not and then we'll tell you why. They're called prostaglandins. We talked about prostaglandins. Last semester, we spent about two minutes on them, when we were talking about the different type of molecules in the body. And when we were talking about fats, we talked about prostaglandins, if you recall, but not a whole bunch. And the reason is, because I knew what he talked about them today, this particular semester. So what are they? Well, they're fatty acid derivatives. That's something like I said last semester, all that means is, is that they are derived from fatty acids, it means that you have a fatty acid and it gets turned into a prostaglandin. And we know what fatty acids All right. Saturday, saturated fatty acids, unsaturated fatty acids, remember that polyunsaturated monounsaturated, we talked about those in a lot of detail, actually, last semester, the fatty acids, so we're going to take a fatty acid, and we're going to turn it into a prostaglandin. And there's three kinds of prostaglandins series one, series two, and series three. And each of those is produced by a specific fatty acid. What fatty acids gamma little linic acid is a fatty acid. arachidonic acid is a fatty acid that goes to pet the milk acid is a fatty acid. Those fatty acids are going to turn into prostaglandins. And so to make it perfectly clear, what we're going to do is we're going to just draw that now. So let's take the fatty acids, put them in a column here. And so this is production of process prostaglandins. Most tissue in the body produces prostaglandins, as I stated last semester. So here are fatty acids. And I'm just going to abbreviate the fatty acids. I don't want to write out the entire name. So gamma linolenic acid, G, l, a arachidonic acid, a ESS ecos, methanolic acid, E P, A, each of those fatty acids, it's a fact. And they are going to be turned into through a chemical reaction, a prostaglandin specific prostaglandins. And so these arrows here are chemical reactions. Now in order to have a chemical reaction, it needs to be catalyzed, and what catalyzes chemical reactions, as we will know, are enzymes. And there are two enzymes that are going to catalyze each of these reactions and they're called cyclo oxygen asis cycle. oxygenase one cyclooxygenase. Two, I'm going to abbreviate them because most often they are when they are named, abbreviated Cox one and Cox two. So coxswain, cyclooxygenase, one cyclooxygenase, two cycle oxygenase, one and two for each of these. And what are we going to get at the end of this, we're going to get prostaglandins. And so I'll put over here in

this column, prostaglandins. And I'm going to abbreviate those PG. So PG one, PG to PG three, I told you there's three types of prostaglandins. And then your notes, you'll notice that I have some letters that are involved in it as well. So PG h one, e two, a three and so there's subcategorize, with letters as well. The more we study, the more we find. And so then we just have to categorize them in different ways. It was thought that at one time there was only one prostaglandin made males by the prostate gland. That's where the name actually comes from. But then the more we studied them all, we found pretty much every tissue makes prostaglandins. So this right here is this picture right here. So what you need to know is is that the series one prostaglandins are made specifically by deriving it from GLA. arachidonic acid does not make series ones that only makes series two. So when the way I have these chemical reactions drawn out is exactly the way that is going to happen. So you don't have any GLA making PG threes to CPA.

14:24

What are these prostaglandins? Do they regulate things just like hormones do? They're going to tell target cells what to do. Now, what do they tell cells to do? In your notes? I have them divided into the series one in threes and the series twos, the ones in threes do these kinds of things. reduce inflammation, reduce pain, make your airways dilate, and then the series to pretty much do the complete opposite. They cause inflammation. constriction of the airways, they cause pain, they cause fever. You cannot have a fever, unless the series to prostaglandin causes you to have a fever. So you have what's called a pyrogen. In your body, a pirate gene is something that causes a fever, like COVID, some virus, some bacteria, for example, some pathogen in the body starts up whole cascade reactions. And within those reactions, prostaglandin twos are involved. And that causes your body temperature to go up. When you have pain part of the process that's going on with pain. It sees prostaglandins when you have inflammation in the body, not always. But the series two prostaglandins are going to be involved in the cascade that causes inflammation. Now, if you look at these things, once in threes, they reduce inflammation, they reduce pain, that sounds fantastic. prostaglandin TOS they cause you to have fever, they cause inflammation, they cause pity, cause those kinds of things. So what books and websites will say is that the ones and threes are the good ones. And the twos are the bad ones. You're not going to see that in my notes. Did we talk about pain last semester? Did I tell you that pain is good? I did Weis pain good. tells you that something's wrong. Yes, telling you something wrong. If you don't feel pain, you're in trouble. Because you don't know something is wrong. You need to get away from it. Or you got to do something about it. So even though pain sounds like a bad thing, pain is a good thing. inflammation can be part of the healing process. Not always. But it can be fever, why do we get a fever, if you have COVID, or whatever the flu, what does the fever do for you makes you feel miserable. But it's helping the immune system fight the pathogen. So fever is good now, too high a fever is bad. Because then we start to denature proteins, right? And then we get in some trouble with a fever of 100 123. And that's no big deal. That's not going to cause any problems that's actually going to help now you might feel miserable with it. But if you can take the misery leave the fever alone, it's part of the healing process to get rid of the pathogen that's within the body itself. So I will not say that once in threes, or goods and twos are bad. I won't say that. Because that's just not the case. Now, can you have too much inflammation in the body? Absolutely, you can. And there are cases where that occurs. And then that leads to some bad things. And we'll talk about some bad things that inflammation leads to in the next chapter. So what I want you to know, obviously, how each of these ones, twos and threes come about which specific fatty acids are involved with that. And then would each of them do the ones in threes versus the twos but do know that again, the twos are bad. All right. Now something else I want to talk about before I move forward. And that's this. Anybody here have a COX inhibitor type of drug that's given. So I'm going to put here COX inhibitor and I'm going to give you examples of COX inhibitors and why

we take them and you're going to actually be able to answer that question for me when I tell you what the COX inhibitors are. A COX inhibitor is going to inhibit the cyclo oxygen asis. So they block production a PGS by blocking Cox one and Cox two. And so, for example,

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ibuprofen an example of ibuprofen would be Motrin. Advil, that's ibuprofen, naproxen. That would be Aleve. Aspirin. All of those are COX inhibitors. All of those are what are called NSAIDs, non steroidal anti inflammatory drugs, anti inflammatory. What are they doing? Well, what they're going to do is that they do block the production of all PGS. But when you have a fever, for example, and you want to make the fever go down, you might take some motoring. If you have some pain, you might take some elite, you have some inflammation, maybe you twisted your ankle and your ankles swollen, and you want the inflammation to go down. Maybe you take once again so moto interleaver, whatever. And what they're doing is is that they're diminishing the inflammation. They're diminishing the pain they're diminishing the fever specifically by decreasing P G to now they're still diminishing PG one and PG three. But what's causing the pain is what was causing the inflammation as well. What's causing the fever as what is To increase in PG two, so you diminish the PG two and then those kinds of things get diminished. It doesn't mean that if you make one in three go down, they're already low to begin with that you're all of a sudden going to start to feel pain, you're going to have inflammation. That's not the way that it works. So because the higher PG two is causing those signs and symptoms, diminishing it is what's going to cause those signs and symptoms to go down. Mainly symptoms really. So what are they going to do? They're going to just do it with arrows. They're going to decrease pain. They're going to decrease so you know what, I'm going to take it from the PG to they'll decrease pain. They'll decrease inflammation. They'll decrease fever by decreasing PG two, it's not in the notes, but I want you to know it. And then we have some stronger COX inhibitors like Celebrex, for example is something that you have to have a prescription for typically people who have arthritis will take Celebrex. I'm not going to worry about that too much. These are the ones that I'd like you to know the ones that are a bit more common that you can go to my right now and buy if you want. By the way, Tylenol is not a COX inhibitor, I've seen websites that say Tylenol is it's not. Alright. Now, I told you from the very beginning, the prostaglandins are not hormones, although some websites and some books will tell you that they are why aren't they? This is the reason they're not released into the blood. Otherwise, they look just like hormones. They act just like hormones that regulate things like hormones, they have target cells like hormones, but they don't do this. The cells that make them do not dump them into the blog. You cannot have a molecule ba hormone unless it's dumped into the blood unless we have endocrine signaling and endocrine signaling is all about the circulatory system carrying the molecule where it's supposed to go. That's what endocrine signaling is. prostaglandins don't do that. And the reason they don't do that is because their Half Life is so short. And so for those of you that don't don't not know what half life is, it's the time it takes half of the molecule to decay, and then another half of the molecule to the cane and another half of the molecule to decay. So I'll give you an example. Let's say the half life of a molecule is one second. Typically, you multiply that by five, and the molecules completely disintegrated and worthless. So if a molecule has a half life of one second, that means within five seconds, it's worthless. If it gets to the target, it's not going to do a damn thing. And so the half life of prostaglandins is about one second. So if you don't the prostaglandin into the blood, by the time it got to the target cells take longer than five seconds, it's not going to work.

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So the longer the half life of a molecule, the longer it will last, the longer it's effective. And so because

prostaglandins has such a short half life, they need to get to their target cells quickly. And so how do we do that? Well, I'm going to show you there's two other signaling mechanisms. One is called Paracrine signaling. And the other one's called autocrine signaling. And that's it your notes right here. So now let's draw that. Alright, let's get these two other signaling mechanisms here. Now, prostaglandins aren't the only molecules in the body that exhibit these two types of signaling mechanisms. There are others and I'll tell you, a few others later on in the semester. So this one will be here on the left will be Paracrine signaling. And then the one to the right is going to be autocrine signaling. And it's very, very fast, or they are very, very fast, I should say. And so we're going to have some cell. And that cell is going to produce a prostaglandin. So some cell and let's make the prostaglandin. We can make it blue. And let's make it a different shape, though. So there's a prostaglandin right there, that's not a nucleus that is a prostaglandin, series 123 Doesn't matter. We're going to have another cell over here.

24:17

We're going to call that the target cell. What can the target cell be whatever the heck you want it to be.

24:25

And so what's going to happen is, is that the prostaglandin is going to be released into the interstitial fluid and make its way to the target. So how long do you think that's gonna take? It's not very, it doesn't have a very long distance to travel at all. It will be less than a second. So by the time it gets there, it'll still be very, very effective. That is the prostaglandin. So that's Paracrine signaling. If you look at the definition in your notes, what does it say? release of a substance from a cell that acts on a nearby cell and adjacent cell, those two cells are very near each other. There About a cell apart autocrine signaling, it's even faster. So there is some cell that's going to produce a prostaglandin. And that prostaglandin isn't going anywhere. The cell that produces the prostaglandin is also the target cell. So it's both can't get any faster than that doesn't matter if the Half Life is short, it will still be very effective. And so that's autocrine. Signaling. Are we good with this? Yes.

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It doesn't look like the prostate gland is the, the actual estate there, right?

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So you'd write yourself in danger as it is. Yep. That move. I mean, it moves, it goes someplace else from where it was produced, but it goes to its target within the cell, and then tells the cell what to do. Within, it's in the cells not going anywhere. You're welcome. Now what? Now let's go back to the endocrine system. Now let's talk about some things that we've actually already visited last semester, we'll visit them again, but then in more detail as we get into the next chapter. And actually, this chapter is, well, we'll visit it as well, in a little bit more detail. So we have these feedback mechanisms, negative feedback, positive feedback, we're very, very familiar with them. Negative feedback is all about physiology, right? maintaining homeostasis. And so when we talk about a hormone, that's going to be our setpoint for the hormone, if you remember, so hormone level, in the

blood, so hormones are released into the blood, right, we can measure the level of hormone in the body by taking a patient's blood, sending it to a lab and say, Hey, lab give us a level of this hormone. So we can measure the level in the blood. And so we have what our normal range around that setpoint. Remember that. So that's the normal level of a hormone within the blood. And if you remember, there's an X and a Y axis here, the x axis is time, the y axis is the level of something in this particular case, it's the hormone. And when it comes to negative feedback, when we get a little high, what are we going to do? Well, we're gonna bring it back down within that normal range. That's negative feedback. And given that these particular hormones are regulating things, and many of the hormones that we're gonna be discussing, are maintaining homeostasis, it's very important that they remain within their normal range, because if they do, well, then they regulate things properly, and homeostasis is maintained. So that's negative feedback, if they start to get a little high, that's negative feedback if it starts to get a little low. So we are keeping our hormone level within that normal range. Now, it doesn't mean that if a hormone is outside the normal range for a short period of time, that that's a bad thing. And we'll talk about that. But we will also talk about when the level of a hormone is outside of his normal range for extended periods of time. That's no good. And so this is very important. So keeping the hormone within the normal range allows the hormone whatever hormone it is, to regulate properly, regulate what whatever it is that the hormone is regulating blood pressure, blood osmolality, whatever things we'll be talking about at a later time. And so that helps maintain homeostasis, which we're going to find is a very important job of the endocrine system. It's not its only job, but it plays a pretty darn big role in talking about homeostasis, the tongue last semester, showed how the autonomic nervous system is involved in it. So to is the endocrine system, this is physiology. That's physiology. That's normal. Now we're going to talk about abnormal. Oh, no, we're not not yet. Because now we're going to talk about positive feedback. Let's visit that as well, these two lines here. Talk about it at the end of the lecture toward the end of the lecture. So I'm going to come back to those two lines. And I'll actually come back to this slide as I do, just to remind you, before we move forward, let's look at positive feedback. Not homeostatic, as we will know doesn't mean that it's bad. It's unnecessary thing. And so we'll draw our homeostatic line again and this is again, hormone level in the blood whatever hormone it is. And there are a number of hormones that act the way that I'm about to show you, and that is within a positive feedback manner. So initially we'll have our normal range, but then all of a sudden, we're going to have a special event. And I'll name a couple of special events that causes the hormone level to go up, up, up, up, up up above it, it has to, if it doesn't, the special event doesn't occur. And so right here is the start of the special event.

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And it's going to go on for however long it's going to go on for. And so this length of time is how long that special event is going to occur. And it's going to be made possible because whatever hormone it is, is going up, up, up, up, up, up, up, up, up. Now it might look like Well, that's not a good thing. It's way above our normal range. It's not, it's necessary, the level doesn't go up, the special event doesn't occur, well, I have a couple of special events that we're actually going to talk about them, at least one of them in some detail. Childbirth, couple of specific hormones, lactation, if mom wants to breastfeed the baby, after the baby is born, we need positive feedback to occur with some hormones that we'll discuss in this chapter, and next, actually. And then once the special event is over, and it's over right there, well, then we go right back to negative feedback in the level of the hormone goes back to what it was prior to the special event. So that's positive feedback. And I'll mention about three hormones that act this way. And when they act this way, specifically,

31:40

now we can move forward. And that is what happens when we lose negative feedback.

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Obviously, yes. Does it drop back down? back quickly? Or was it I mean, it just depends, you know, it could take days weeks, it just depends on what mechanism we're talking about. Okay. But the bottom line is, what I want you to know is, is that it will drop back down to normal levels. If everything goes right. Now, let's talk about when things go wrong. What happens when we lose negative feedback? Well, then we have what's called hypo secretion in hypersecretion hypo secretion, too little hormones being released over an extended period of time hypersecretion too much hormones being released over an extended period of time. So let's draw our homeostatic line again and see what it looks like. So again, this is hormone level in the blood. And hypo secretion is that it is low over an extended period of time now how long it just depends on the condition. And so we have hypo secretion. Why are we hypo secreting the hormone because we've lost negative feedback, what should happen under normal conditions, it should come right back up. That's what should happen. But that's not what's happening because something went wrong, what kinds of things have gone wrong? I'll tell you a few examples in this lecture, and then a whole bunch when we get to the next chapter when we talk about a lot of pathophysiology. And by the way, this is pathophysiology. This is when things have gone wrong. And we can certainly go the other direction when we are chronically high, so we're chronically hyper secreting. Both are bad. And we'll talk about specific hormones. And what kinds of things will happen with Hypo secretion and hypersecretion of specific hormones. And we'll talk about specific syndromes and diseases. We're going to do a lot of pathophysiology in the next chapter. Because it's just a good way to learn what's happening with these particular hormones. So we lose negative feedback. That's why we're hypo secreting or hyper secreting. So we've lost homeostasis. And it leads to bad things. How bad? Well, it depends on the hormone. It depends on how long depends on how high and how low. But certainly, it can lead to death, unfortunately. And we'll talk about these things in the next chapter for sure. And so this is pathophysiology.

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That's the more fun stuff to talk about. And when we get to chapter two, we'll talk about some fun stuff. In my opinion, it's fun. So back to that many, many, many, many, many times. That's the other thing too is that I'm telling you how things work with the endocrine system. When we get to the next chapter. I'm going to keep on coming back to this and all the other things that we're going to be talking about discussing in this particular chapter. So it's going to get reinforced, reinforced, reinforced, reinforced. What's next neck. Next thing we need to talk about is patterns of regulation. So let's go back to our first picture. So we have a gland here, and it produces a hormone and it releases a hormone. does that how does that gland know how much to produce? How does that gland know how much to release? What's controlling the gland, what's regulating the gland, I'm showing you that this gland is regulating this target cell, this hormone is regulating that target cell. But the gland is regulated as well has to be just not random. It's got to produce just the right amount, it's got to release just the right amount. So what regulates glands? That's what we're going to talk about now. And so patterns of regulation, non hormonal, neuro, and hormone. And so what does that mean? Well, it's going to draw three different columns here, I'll give you a break soon against the same thing from last semester, after about 3738 minutes, and we'll give you a break. So we'll just put the beginning of this picture on the screen. And then I'll give you a break. So this is patterns of regulation, regulation of what regulation of the glands again, it's not always a gland No, most often is the gland of hormone production and release. So we're gonna have three different columns here, the first

column, I'm going to call a non hormonal. Second column, I'm going to call hormonal. And then the last one is going to be neuro. You know, put a hard line here, and a hard line here. By the way, when it comes to non hormonal, I'm just going to tell you right off the bat, all hormones are controlled or regulated by things that aren't hormones. That's what non hormonal means. And I'll even give you examples of those. All hormones are controlled, regulated by some other hormone. But only some hormones are controlled by your nervous system. Just some, we actually already know one of them, because we talked about it last semester, and we talked about the autonomic nervous system. And then I'll just put a hard line there. When we come back, we'll continue the picture in the store.

37:38

Okay, folks, let us continue our story. So again, we're talking about what is regulating these glands. And we have a number of things that are going to do that. So non hormones. So something that's not a hormone. And so I'll just have a few here, I know I have examples in the book. And I'll go back and look to make sure that I have those included in here. But like blood pressure, blood pressure is not a hormone that is a non hormone blood pressure can regulate a gland. blood glucose levels, the amount of glucose in your blood is detected by a gland and then that therefore dictates how much hormone that gland is going to produce a release. If the gland is supposed to respond to glucose, that is, not all glands are going to respond to blood pressure. Now glands are going to respond to glucose, just a short few which ones I'm going to tell you in the next chapter. When we talk about specific hormones and specific glands. Blood glucose, blood pressure, I know I'm talking blood, this blood that it doesn't necessarily have to be blood. By the way, these are just the ones that kind of came to mind for me. Let me look at announced to make sure that I didn't miss one that I had in the notes that I really want to put up on the screen I have blood glucose stimulates the release of a hormone cortisol. So I gave one example in the notes. But it could be a bunch a bunch a bunch of different things, things that are once again, hormones. And so what that's going to do is it's going to affect this gland, I'm going to call this gland x, which re releases hormone x. And so these things right here will affect that gland, how much hormone it produces, how much hormone it's going to release. Now, here's the other thing to keep in mind. If you have a gland that is regulated by something, it means that that gland is regulating that something meaning life. If blood glucose is affecting this gland, it means that that gland is going to affect glucose level. So I'll give you an example. I don't have cortisol and also give me another one insulin, you know, assume you guys know what insulin is or at least heard of insulin. So you eat a meal, and it's got carbs in it, which means it has glucose in it. And that glucose is going to make its way into the blood your glucose levels go up. So the glucose levels go up. The gland detects it and releases insulin. And then the insulin is released into the blood goes to its target cells. And then the glucose is taken out of the blood. So the glucose is regulating the gland, but the hormone is regulating the glucose. That's why these things affect the specific claim. Alright, we'll talk about this in detail later. But I just want to make sure that you understand that if something affects the gland, it means the gland is affecting it. It's this beautiful pathway between the two, there's a beautiful friendship between those things that regulate the gland, and the hormone that the gland releases. All right, hormone. We're gonna have two glands. Now, we're gonna have gland x again, I'm always gonna have gland X, releasing hormone x and these particular examples. But now we're going to have some other hormone, I'm going to call the hormone y. So we're going to have gland y, whatever gland that is releasing hormone ly affecting hormone x. So that's hormonal regulation. And once again, when it comes to every single gland in the body, there's some non hormone that affects it. Every hormone in the body is affected by some other hormone. That's why I have all hormones. I can't think of any exceptions. And I purposely put gland X and gland x, because I don't want you to think that there are those hormones that are affected non hormonal II, and that are those hormones that are regulated, just hormonally. If I have all hormones, all hormones, that means the same hormones are affected by hormone, other hormones and non hormones. That's why I wanted to make

sure that you see that I have gland x, y, and x is the same stinking gland, whatever their gland is. And then last but not least, there are a certain number of hormones that are affected by your nervous system. So I'm going to draw a picture that we saw last semester quite a bit. I told you, we'd see it again, this semester, there's our brainstem and our spinal cord. So brainstem, cervical, thoracic, lumbar, sacral. Remember all that? And then we have and this is going to be the autonomic nervous system, not the somatic nervous system, by the way. And so I don't know, I'll just take it right out of here. Thredbo, lumbar region, short preganglionic long postganglionic going to gland X, whatever gland x is not going to name it.

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releasing hormone X, although last semester, I did give you a specific example, Did I not? Part of the thoracic region, one long preganglionic neuron? What is it going to what gland? Beautiful the adrenal gland, which is shaped like a triangle, we'll see that this semester. I have to put glands because we have two of them. So there are certain hormones from the adrenal gland, the adrenal medulla that releases epinephrine, norepinephrine, we'll talk about it in the next chapter that's controlled narrowly. So there are a handful of hormones that are regulated by your nervous system. So that's what's telling you when, when the producer how much to produce, how much to release. Now moving forward, now we have to talk about patterns of hormone release. So we have these things that are regulating these grains. And these glands are going to produce a certain amount of the hormone release a certain amount of the hormone. And when they release that hormone, well, where's it going? Well, of course, going into the blood, that's just where hormones go. And when these particular glands are releasing the hormones, they're going to release them in certain ways. They're going to release them chronically, acutely cyclically. And so what does that mean? I'm about to show you. So in the next section of the notes, we're going to be talking about this. And so we're going to actually draw these pictures again. And as I do, I'm going to talk. So the first one we're going to do is chronic release. And again, we have an x axis and a Y axis, I just don't draw the X and the Y axis. But you know what? I'm going to do it in this particular case. When don't I draw the X and Y axes? Well, when I do these, you know, these set points. So there should be an X and a Y axis here, again, the level of something versus time. I'll do it here in this picture. No, you know what, I'm not going to, you know what, I changed my mind, I just want to be consistent. Nevermind. I'm just going to draw a setpoint line. So here's that setpoint line. I'm not even draw a setpoint line. I'm just going to do this. That's the level of the hormone over time. It's not changing very much. That would be chronic release. So if you have a gland, and it just releases about the same amount of hormone over time, every single second every single minute, it's releasing the same amount. level of that hormone isn't really going to change very much in the blood. And we call that chronic release. And so it's called so patterns of hormone. So that's the word that I use. So I'm going to do it up here. Patterns of hormone release. That's a pattern. Over time, it's not changing too much. Every hormone is going to be released chronically. So I'm going to put all hormones again over here. All hormones are produced by gland and released into the blood, nearly constantly, over time. And I give an example in the notes, but we're going to be going over these examples over and over again in the next chapter. So it's not that important right now. Then we have another pattern of release, to where you might have, it looked like that. But then all of a sudden, boom, a big old spike of the hormone in the blood. But then it comes back down. In order for that to occur, you need to have some stimulus right here. That's called acute release. Acute meaning in a very short period of time, all hormones can be released in this manner as well. And if you look in the notes I have in quotes, it's unplanned. Now I'm going to explain what that means. Because with chronic release, or not chronic but cyclic release, I have, it's a planned event. And so what do I mean by that? Well, I'm going to go back to our insulin story, and eating a carbohydrate meal or any meal for that matter. But we'll just say you had a big old plate of pasta, and you had some Twizzlers, a lot of carbs, like glucose. And so the glucose level went up in

your blood. And so the glucose level goes up in your blood, it's going to stimulate a gland to release a particular hormone, that would be insulin. And that insulin is going to spike at that point, because your glucose levels have spiked. And that needs to happen, because we need to get those glucose levels back down.

47:16

Because when glucose levels are high in the blood over extended periods of time, bad things happen. I'm going to talk about what the bad things are. When we get to the next chapter, and we talked about diabetes. Not yet though. So that's an example. It's not plan. Now, you might plan to have pasta every day at noon, that might be your plan. But the body doesn't know that it's not something programmed in your body. And that's another word that we can use the word programmed, there are certain programs in your body that are supposed to happen at certain times of either the day or the week, or whatever, that's going to be cyclic. But this is unplanned to the body, the body doesn't know another example, let's say we're teaching all of a sudden, I don't know, some rabid raccoon runs into this room, I can guarantee you at that particular point in time that you are going to activate your sympathetic nervous system. And it's going to cause a whole bunch of epinephrine and norepinephrine to be dumped from your adrenal gland. That would be an unplanned event. That's not something programmed in your body to occur. And so you would get a spike in epinephrine and norepinephrine, otherwise known as adrenaline, and noradrenaline, we don't call them that. We call them norepinephrine and epinephrine. And so you get a spike of it. And then when the raccoon decides to go away, we all kind of calm down, the parasympathetic nervous system takes over again. And the level of those hormones goes down, just like the level of insulin would go down after the blood glucose levels go down in the blood. Yeah,

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so isn't it, like when you mentioned that the examples are like after? Lunch? You know, right. And so like, let's say, after four hours, you know, like, how are you happy? Like, I don't know, maybe after four hours, like, like, by the time it gets here for like, four hours later, you know, when you have your next meal? Like, that's one glucose level goes back down.

49:08

Yeah, that's, that's a pattern you're gonna see. And that that pattern is going to be dictated by insulin. Absolutely.

49:15

Like, like an example. That explains like acute relief.

49:21

That is actually that's actually what it is. Yeah, that's a cute relates both of the examples that I just gave. It's acute release the rise in glucose in the blood, and the response of insulin, the raccoon running in here, and the response of the adrenal gland, dumping all that particular hormone, that's

running in here, and the response of the adrenal gland, dumping all that particular hormone, that's what the spike is. So that's what that is a big huge rise in the level of the hormone and then goes back. But then it could spike again, defense, but we have to have some stimulus. And the stimulus that's going to cause the release of particular hormones is going to be dictated by well what the hormones job is. Now we're going to see something that looks very very similar. This actually is going to look just like that. But the events are going to be different. So we're going to have some stimulus and it's going to cause a spike. And then it's going to come right back down. Again, that's the level of the hormone, let me make sure that you understand this is that this is the level of the hormone in the blood. We have a stimulus. But this stimulus is planned. And when I say plan, I'm talking about the body, we can also use the word programmed.

50:36

So what would be an example of this, I'll give you a couple. during deep sleep, there's a big huge rising growth hormone that is programmed, that is planned. That's what's supposed to happen if you sleep long enough and deep enough. So that would be an example. That is in the notes, I'm pretty sure I have that as an example of notes. And we'll talk about these again, when we get to the next chapter. Increase release a growth hormone to induce sleep. That's an example. Another example of an that happens daily. Another example would be the menstrual cycle, which we'll be talking about when we get to the reproductive system, certain hormones go up at certain times of the month luteinizing hormone follicle stimulating hormone estrogen, progesterone, it's timed perfectly. And when they're timed perfectly to rise into fall at certain times of the month, then your menstrual cycle proceeds the way it's supposed to proceed. And if you screw that up, somehow, oh, not if you screw it up. But if it gets screwed up, somehow explain how, then the menstrual cycle does not proceed properly, you might miss menstrual cycles, for example, we're going to talk about it. But those are a couple of examples. Again, cyclic and acute look exactly the same, but the stimulus is different. And so this is going to be the cyclic cyclic release of the hormone. Let me make sure that we understand that of the hormone. Now, is it all hormones? Nope, it's not. Some, I gave you a few examples there. Now something else. I want to mention, I have the word all hormones, all hormones here and some hormones exactly as I have it here. I don't want you to think that they're correlated together, I don't want you to think that the sun hormones that are regulated narrowly, or the sun hormones that are cyclic be released know, two completely different stories, all right.

52:36

Now. Now there's some stuff in the notes that might look a little bit daunting. And it's the last couple of pages of the notes, I have scenario 123, and four, what I'm going to do is I'm going to simplify, it's still important to know the information, but what I want you to do is not so much take the words in your notes, but take the words that I'm going to have on the screen, and the words that I'm going to say to help you understand what we need to know from this. And it's going to be mentioned again and again and again and again, in the next chapter because we're going to be talking about specific examples of these things. So what we're going to do is this, so that last page in your nose, your last two pages, I don't even freaking remember, maybe it's the last page, it doesn't matter. Scenarios 123 and four is what I'm talking about. And a little bit before it is combining two things. And what are those two things, those two things are hormonal regulation, okay, is one of the things that we're going to be talking about when it comes to that particular last page. And hypo secretion hypo secretion. So we're going to combine pathophysiology, we're gonna do a lot of pathophysiology. Now, in a general sense. In the next chapter, we're going to talk about specific conditions. All right, when it comes to hypersecretion, and hypersecretion. So let's take this information that you see on the

screen and combine it with this information that you see on the screen. And actually now what's going to happen to is, if you recall, I said that we would mention it later. These two lines are going to come into play in the story that we're about to tell. This is going to be what we assume, again, hormonal regulation. So we're going to have two glands of land x and the gland y. And this is going to be our assumption. So, gland x squared y. gland. Excellent. You make it the right shape was the square or gland Why was the squaring gland Why was this oval? Can you so

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here's our gland why? releasing hormone why, and it's going to be regulating gland X. Which releases hormone X. And I'm gonna put a plus sign here that talks assigning stimuli.

55:02

So this is our assumption, given our story here. If we increase hormone y, given it has a stimulatory effect on gland X, that should lead to an increase in hormone X. That should hopefully make sense. And if we decrease hormone y, well, then the opposite is going to happen. We're going to get a decrease in hormone x. That's the assumption we're going to make. Now, could I have had gland why inhibit gland acts I could have, I just chose not to. Now what? Now let's get into these scenarios, we're going to talk about hypersecretion and hypo secretion. So this is going to be let's go hypersecretion first. And again, this is not good. We don't want this, we know that we've lost negative feedback. And so what we're going to do here is this, we're going to draw our homeostatic line. And then, unfortunately, we're gonna hyper secrete some hormone, the hormone that we're going to hyper secreted each and every one of these particular examples is going to be hormone X, that's where the story is going to start. So this is hormone level hormone, X level in the blood. And we're hyper secreting, we don't want to do that. Now, we have another hormone that can read that can regulate hormone x, and that's hormone. Why? Well, so let's talk about a couple of scenarios where something like this will happen, where we lose negative feedback. And there'll be two scenarios when it comes to hormonal regulation, if we're just talking about a one to one relationship, gland y and gland X together. Here's something else that I want you to understand, too, is that I have a one to one relationship with hormonal regulation, there could be four glands that regulate a single gland. There can be multiple, multiple, multiple things that are not hormones, regulating a single gland. And once you understand that, you're going to see in the next chapter, I'm going to show you over and over again, but I just want to make sure that you understand that it's redundancy, which is a good thing. And I'll talk about why later. But anyway, let's get back to this story. So how can that happen, which again, is not a good thing. So we're going to go once again, gland y. And we'll label it hormone y. And we're going to have gland X. And you're going to answer some questions for me. releasing hormone x Now, we know that hormone X is high. How do we know that? Well, because we took our patient's blood and we sent it to a lab. When we say lab, what's the level of hormone x and this patient? And by the way, if we know that hormone X is regulated by hormone why we're going to ask the lab give us hormone y to so that we can really get a good picture of what's going on with the patient. So we know that hormone X is high because the lab told us now let me ask you this gland why hormone why regulates gland X hormone X? If that's the case? Tell me what high levels of hormone x should do to gland why? What should we do to hormone? Why should we make hormone? Why go opera should we make Carmine why go down? If we want to get hormone x back down to where it's supposed to be? Yeah, we need to lower hormone. Why? Because if you lower hormone, why what happens? Or mon X goes down. That's already been established. And that's what we want to have happen. Because hormone X is high we need to do here. So there's a feedback mechanism. gland y given that it's

regulating gland X, and hormone X can detect the level of hormone X in the blood, it can detect it, it better. How else would it be able to regulate this hormone if it didn't know what the level of the hormone is? It can't guess. So gland y can detect the level of hormone X in the blood. That's how it's gonna work. So there's a feedback mechanism. And you just told me that we should inhibit gland why? So I'm going to put a negative sign there. That's what should happen if negative feedback is working here. We should inhibit gland why and what should happen is hormone Why should go down and then gland x should be inhibited as well. And then hormone x should go down so we should have the normal level of boat. But what's the bloodwork going to show us the blood work is going to show us this.

1:00:03

It's like what the hell is that about? That's not supposed to happen. The gland is supposed to be inhibited, the level of the hormone should go down. If negative feedback is working hormone, why should be low in hormone X is high. But it's not, which is why we're up here. So given this scenario, given that bloodwork, seeing that hormone, x and y are high, like that tells us where the problem is. Where's the problem? The problem is with this gland, it's not behaving properly. It's supposed to be inhibited. And yet it's not. It's actually being stimulated. Let me ask you this. Is gland X behaving properly? Isn't it? What should happen to gland X, when hormone wise high, it should release more hormone X. gland X is doing exactly as its told, it doesn't know any better. It's being told to do what it's supposed to do. And so it's doing it. So why the hell is Glenn y doing this? I'll give you an example. Cancer? Do you think cancer gives two shits about homeostasis? Of course it doesn't. So we have a cancerous gland that is just pumping out a bunch of hormone y causing a whole bunch of hormone x to be released, which then leads to bad things? Are we gonna talk about some tumors of glands in the next chapter? Of course we are? We absolutely will. Okay, specific conditions with specific hormones we're going to learn about. So that's one scenario. Here's another scenario. So we're gonna have our gland y is going to be releasing hormone y, we're gonna have our gland x, again, is going to be releasing too much hormone X. There should be a feedback mechanism. It should inhibit the gland, this specific gland. And when you send that bloodwork up, lo and behold, hormone y is low. And when hormone y is low, well, then hormone x should be low. But it's not. Is gland wide behaving properly? Yes or no? Yeah, it's being inhibited by a high level of hormone excess. What's supposed to happen who's not listening here? gland accident listening. And x is where the loss of feedback or negative feedback has occurred. So now our problem is here. What could be happening here? Well, we could have a tumor, this claim, then necessarily have to be a tumor. By the way, I just threw that out there. Because I think we all know what a tumor is cancerous. Doesn't necessarily have to be that though. But commonly, we'll see. And when we talk about these kinds of things, the next chapter tumor is going to come up a number of times. And so we have a role gland over here. So why is the level of X high in this particular situation? Because the gland is can't gland access cancers, not gland. Why? Why it's so important to know. Because now you know how to treat the patient. You're not going to go after gland, why in this case, gonna go after gland x. Or in this particular case, you're gonna go after Glenn, why not gonna go after gland x, given the bloodwork? And you know, like that immediately? How do you know to test for these hormones, by the way, because you look at the signs and the symptoms of the patient based on the hormone, we're going to learn the functions of hormones. It's a big part of the next chapter. And when there's too much or too little, there are certain signs and symptoms of a particular hormone that will point you in that direction. And then it's up to you to diagnose the patient will do some blood work. It could be that these hormones come back normal and the signs and symptoms of the patient have absolutely nothing to do with the freakin hormones. And it's something else and you got to figure it out. It's gonna be most of your job someday. All right. Now let's go hike boasts accretion, let's go the other direction. Again, no good. So we are on the draw. I drew my line. We're good. We're good. We're good. We're good. Now we're not good. Plan x plan Y

again. So here's our gland. Why the stories you know, it's going to be the same except while we're going in the opposite direction here when it comes to the amount of hormone that's being released. So we have low levels of hormone X. There should be a feedback mechanism. Instead of inhibiting, we're going To stimulate, we want to raise hormone X, how can we do that? Stimulate gland? Why and make gland Why are hormone Weigle? Up which will make hormone X go up? That's what should happen. It's obviously not, because we are chronically low with hormone x, and that's hormone x. This is hormone X level in the blood.

1:05:25

So where's our problem? Well, our problem is, once again going to be gland, why? The levels are low, they should be high. So we've lost negative feedback again. So here's our problem. Now, what could happen here? Could it be cancer? Yeah, cancer can destroy a gland. Instead of just making the cells cancerous and just pumping out, eventually it could destroy the gland. So I'm just going to say destroy, or your immune system can eat the glandt. We're going to talk about some autoimmune conditions. type one diabetes, for example. Hashimotos disease. Those are autoimmune conditions where we're eating specific glands. And then we can also have another scenario here, where we have gland why hormone y gland X. Hormone X is once again low. We know that because we did blood work should be a feedback mechanism should stimulate in this particular case, it does gland wise behaving beautifully. It's desperately trying to raise the level of hormone x, but it's not going to work. Well, because we're destroying this claim. Maybe this is Addison's disease. We'll talk about Addison's disease and other autoimmune conditions condition or an autoimmune condition at least Addison's will say, all right. So this is what I want you to know from that stuff that you see in the back of the house was scenario 123. And four, I don't need you to know that this one, scenario one and two, and three, you know, we're going to apply this stuff in the next chapter. Now, the very last thing in that first chapter is this primary condition, secondary condition, I'm going to save it, I'm going to save it. And the reason I'm going to save it is until the next chapter, that is because when we get the actual primary and secondary conditions, we're not done, we're gonna go to the next chapter. I wasted no time this semester, because we have so much stuff. I got seven and a half minutes left, I think, yep, eight minutes, actually. So we're gonna, we're gonna dive into the next chapter. So we're going to come back to this, I promise you, we're not going to we're not going to skip this stuff right here. Right? This is more pathophysiology stuff, by the way. Next chapter chapter two, now we get to talk about specific hormones. And the way we're going to tackle it is we're going to start with the hypothalamus and then work our way all the way down to the pancreas. Talking about all these hormones, I don't even know how many maybe 20 or so hormones, give or take. Everything starts with the hypothalamus. And we know what the hypothalamus is you guys learned about last semester, specifically with lecture Seaver. That little teeny 1% of your brain, right? Hypothalamus is kind of considered the brain of the brain. Hypothalamus is part of the nervous system. It's also part of the endocrine system. Your hypothalamus directly or indirectly regulates most endocrine activity. What does that mean? It means that hypothalamus is regulating a whole bunch of stuff within your body almost every hormone in a direct or an indirect manner. I'm going to show you how your hypothalamus produces eight neuro hormones, there's the word neuro What's so special about a neural hormone? Nothing. It's a hormone like any other hormone in the body. It just happens to be produced by neurons. So when we drew our very first picture, I said, that is glands but not always that produce hormones. When I'll give you an example where it's not a gland. It's not epithelial cells, it's actually neurons. So your hypothalamus has vana for what glial cells and neurons, some of the neurons in the hypothalamus produce hormones. And if they're produced in those neurons, we just call him neurohormones. Nothing special about him whatsoever. They act the exact same way. So we have these neural hormones, and there's eight of them. Six of them are what we call releasing and inhibiting hormones. Oops. And then we have the other two, which we have here. So what I'm going

to do is I'm going to draw that. And this will be the last thing we do tonight. And then we're going to continue on with this on Thursday. Now I'm going to draw the hypothalamus is that just this big rectangle across the screen, so hypothalamus. And here it is. That's the hypothalamus. And we're going to put all eight of those hormones in the hypothalamus. Now I'm going to abbreviate the names because the names are long. The first six are what are called releasing and inhibiting hormones and I'm going to use different colors and they tell you why we use I use the colors that I'm going to use when they use green and red. The first one is gonadotropin releasing hormone, G NRH. And then the next one is pH, at least I think it is, let me make sure I want to put these in the same order that I haven't enough. It is pH. pH, I'm going to draw in red.

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Because it's an inhibiting hormone. It inhibits something. releasing hormone is stimulatory. So green for go red for stop. How clever is that? Then we have growth hormone, releasing hormone. And then we have growth hormone. inhibiting hormone, the name of the hormone, they tell you exactly what they do. Then we have two more that are going to be green. We have TRH thyrotropin releasing hormone. Then we have corticotropin releasing hormone so those are stimulatory. So those are the first six, then I'm going to put this imaginary line here and put over here not red or green because these are not releasing and inhibiting hormones. Oxytocin an antidiuretic hormone ADH. So those are the eight. What else are we going to put in this picture, we're going to put the pituitary gland in this picture. We have the anterior pituitary gland, we have the posterior pituitary gland. So this is going to be in I'm going to label it that's the anterior pituitary also in the brain and we have the posterior pituitary over here. Also in the brain these first six hormones are releasing in the inhibiting hormones are going to regulate hormones of the anterior pituitary. It says right here in the notes. Six releasing, inhibiting hormones regulate the anterior pituitary or that right there it is. And so what's going to happen is this, we're going to have these red tubes. And these red tubes are blood vessels. And these red tubes are going to circulate these hormones from the hypothalamus to the anterior pituitary gland. And in the pituitary gland, we're going to have hormones that are produced and released and I'm going to write down what they are. Go to banana tropic releasing hormone controls to hormones of the pituitary gland ludie ionizing hormone and follicle stimulating hormone and so GnRH will make its way and stimulate the release. What kind of regulation is this by the way? Hormonal, one hormone regulating another right. PIH, prolactin but it's going to have an inhibitory effect. Again, there we have hormonal regulation ghrh and GH i h do different colors. Both affecting growth hormone TRH is going to affect thyroid stimulating hormone TSH and CRH is going to affect adrenal cortical Tropen. Hormone both are going to have a stimulatory effect. Again, hormonal regulation one hormone regulating another well, we hear the stories different. Put a little holding up a little hole they're not going to have a blood vessel here. Instead, I'm going to have a tract tell me what a tract is, please. It's a bunch of white guys with an A analogous to a nerve. Axons remember that from last semester, a tract is a bunch of axons. What is producing these hormones? Neurons the cell body. So we have these cell bodies over here in the hypothalamus. They produce the hormone and then these hormones oxytocin and ADH. They travel down the axons to the very edge of the presynaptic terminals. Last semester I talked about how neurotransmitters are released the presynaptic terminals these hormones are going to be released at the presynaptic terminals and where they're going to go. We're going to go to the anterior the posterior pituitary gland. Your posterior pituitary gland does not produce any hormone. Now, you might say to yourself Well doctor All right thought you said that hormones need to be dumped into the blood for them to be hormones they do. The posterior pituitary is going to dump these two hormones into the blood. It's gonna happen. All right. Okay, folks 620 I'm done. I will see you guys on Thursday when we continue this story Serbian

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SUMMARY KEYWORDS

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Alright, folks, here we go. So last thing we did is what's up on the screen. So we talked about these eight neural hormones that are produced by the hypothalamus, six of them are releasing and inhibiting hormones, which are directly affecting the anterior pituitary. And then we have these other two that are simply delivered to the posterior pituitary. So what we're going to do today is we're just going to start knocking off these hormones and talking about them one at a time. And we're going to start with these pituitary or these posterior pituitary hormones, oxytocin will be the first one that we discuss. And so let's do that. Now, when we talk about these hormones, they're going to be tackled the same way. We're going to start with function, and we're going to talk about what stimulates them. And then we're going to talk about what inhibits them. So we're going to do the physiology first. And then we'll do patho fits, we'll do hypersecretion hypo secretion. And we'll get into certain condition syndromes, diseases that are going to be very, very important for you guys to know. So with oxytocin, let's talk about functions first, stimulates uterine contractions, I'm going to assume you know what the uterus is, right? Females have a uterus, that's where the baby is going to go and grow if mom is pregnant. And so oxytocin, which is of course, released in the brain will get released into the blood, its target is going to be the smooth muscle of the uterus. And it's going to cause that smooth muscle to contract. So that this so that the uterus can contract now, why would that be happening? It's going to help the baby birth, baby needs to leave the uterus, the baby isn't going to kind of do this, it's got to be pushed out of the uterus, and the contraction of the uterus is going to do that. So oxytocin is involved with the birthing process. And we'll talk about that in just a second. What else stimulates the release from the lactating breast. So oxytocin is also going to be involved in lactation. So when Mom has that baby, and she wants to breastfeed the baby, oxytocin is a hormone that's going to allow her to breastfeed the baby. When it comes to oxytocin, most books, most web pages that you'll see online, talk about oxytocin in relation to what it does for females, well, males have oxytocin as well. And one of the things that oxytocin does in males is it facilitates the transport of sperm within the reproductive tract that is the male reproductive tract. And then the last thing here is it plays a role in bonding. Oxytocin is known as the love hormone. So when you bond with somebody, when you become friends with somebody get to know that particular person and you start to get closer, you feel closer to that person, in part because oxytocin is being released as you are doing that. Now, something else I want to add here is that these aren't all the functions of oxytocin. And the functions that you see underneath each of the hormones we're going to discuss are not all the functions of those hormones. There are many, many, many, many more, we just don't have time to talk about them. So the ones that are in the notes are the functions that I obviously want you to know. Now, those are the functions of oxytocin that I want you to know what's going to stimulate oxytocin? Well, dilation of the cervix is one of them. Do we know what the cervix is? It's the opening to the uterus. Why would the cervix be dilating? Anybody know? The baby's getting birth, babies got to leave the

uterus. And so that opening can't remain very, very close. It's got to dilate. So if the cervix is dilating, that's an indication that the birthing process is commencing. That means oxytocin has to be released to squeeze the uterus to help the baby birth. And so that is a signal for oxytocin to be released, that it's a stimulus for the release of oxytocin. What else distension of the uterus, it's all part of the birthing process once again. So more and more oxytocin is going to get released, suckling at the breast that has to do with lactation, every time the baby breastfeeds, it's going to cause more and more oxytocin to be released. And by the way, as that cervix is dilating, more and more oxytocin is going to be released as that uterus is distended during the process of birthing, more and more oxytocin is going to be released, what kind of feedback mechanism does that sound like? That sounds like positive feedback to me. And that's exactly what it is. And actually, because of that, let's do this. So let's draw the level of oxytocin during those two special events. And we have two special events here, right, the birthing process and lactation. And so this is going to be oxytocin. And again, let's remember that this is oxytocin levels in the blood.

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So when I talk about the level of any hormone, it is the blood level of that hormone. Now before this special event, oxytocin is working in a negative feedback manner. and it is staying within its normal range. But then we're going to have the commencement of this special event. And we're going to actually name them specifically, birthing process will be one. And then I'll put or lactation. But the feedback mechanism is going to look exactly the same. And so as these special events are occurring, as we know, the level of this particular hormone is going to climb, and eventually it's going to kind of reach a plateau. And so we have a positive feedback mechanism going here. And we must if I see Tosun, was still acting in a negative feedback manner, you're not birthing the baby, you need the level of oxytocin to go up, up, up up up, in order to squeeze the uterus hard enough to get the baby out. In order for that baby to be fed milk from mom, the level of oxytocin must be high enough in order for that to occur and what oxytocin is doing. And that feeding process is allowing what's called the expression of milk, that's kind of the fancy way of saying the milk is released from the from from the breast, so that baby can actually feed another hormone is going to be involved in lactation prolactin, we'll talk about that later, that hormone allows for the production of milk. So oxytocin does not cause the production of milk. That's prolactin. Oxytocin allows for the release of the milk, the expression of the milk, so you need both of those hormones to work in concert with each other. So as these, as these two special events are occurring, we're going to have the positive feedback mechanism happening. But then, once these particular processes are done, there's no longer a reason for the level of oxytocin to be high. And so we're gonna go right back to negative feedback. And we're going to be here. Now, when we looked at this picture, and we do it at the end of class, you guys told me that this right here with the six, releasing and inhibiting hormones affecting these hormones of the anterior pituitary that that was what hormonal regulation, right? These hormones regulating these hormones. So that was a hormonal regulation. That's something that we established in the last lecture. Well, we hear we have regulation of occurring as well. When it comes to the birthing process or lactation, causing the release more release of oxytocin. It's not hormonal regulation. Now, certainly, birthing process and lactation are not hormones. So could it be non hormonal? It could, but what's the third one? What's the third regulatory mechanism we talked about? Neuro. This is neural. So what this does is this, at the initiation of the birthing process, the initiation of lactation, what these will do is that they will initiate an autonomic reflex. Did we talk about autonomic reflexes last semester? Of course we did. We did the baroreceptor reflex. We did the micturition reflex, we did the defecation reflex. Those are autonomic reflexes. Well, here's another one. And so when it comes to this, and what kind of regulatory mechanism we have going on here, it's neural regulation

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via the autonomic nervous system. Now, one of the things I want to add here is this.

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If the birthing process is not occurring, and it's not progressing properly, and the baby just isn't getting birth, it could be because oxytocin is not being released. And what can be done is is that she can be given an artificial oxytocin. Does anybody know what that's call? starts with a P. Pitocin. Anybody here ever hear Pitocin Pitocin is something that you would give a mom who's not birthing properly, to make the birthing process happen, and so Pitocin given if the birthing process does not proceed properly. She's stuck in labor for a very, very long time. So you tell me Pitocin is what to the oxytocin receptor starts with an A, an agonist, right? We learned that last semester, it was the second chapter, remember that? And so I'll put that over here. Pitocin is an agonist of the oxytocin receptor it will bind To the oxytocin receptor, just like oxytocin does and activate that receptor. It's got the same target. So that's an example of a hormone that works in a positive feedback manner. It also works in a negative feedback manner. Of course it does, we can see that here, but during the special events, positive feedback manner. Now, one last thing that's in the notes, what else is going to stimulate oxytocin? Well, that's intimacy. And that's, again, the love hormone bonding, and there's all kinds of different intimacies, you just get to release more oxytocin and it helps you bond with people. Okay, to make a connection. What inhibits oxytocin, we really don't know of anything. When you have too much oxytocin, what's gonna happen? Not a whole lot? What about too little oxytocin? Well, obviously difficulty with the birthing process, which is why you would then give Pitocin and then you would have difficulty with lactation, of course, you would, you wouldn't be able to express the milk, you might be able to make it, you might have enough prolactin, but if you don't have enough oxytocin, you're not gonna be able to breastfeed baby. And then what else when it comes to males, you might not transfer that transport that spring quite as well. Actually, Tosun says simple one, because there's really no pathophysiology here. The next one, however, we're going to have some pathophysiology. And the next one is ADH. ADH is the other hormone that's being released by the posterior pituitary. And so let's talk about ADH. What are the functions of ADH, there's two that I have in the notes, one of which you're going to have to know and that's the first one Regulates Blood osmolarity. Tell me what osmolarity is again. Measure of the concentration of the fluid, right, that's all osmolarity is, tell me what the normal range of osmolarity is, please, I'll give you the first number 280 200 300. Remember that. So 280 to 300 is a normal osmolarity. Within the body most places in the body. There's some exceptions, we'll see one of them this semester. Blood osmolarity, between 280 and 300, is regulated by ADH. That's why you have that concentration range. And we're going to do a picture of it just a second. And ADH is going to do that because it's going to tell the kidneys how much water to transport into the blood. And if you can concentrate or I'm sorry, if you can regulate the amount of water in the blood, you can regulate the concentration of the blood. The story you don't have to know for right now is how it regulates blood pressure. And it doesn't even play a big role in the regulation of blood pressure. But we're going to talk about that later on in the semester when we do talk about blood pressure in the cardiovascular chapters. So even though you don't have to know it, now, you're going to have to know what again, you're going to see ADH three times this semester. Obviously, this is the first time you're going to see it the cardiovascular chapter you're going to see in the kidney chapter, I'm going to show you why you're going to see it the kidney chapter. Because the kidneys are the target of ADH. It's not the only target, but it's a target that we're going to be talking about. So what we'll do here is I'm going to show you how ADH works. We're going to draw the hypothalamus. And the hypothalamus, of course, produces ADH and then delivers it through the tracks, as we know to the posterior pituitary. So that's going to be the posterior pituitary.

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And there goes ADH down that track. And now the posterior pituitary has it and the posterior pituitary is going to release it into the blood. And when it does, it's going to have a specific target. And the target of ADH is going to be the kidneys. Now it's not the only target of ADH, but it's the one that's pertinent to this story. I'm going to make that plural because most people have two kidneys, I'm going to put a blood vessel. And that's going to represent the circulatory system. And ADH is going to make its way through the circulation to the kidneys bind to an ADH receptor of a kidney cell. And now the kidneys are going to know what to do. And so what are the kidneys going to do? Well that's more blood over here. So that's blood vessel. And so what ADH is going to do is it's going to tell the kidneys do that transport water into the blood. And if you control that you control the concentration of the blood such that if you increase ADH release what you're going to do is that you're going to cause an increase in water transport into the blood which means you are going to To decrease osmolarity, you're going to decrease the concentration of the blood, of course you will, you're going to add water to it. And then the opposite is going to hold true. If you decrease ADH release, well, then of course, you're going to decrease water transport into the blood, you're going to concentrate the blood, you're going to make the osmolarity go up. Oops. And then we're going to give an example of this two examples. So that's how it works. Now what? Well, let's look at blood osmolarity. And how if it goes up, what's going to happen to ADH and if it goes down, what's going to happen to ADH and the transport of water to maintain homeostasis. So this line right here, that's not ADH level, that's going to be blood osmolarity, let's make sure that we understand that. So this is blood osmolarity the concentration of the blood. And as we just determined just a little bit ago 280 to 300 is the normal range. And those numbers indicate the concentration of the blood. Now what's going to happen and ADH is allowing that to be where it's supposed to be because just the right amount of it is being released. But now what's going to happen is our bloods going to get more concentrated. And I'm just going to make up a number. I don't know 340. So now we're more concentrated solute concentration has just gone up. Now why would that happen? I can give you a couple of examples. Simple ones. One, you're dehydrated. You're not drinking enough water. And all the fluids of your body get more concentrated. So dehydration could cause this. Or it could be and or actually, you can see a lot of food. Especially maybe salty foods, processed foods. Foods have soul you will sell us they're gonna end up in the blood. So we could concentrate our blood needs to matters. What do we have to do? Well, we have to do this. We want to be within that normal range. We don't want to be above we don't want to be below. So what's going to happen as a result of this is that number one as we do as we have a more concentrated blood, we are going to increase ADH release. Does that make sense? We want to get more water into the blood. And as we do aren't we going to decrease osmolarity is that what we want to do? Of course we do osmolarity is too high we need to lower it how are we going to lower it you increase the release of ADH and so as we do that, as you increase the release of ADH you increase water transport into the blood and as a result number three you're going to lower osmolarity until we get back to where we're supposed to be what kind of releases that by the way what kind of release do we have we have chronic we have a cute we have cyclic? What do you think that is?

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A cute. That's a cute release. What kind of regulation is this? We're regulating ADH. What kind of regulation is this? We have neural. We have hormonal, we have non hormonal. What's this? It's not hormone blood. osmolarity is affecting ADH. It's non hormonal. So we have a non hormonal regulation. Let's go the other direction. Let's be thorough. Okay, let's say our blood osmolarity is too low. I'll make up a number I don't know 250 Well, why would this happen? Why would your blood get

a little bit more dilute less concentrated? Well, I'll give you two examples. You're not dehydrated. You're over hydrated. You're drinking too much water or you're not eating you might be starving. When you eat you eat solutes if you don't eat you don't have solutes will slowly loosen up in the blood. And if you're not eating, you're not going to have enough salt use in the blood so the blood becomes dilute. And so then of course what's going to happen here is and I won't go 123 I'll go ABC. So a you're going to decrease the release of ADH and when you do that, b You're going to decrease water transport into the blood If you don't transport as much water in the blood, well, what are you going to do? You're going to concentrate the blood, you're going to increase osmolarity pinkos. Now, here's something else I want to add to the story. I know that when I talked about, I'm going to go back to the picture that we did in the last lecture. And it was here. Okay, so we've established this as a cute release, right? Even if you decrease the release of something, it's still considered a cute because you changed it from chronic. Let me explain that just to make sure that you understand. So. This is acute release. That makes sense, right? We're increasing the release of ADH at that time. If you decrease the release of ADH, it's still it's still acute. Why? Because it was chronic. And now you've changed its release, increase or decrease. Now something that I forgot to mention in the last lecture, but I'm mentioning it now, this is still acute. Even though you're decreasing the release of ADH. It's an acute event due to some environmental factor, whether it's external environment, or internal environment, in this case, its internal environment, you've changed the environment of the blood. Just want to make sure that we understand that and it's non hormonal, the whole the whole thing here is non hormonal because it's blood osmolarity regulating ADH release. So this whole story is non hormonal. And so I'll put that here. It's non hormonal. Why? Because it's osmolarity. That's not a hormone regulating ADH. So acute, non hormonal, so we're visiting that. Are we good with this? Yes. Here's the thing. If you know the function of a hormone, you're gonna know what stimulates it, you're gonna know what inhibits it. What stimulates ADH, high blood osmolarity. What inhibits it, low blood osmolarity. And of course, that makes sense given

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what ADH does to the kittens, what ADH does to the kidneys, it should make perfect sense. And that's the way you're going to approach all these hormones, you always start with function. If you know what the function is, everything else is going to fall into place. That's where it should all start. Now, let's do some patho fits too much or too little. Again, don't worry about the blood pressure blood volume stuff, don't worry about that. Oh, one other thing too is as a stress hormone. You're going to release more ADH during times of stress. Now what stress stress can be physical stress to the body mental stress or emotional stress. And I'm going to tie that together, we're going to see that there are many stress hormones that are released. And there's actually a list of them that I'm going to have at a table. I don't know if it's in your notes. If it's not your notes, I'll present it to you guys. And it'll be a picture on pilot as well. Now talk about stress and stress hormones and what they do and how they're bad or how they can be bad. I mean, they help us deal with stress. But too much stress leads to bad things. And we'll talk about that a little bit at the end of this chapter. But again, blood volume, blood pressure, don't worry about it. We're going to worry about it in the cardiovascular chapter. Stress, though, do worry about it. Do know that. Now let's do some hypersecretion hypersecretion. So now what we're going to do, we're going to do the patho phase, we're going to do that. And so let's talk about too much or too little ADH and why they're both bad. And what I'm going to do is I'm going to just do a little compare and contrast. So I'm just going to have two different columns here. On the left hand side, the syndrome of inappropriate ADH, that's hypersecretion. Were secreting too much ADH versus too little ADH, known as diabetes mellitus. I'm sorry, incipit is not mellitus. And that's hypo secretion. Now I'm going to assume that when most people hear the word diabetes, they think insulin and blood glucose. That's diabetes mellitus. This is diabetes insipidus. Those two conditions have absolutely nothing to do with each other. We're going to talk about diabetes mellitus toward the end

of this chapter. This is not it. This has to do with ADH. And that's it. So let's compare and contrast again hypersecretion. So we'll just put a couple of arrows up for ADH. What's going to happen as a result of that, well, we already know. Too much water is going to be transported into the blood because well that's what ADH does. And so what that's going to lead to is increase water transfer. Sport and a big increase depending on how severe it is, by the way, you could have a mild form of this, you could have a moderate, or severe form of this and that's going to dictate how much water is going to get transported increase water transport into the blood. Which leads to then of course, blood that's very dilute with a decrease in osmolarity. complete opposite over here with diabetes insipidus really low levels of ADH, which means a decreased amount of transport into the blood that is water. And then obviously, the blood is going to get too concentrated. How dangerous are these, they can kill you. Depends on how severe they are. With diabetes insipidus, you can get dehydrated, in no time your patient gonna be dead in less than 24 hours with this particular condition if it's severe enough. So that's the worst case scenario. But certainly when you start to change the osmolarity of the blood, you're going to affect a whole bunch of stuff. We talked about some stuff last semester, you're gonna change membrane potential. First, you are going to change the concentration of sodium potassium chloride to those things change membrane potential, of course they do. So it can become very dangerous for a number of different reasons. And so these are two completely opposite conditions. Both of them again, could be very, very dangerous. So that's that for that. Are we good with this? Alright, so what are we done? Well, we just took care of the posterior pituitary hormones. Now what are we going to do? We're going to move on to the anterior pituitary hormones. And the first ones are LH and FSH. So we're going to this one here, we're just going to cross them off. So we cross these two off. Now we're over here with these gonadotropin hormones Udi and Ising, and follicle stimulating hormones, we're going to see that they work together in many cases, we're going to see that in this chapter, we're going to see it in the next chapter in the reproductive chapters lecture series is going to talk about LH and FSH a little bit. I'm going to talk about him again when we get to the reproductive chapter, which is what I'm not going to spend a ton of time on. But I'll spend a little bit of time on. So let's go back to the notes and let's talk about

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these two hormones. So luteinizing hormone is going to be the first one let's look at the functions stimulates testosterone production in males, which is going to help with sperm production, you need to start slow to produce sperm. So luteinizing hormone along with FSH and FSH directly stimulate sperm production, as we're going to see work together to produce sperm. But LH has its own little function. And that is, make testosterone. What else in females helps regulate the menstrual cycle? And that's really all I'm going to say about it when it comes to the menstrual cycle. Because I'm going to go into the menstrual cycle in detail when we get to the reproductive system. I'm going to show you how it happens. You're going to have to wait, what stimulates LH GnRH? Well, we already knew that. How do we know that? It's right here. So there's a releasing hormone from the hypothalamus that neurohormone is going to is going to stimulate LH and FSH for that matter. What else? Well, of course, low testosterone levels, of course, actually, let's do this one really quick. So what we'll do here is this is actually going to be hormonal levels, and the hormone is going to be testosterone. And we're going to talk about testosterone in the next chapter in the reproductive chapter. But I mentioned it here, obviously, because Lh is what stimulates his production. So we have our testosterone levels, everything is good. And then let's just say all of a sudden the levels start to drop. We don't want that we want them within the normal range. And so what's going to happen? Or what hopefully happens is that we're going to raise the level. Well, what's going to do that for us? Well, right here at this particular point, we're going to increase LH release more of it's going to get released. And then we'll raise testosterone production. What kind of regulation is this? Testosterone is a hormone hormonal. So this is hormonal, hormonal regulation. What kind of releases this cyclic,

chronic, it's acute. This is the acute release. So acute release of LH Alright. And then we'll be thorough, let's say the levels are a bit too high. Well, now we're going to decrease LH reliefs. This is still acute. It's just that the levels have gone down. There was a change from chronic and by the way, we were chronically releasing LH over here. Not that that's the trace for LH, but we were prior to this. And so then once again, we are going to lower the testosterone levels. And that's one danger, like taking anabolic steroids, which I really don't get a chance to talk about. But what you will do is that you will fool the body into thinking that it has high levels of testosterone. And if you fool the body into thinking it has high levels of testosterone, you're gonna stop making testosterone, you're gonna stop releasing LH. And depending on how abusive you are with anabolic steroids, it can lead to some long term problems that takes some drugs to kind of kickstart the production of testosterone again. So anyway, hormonal release, I'm going to put a cutie over here just to make sure that we understand that it is both even though we're decreasing the release. And of course, it's horrible. Of course it is. Now what, let's go back to the notes. What stimulates LH again, we just established low blood testosterone, what inhibits LH we just established it high blood testosterone and of course, low GnRH. So that's the fits all this is the fits this picture physiology. Now let's do some patho fifths. What happens if you secrete too much? LH we're going to have really stinking high blood testosterone levels, can you have too high level you can't. What else and this is females, the menstrual cycle gets screwed up. And you have what's called amenorrhea. What is amenorrhea amenorrhea is not having a menstrual cycle when you're supposed to a five year old little girl does not have amenorrhea. She never had a menstrual cycle to begin with. But once the female reaches puberty, and she's supposed to have a menstrual cycle every month and she misses one that's called amenorrhea. LH, regulates the menstrual cycle. So if the levels of LH are either too high, or too low

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amenorrhea can occur. We're going to see in the next chapter, that Lh is released in just the perfect way during the menstrual cycle in a cyclic manner that allows the menstrual cycle to proceed properly. So off is always high or always low, that's no good. It's supposed to be low at some point higher at other points low again, it's going up and down during the menstrual cycle is I'm going to show you if it's chronically high or chronically low, menstrual cycles not going to work properly. So that's what that's about. What else low of course, low blood testosterone and if your testosterone levels are low, your sperm count is going to be low because you need to start strong to produce sperm show you in more detail in the next chapter. Now because of the low blood testosterone this guy's decreased libido that we'd all libido is. sex drive, now can be in the mood. Erectile dysfunction. Stuff ain't gonna work. If your testosterone levels are low, and it could be because of LH hypo secretion. There are other reasons that your testosterone levels could be low but LH hypersecretion is certainly one of them. All right. Next one on the list is follicle stimulating hormone again works. Along with LH I've already talked about testosterone and sperm production. follicle stimulating hormone actually directly stimulates sperm production. LH is involved because it stimulates the production of testosterone, which is needed for sperm production. So we can see how these two hormones are working together. And I'm going to show you again in the next chapter, when we get to the reproductive system. It'll also stimulate estrogen production, that is follicle stimulating hormone DUTs. And again, the menstrual cycle, FSH is also involved in the menstrual cycle, and works in a cyclic way, just like LH does they actually mirror each other. What stimulates FSH? Well, we already know GnRH does? Of course we do because we've seen this picture. GnRH stimulates FSH, what else do we need to know? You don't know what inhibit is, oops, we're over here. low estrogen levels. That should make sense. If your estrogen levels are low, we need to raise them. This is the hormone that'll do it for us. So we would stimulate this particular hormones production and release. And then low testosterone levels in males that doesn't have anything to do with follicle stimulating hormone directly, but it has to do with sperm production. Again, I'm going to put that story together in the next

chapter. So if it's not making perfect sense right now it will in the next chapter, I'm going to show you how LH and FSH work together when it comes to testosterone and sperm production. All right, I promise. What else? Well, low GnRH. Oops, nope. High blood estrogen is going to inhibit FSH. Of course it will. If your estrogen levels are already high You're not going to want to release a hormone that makes it even higher. So you'll inhibit this hormone inhibin. I'll show you what that is in the reproductive chapter. So more to come on these hormones, I promise. Low GnRH Of course, and then stress, stress will inhibit the release of FSH. It doesn't mean it's a stress hormone. By the way. Stress hormones are hormones that are released because of stress. If a hormone is inhibited because of stress, it's not a stress hormone. Stress hormones are hormones that are stimulated by stress released in response to stress. So it's not a stressful one. So when I put that table or that list together, FSH is not going to be in a list of stress hormones. Because it isn't one ADH is and there's going to be many others as well. Now affects if FSH hypersecretion if you release too much of it, of course, you're going to have high estrogen levels. Why? Because it makes estrogen and causes the production I shouldn't say it makes it it causes the production of estrogen, too much of it too much estrogen. And then once again amenorrhea to high level of FSH or to lower level of SF FSH, the menstrual cycle does not happen. The way it's supposed to, just like with LH has to be released in just the right way. low estrogen levels of course, if FSH is low, and then of course a decrease in sperm production because FSH stimulates sperm production. All right. On to the next one, again, more on FSH and LH later, although the LH and FSH story are going to be repeated with prolactin, you're going to see how and why. And actually, before I do that, let's just take a break. When we come back, we're going to talk about prolactin.

37:03

I think we are all back. So let us continue. So next on the list is prolactin. And so let's talk about prolactin. Let's talk about functions of prolactin, prolactin along with oxytocin, it's going to help with lactation. So oxytocin once again, a lot for the expression of milk, we got to make the milk and that's what prolactin is going to do. So it has the same target as oxytocin does the mammary glands, stimulates breast development in females and males. Modulation is a fancy word for regulates the production of testosterone and sperm in males. Now, what's going to stimulate prolactin? Estrogen and that's something we're going to talk about later. Suckling of breasts that has to do with lactation, and breastfeeding the baby of course, and then decreased levels of pH and then what inhibits prolactin pH continuously inhibits prolapse and I'm going to tell you what that means. And so what we're going to do here is we'll do this so prolactin levels are typically really, really, really, really low. And that's prolactin. So prolactin is low, almost always accepting the lactating female. Why is that it is continually inhibited tonically inhibited by prolactin inhibiting hormone, and that prolactin inhibiting hormone is coming from the hypothalamus, as we will know it's sitting right here, and then just continuously release, it trickles on over to the anterior pituitary, and it just keeps prolactin levels low. Except again, when a female is lactating, and wants to produce milk for the baby, then the levels go up. And that's something that we're going to be talking about. Now. Let's go patho Phys. Now hypersecretion of prolactin and this might not make any sense, but it will when I'm done telling you a story. And that story is going to be lactational amenorrhea. So I'm going to tell you why this actually happens. But the first thing that we're going to do is patho phys hypersecretion, not due to lactational amenorrhea. So the effects of prolactin. hypersecretion is that it inhibits GnRH. So let's go back to the first picture we drew when it came to this particular chapter and that was this one GnRH is regulating LH and FSH. We can clearly see that we've mentioned it a couple of times today and then PIH is regulating prolactin prolactin levels go up, up, up, up, up, up, up, up up, what prolactin will do is actually come back and inhibit that hormone right there. And I'm going to tell you why that happens. It's an evolutionary thing. And it's about what's called lactational amenorrhea. But before we get to that, let's talk about some other things. So if high levels of prolactin are going to inhibit GnRH, well,

that means that LH and FSH are going to be inhibited because GnRH controls LH and FSH. So high, prolactin is going to indirectly cause a decrease in LH and FSH. And so let's look to see what happens and remind ourselves what happens when we have low levels of LH and FSH. Let's go back over here. Low levels of FSH. What's our I'm sorry, LH, low testosterone levels in males, which will decrease libido. Erectile dysfunction in females amenorrhea. Low levels of follicle stimulating hormone low estrogen levels amenorrhea decrease in sperm production. So now what we see here is high levels of prolactin inhibiting GRH, then causing low levels of LH and FSH cause low estrogen levels. Yes. amenorrhea. Yes. Low testosterone causing decreased libido, erectile dysfunction, decrease in sperm production. So it's just a repeat of what we just learned a second ago. But I've given you a specific reason as to why LH and FSH levels would be low. Because prolactin is inhibiting them indirectly through GnRH. Now, this is something completely separate GALACTA Ria, so again, we're still having high levels of prolactin here.

41:46

This is somebody who's not breastfeeding, by the way. GALACTA Riya is spontaneous milk production, it's got nothing to do with lactation whatsoever. She did not just have a baby, her levels of prolactin just went up. Now, why would they be high, it could be a tumor, it could be a tumor of the pituitary gland causing an increased level of prolactin, that could certainly be one of the reasons that is occurring. Again, we're talking pathophysiology right now, we're not talking naturally high prolactin levels due to lactation. And so let's actually do that here. So again, really low levels of prolactin, why PHS continuously produce unless we have a special event. And what's our special event going to be lactation. Prolactin, by the way is not involved in the birthing process. Oxytocin is but oxytocin and prolactin together are going to allow lactation. So this is lactation. And what's going to happen as a result up, up, up, up, up, it'll plateau at some point. We need this to occur. Why? Because if we don't, the baby's not going to get enough milk, the baby will feed one time and then won't be able to feed ever again. The more the baby feeds, the more prolactin is going to be released. So we have once again a positive feedback mechanism. And once again, this is going to initiate that is the baby suckling the breast is going to initiate an autonomic reflex. So we initiate an autonomic reflex. And so once again, we're talking neural regulation. And then when mom decides, you know, why didn't want to breastfeed anymore? Well, then we're back to low levels again. The negative feedback takes over. And as long as mom wants to breastfeed, the level of prolactin is going to be high level of oxytocin is going to be high, whether that's one or two years. If it's monkey breastfeed for seven years, if she wanted to the level of oxytocin and prolactin would still be high, given this positive feedback mechanism. Now, let's go back to the hypersecretion. Oh, and before that, we'll just do hypersecretion just to kind of get it out of the way, low levels of prolactin while you're going to have a lack of milk production. So you might not be able to breastfeed babies in males, low levels of prolactin, we don't know of anything that goes wrong. The levels are so low to begin with, that lowering even more is not going to make one bit of difference. So no known effects in males. The only known effects that we know of is going to be low effects during lactation where you're not going to be able to breastfeed the baby. So that is patho fits. Now let's talk about when prolactin levels are high, naturally during lactation, so it's going to be hypersecretion but during lactation, and I'm going to show you why this is going to To be this evolutionary thing that occurs and is going to occur for a reason, that is why hypersecretion is going to cause this stuff to occur, I'm going to show you something called lactational amenorrhea. That's what we're going to do now. So it's just one little itty bitty line in your notes. But it's going to take an entire page to describe. So I'm going to show you now. So this is lactational, amenorrhea, something that happens once again naturally

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during breastfeeding, this is 100% Normal. So what we're going to put in this picture is the hypothalamus, the anterior pituitary. And so here's the hypothalamus. And I'm just going to draw it as I always do as a rectangle. And it's going to be GnRH. And so GnRH is going to be released here, because that's part of the story. And then we're going to have the anterior pituitary down here, and I'll label it and it's going to be released and I'm gonna put it over here on this side, prolactin and a lot of it. Why because mom is breastfeeding, this is during lactation. So number one, we have really high levels of prolactin. What is that going to do? Well, we already know, it's going to feed back onto the hypothalamus and specifically inhibit GnRH. And so number two, we had this inhibitory effect on JRH production and GnRH release. And so number three, we're going to have really low levels of GnRH. And if we have really low levels of GnRH, oops, well, then we're going to have really low levels of LH and FSH. And as we know, because we just saw it a number of minutes ago, when LH levels are really, really low and FSH levels are really, really low. The menstrual cycle gets screwed up. And during the menstrual cycle, what's going to occur about midway through their menstrual cycle, does anybody know what it's called? Starts with a no you ovulate but if you don't have a menstrual cycle, you're not going to ovulate. So no ovulation that'll be number five. And this has to do with amenorrhea. So I'll kind of put in here in parentheses, not that amenorrhea specifically means no ovulation but certainly without the menstrual cycle, the ambulatory process is not going to happen. And so oops. If you don't ovulate, what can you not get? You can't get pregnant. ovulation is the release of the secondary Oh site from the ovaries. Without that oh sign out there, the sperm has got nothing to nothing to fertilize. And so no pregnancy. This is why this happens. This is why high levels of prolactin cause the low levels of LH and FSH, it's trying to prevent pregnancy. And by the way, for about the first six months during lactation. It's 98% effective against getting pregnant. first six months, and then it kind of drops off after that point. For six months during lactation. It's about 98% effective now that's 98%. That's not 100. The number of years ago, I actually had a student come up to me after the lecture and said I was I was one of those 2% She got pregnant while she was breastfeeding her baby. Now why? Why would the body try to prevent pregnancy during breastfeeding? Let's think about that for a second. We have a baby, the baby was just born. what the body wants is for mom to take care of the baby that she has devote all of her energy to that baby. If mom gets pregnant, that's going to start to tax the body. And so she might not have the amount of nutrients that she normally would have to feed the baby that she has. This is an evolutionary thing. The body wants mom to take care of the baby that she has. She doesn't want mom to get pregnant and therefore have to take care of that baby as well. So again, this is completely normal and natural. It can lead to some patho physiological things that were disgust. Of course, over here in males is the same thing that happens in males, high levels of prolactin in males are going to cause a decrease in LH and FSH. It's the same physiology in the males as it is in the females. It's just guys are breastfeeding anybody. But the effects of high prolactin are going to be the same. I want you to know lactational amenorrhea, I want you to know the you know, all the steps over here. How does too terribly complicated? And I want you to know, why are we good with this? Yes,

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actually, so my kids gave him was like, it starts out first with what my level four like and then it goes to a negative feedback right to the

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hypothalamus and the hypothalamus,

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or the GnRH. Lower the LH and as ah, correct. And then from there, that's where no ovulation or pregnancy or birth, correct? That's correct. Okay. Just double checking. I will just double

51:06

check. Yep, that's how go. Yep.

51:08

Yes. So, like you're having a diagnosis. How do you know which ones like causing the issue? Because you said that the low amounts of one of them will cause

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when you say one of them, one of them as in

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take you to go back to the other side with all the phones. Yeah, also, you said high amounts of prolactin will go back in, like, over Yep. So then, how do you know if it's prolactin or if

51:47

it's the LD like a GnRH problem itself? Yes, you do bloodwork? Oh, yeah, I used to do bloodwork. So if you're having these kind of issues, let's say a male has low testosterone levels, you know exactly why you mentioned the sandstone, you measure LH, you measure FSH. By the way, if you do blood work, you're not gonna be able to get any of these hormones, because they don't go into the general circulation. These hormones have these little blood supplies here that go directly in the hypothalamus to the anterior pituitary. Now, you can measure the level of these particular hormones, but it's expensive. Got to do MRI. To measure these, you're not going to do with blood work. That was a good question. So yeah, you would just simply do blood work and you would measure the level of prolactin, LH, FSH, testosterone, you'd measure all of them, and then come to a conclusion based on all that. Alright. Now, on to the next one growth hormone, this will be the last one we do tonight. So growth hormone. What time do we got here? 620. Right. Okay. So growth hormone let's talk about is functions. One of its main functions, the name tells you exactly what it is. Stimulation regulates the growth of tissue and repair that tissue as well. And then it does a number of other things. And again, this is not an exhaustive list, but it's the one that I'd like you to know. It increases your blood glucose levels via gluconeogenesis, the production of glucose from nine cards, if you remember from last semester, it increases your fatty acid levels in the blood via the breakdown of triglycerides. Remember what triglycerides are, I hope, and that's a process called lipolysis. Growth Hormone also stimulates the production of protein by telling cells take some amino acids from the blood, stick them together through transcription and translation. And let's make some protein. And so these are its

functions. Now, you know, what I'm gonna do here actually, is this, let's do this. This is going to be growth hormone level, right here. And we're going to talk about what stimulates growth hormone. And it's going to revolve around what the functions are. And so what we're going to do is take those functions, and we're going to combine them with what stimulates growth hormone. So I'm going to take that right there along with the functions and I'm going to draw this picture. Cells growth hormone is released. Most of the time level of growth hormone in the body in the blood is actually fairly low. There are times when its levels are going to go up, especially during deep sleep. I mentioned that actually, in the last lecture. If you recall it, I'm going to stick it in this picture. So what I'm going to do is this, so we have chronic release initially, and I'll actually put it there and it's a low amount. And then I'm going to have some stimulatory effects here. That's going to cause it to increase its release. So right there, I'm going to say that let's say that our blood glucose levels are low. Let's say you missed a meal or two or three, and you're hypoglycemic. sending notes, the word hypoglycemia. Good. Hypoglycemia simply means low blood glucose. Well, we already know growth hormone is a hormone that increases blood glucose levels. So of course, we would want to release more at this point, right. And that's exactly what we're gonna see. We're gonna see an increase in release of growth hormone. And we can start to get those blood glucose levels more normal level to come back down. What kind of release is that? That's a cute, what kind of regulation is that? nonhormonal glucose isn't a hormone, right. And it's regulating growth hormone. So it's not hormone, then I'll put over here high amino acids. Okay, so you just say a whole bunch of proteins, protein was broken down into amino acids, it got absorbed into the blood. And then you're gonna get a spike of growth hormone because the growth hormone is gonna say, hey, cells take the amino acid special protein out of that. And so up, we go again. And once again, that's acute release, then we go back to chronic, and then maybe right here, we have low fatty acid levels. And we know that growth hormone is something that causes an increase in fatty acids in the blood, because it breaks down fat, it burns, fat lipolysis, the breakdown of triglycerides, which is a storage form of fat. Once again, up we go. And once again, that's an acute release. And these three right here, they're all not hormones. So that's non hormonal regulation.

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Let's do a couple of others. So we're going to chronically release. And let's say that we decide to exercise when it comes to exercise, if you want to get growth hormone to be released, it's got to be vigorous. So you can't like you can't have acid when you're exercising to get a nice big ol spike of growth hormone. So rigorous exercise

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you get another spike out will be acute, once again. Now is exercise a hormone? No, but it's not non hormonal, non hormonal, typically mean some some molecule some chemical. So it's not non hormonal? It's certainly not hormonal. What do you think it is? It's neuro. So that will be neuro. I'll also put here because it's also neural stress. And when I talk about stress, again, physical mental, emotional stress doesn't matter, will cause a spike in growth hormone. And that's mediated by the nervous system, causing that spike. Then what I'm going to do here is chronic chronic chronic, and then we're going to go to sleep. And hopefully, we get enough sleep towards deep although was happens within about an hour or two, after you asleep. Look, what I'm going to do here, much bigger than the other ones, for the most part, for the average person. It's still what kind of release here is this acute. Already mentioned in the last lecture, this is actually cyclic. This is programmed. during deep sleep, your body is programmed to release a lot of growth hormone and for the average

individual, most people, that's when you're going to have the most growth hormone released during the day, which is why I made the release a lot bigger than the other ones. Also neuro. So growth hormone, and that's why you need to get enough sleep guys. When you sleep, you are repairing your body because of the rigors that you've gone through the day. Even if you've done nothing but lay down and watch Netflix, you still have to get some sleep because you have told the body not maybe as much as somebody who's a bit more active, but you still need your sleep. And if you don't get it, it will catch up to you at some point. So let's go back here. So we took care of all these, again functions. There's deep sleep Isobelle during deep sleep, low blood blood foods. Low blood glucose levels, high blood amino acid levels, low blood fatty acid levels, there's stress, there's exercise, it's all of them right there. I'm showing you what stimulates obviously because the levels are going up. And again, a few not horn models and a couple of neurons that are They're controlling growth hormone release. And then of course, ghrh. We already knew that one. And I'll just point it out to you again, here's ghrh growth hormone releasing hormone stimulates growth hormone growth hormone inhibiting hormone inhibits growth hormone. High blood glucose levels hyperglycemia. Of course, that's going to inhibit growth hormone. Because it's already high to begin with. If you don't have as many amino acids in the blood, you're not going to stimulate as much growth hormone because well, there's not as many amino acids in the blood to tell cells taken let's make some protein.

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Your fatty acid levels are already high, you're not going to stimulate a hormone that causes your fatty acid levels to be even higher, you're just not going to. It's just simple, negative feedback. The lowest levels of growth hormone are shortly after you wake. This is something else that's very important. By the way, I'm going to show you a number of hormones that are that are released in a cyclic manner. growth hormones, one of them, cortisol is going to be one ACTH. And such, it's important for you to know that the same thing with the menstrual cycle hormones during the month, when you take your patient's blood and you're evaluating them, you have to know when these hormones are high or low. Because if you take it a certain part of the day, and it's like Jesus would call Hi, this is well it was at this time of the day. Or Look how high Lh is, well, it was at the middle of the month, and it's a female, and it's supposed to surge at the middle of the month. So it's important to know, cyclic hormones and when they're high, and when they're low. When you do bloodwork and interpret that blood work, it's going to be very, very important. Not that you're probably going to take growth hormone levels, you know, you're not going to stick somebody whether deep sleep, probably not. But certainly, you would know that the levels of growth hormone are going to be pretty low. And typically a lot of blood work is done in the morning, because people that the fast a lot of times with blood work a good 12 hours. And it's just easier to just go in the morning before you eat breakfast and get your blood work. So again, these are things that are very, very important to know. So anyway, that's the physiology of growth hormone. Now let's talk about some patho fits the effects of hypersecretion and growth hormone. Abnormal lengthening and thickening of bones. I don't know if you recall that last semester when we did the skeletal system. And I mentioned a few hormones that affect bone growth both in length and width growth hormone was one of them. When we talk about growth in length, the only people that grow longer and taller are people who still have a growth plate, no one in this room still has a growth plate. So if you had a lot of growth hormone release in your body, let's say right now, from this point forward, you're not going to get taller. Because you don't have a growth plate anymore. There's no way for your bones to get any longer. But your bones can get thicker. That can happen till you're a gazillion years old. So that's the thickening of the bones. But certainly abnormal lengthening of the bones is going to occur if you're young enough, during let's say, adolescence in your teenage years. And what's going to happen then is that you're going to be very, very tall, abnormally tall. Now it doesn't mean if you're really really tall, doesn't mean that you necessarily had a growth hormone issue. Like you know, professional basketball players average height is like 6768, Shaquille O'Neal

over seven foot tall, for example, he didn't have a growth hormone problem, he just was genetically gifted from his parents, his parents genes allowed him to be very, very tall. But there are some people who again have growth hormone problems that would not have been anywhere near as tall as they were had they not had the growth hormone problem. And I'm actually going to show you a picture of somebody who you're probably familiar with, what else is going to happen with hypersecretion of growth hormone, abnormal organ growth, enlarged heart, enlarged kidneys, enlarged liver. And some people might think to themselves what an enlarged heart, that's great, you can pump more blood and large liver, you're going to be more efficient in doing the two to three dozen functions of the liver, where your kidneys gonna be able to filter your blood better, no organs need blood supply, and that blood is giving oxygen and nutrients to survive. If your organs are too big, you're not gonna have enough blood to get to the organs, and they're going to die. So can lead to organ failure. So it's not a good thing at all. elevated blood fatty acid levels, of course. Because growth hormone increases your fatty acid levels, we already know that again, if you know the function of a hormone, you're gonna know what stimulates it, you're gonna know what inhibits it, you're gonna know what happens when there's too much of it, or too little of it, given the function when you study these hormones, that should be the center of your studies is function. If I told you to just study one thing, it would be function. Not don't just study one thing. But if you had a choice of studying just one thing, it would be function. Everything else falls into place after that. So increased blood fatty acid levels can lead to cardiovascular disease. Increased levels of blood glucose can lead to type two diabetes and it's diabetes mellitus, not insipid. It's the one that we talked about with ADHS mellitus when it has to do with blood glucose. None of these things are good.

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often caused by tumor, the pituitary gland hypersecretion, a growth hormone. If it's happening while the child is growing, they will develop a condition called gigantism or giantism. They will just be really, really tall. If it starts into adulthood, or it progresses into adulthood from childhood, they will develop a condition called Acral megali. I'm going to show you a very famous person who had acromegaly, Andre the Giant. I have a picture of him at three different stages in his life. Here he's about 18 years old, and he's already over seven feet tall. So he had pituitary gigantism. Then this is probably him in his maybe middle to late 20s. And this is him in his late 30s. I think maybe very, very early 40s. If you look at him, it looks fairly when it comes to his facial features especially. Does it look I don't want to say abnormal. But if you look at him here, look at his jawline. It's getting much much thicker. His forehead is starting to protrude more. definitely hear jawline, definitely there, his fingers were just thick as hell. All of his bones were they take on this physical appearance. And what's happening here is is that the bones just continue to grow, to protruding head forehead that just more and more bone growth. So he's getting a thickening of the bones. Andre the Giant suffered from cardiovascular disease under the giant suffered from type two diabetes. Andre the Giant died in his middle to late 40s, which is actually kind of long time for somebody with acromegaly. He was actually an alcoholic as well. He knew he was going to die. And he just kind of live life to the fullest. I guess he was this gentle, gentle, gentle, beautiful person. You guys ever seen Princess Bride? Whoever hasn't seen Princess Bride it was made before you guys were born. But it was he was the giant in The Princess Bride. It's a freaking awesome movie. I just actually watched it again. I don't know just just recently actually. He's famous for being a professional wrestler, Andre the Giant back in the 80s. So anyway, he had this particular condition and typically people and there's no treatment for it. By the way, there's no treatment. Unfortunately, we're just not smart enough to figure out how to treat it yet. Even though it's a benign tumor, there's no treatment for it. So now let's go the other direction. And let's talk about hypo secretion and a condition called pituitary dwarfism. So a pituitary dwarfism. It's going to hurt to happen during childhood. And the person is going to be proportional but short in stature. What does that mean? Well, I'm going to show you a couple of pictures of this. So here that's

not a doll. Even though it looks like a doll. That's actually a person. She's probably no more than 18 to maybe 24 inches tall. But if you and she's an adult, if you look at her, she's proportionately small. It's like somebody like shot somebody with a like a little shrink ray, and everything got proportionately smaller. Over here, this dude is not seven feet tall. He's about probably five, eight or five, nine. These guys are just really, really short. They have pituitary dwarfism, but they're proportionately small. their arms, their legs, their head, their torso, it's all within proportions. You know, that is. Slide on Messi considered the best soccer player in the world has been for a number of years him and Ronaldo. He had pituitary dwarfism. And so what happened with him is he's born in Argentina. And they were playing soccer at this big tournament in Argentina. And there was a team from Spain there, Barcelona team. And they saw him playing. And he was really, really good. And they approached the parents. They want to know how old he is. And they were and he was much older than what they thought. So well. He's very, very small for being that old. The parents said, Well, he has pituitary dwarfism. And the Barcelona team said you know what? If you give us your son, what not give us your son. We will take your if we let Him belong to our Spanish team. We will pay for the treatment. And what's the treatment for pituitary dwarfism? It's easy. You just give Human Growth Hormone you give HGH you have to give it to them at a certain age or it's not going to work. So you have to ask yourself, why didn't she get it? Why did these guys get it?

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It costs a lot of money. You're looking at it at least 1000 bucks a month for human growth hormone. We can get growth hormone from pigs, or cows. But it doesn't work in humans. It does not stimulate the growth hormone receptor in humans. It has to be human growth hormone. And there's not a whole lot of human growth hormone out there. That's why it's so expensive. demand is high. The supply is low. It's just plain old economics. So he was lucky now. I think he was about 10, maybe 11 years old when he started. Had he started earlier. He'd be taller than he is. Now. He started tall do I think Messi is what 5556 He's kind of a little guy. He served done well for himself. What does he make like $100 million a year. I mean, he's ridiculously rich and famous. And he's a tremendous athlete. But had that Barcelona team not seen him. He would have never been Lionel Messi. He would not be a multi multi multi multi, probably close to a stick and billionaire by now. But he was lucky. And that's all it's gonna take is just taking the hormone at a certain age that will allow you to grow otherwise, you are not going to grow. Now, when it comes to pituitary dwarfism. Most people who have dwarfism do not have pituitary dwarfism. Most people who have dwarfism have what's called a condor, plastic dwarfism. And I'll give you a couple of pictures of that. You guys watch Game of Thrones? That's Tyrian. Okay, that's the man from the Jackass movies. They have what's called a condor, plastic dwarfism. They don't have any problem with growth hormone. Their growth hormone levels are the same as anybody else who doesn't have a pituitary problem. The problem with the Congo plastic dwarfism is a growth plate problem. If you look at their heads, they're the same size as a grown adult. What's different is the length of their lens, their fingers, they're much shorter and they're kind of deformed to because they have a growth plate problem. It's a genetic condition that they're just they're born with it. So I want you to know that I want you to know that a kind of plastic dwarfism growth hormone levels are completely fine. With pituitary dwarfism. The levels are very, very low, which is why they look so different. pituitary dwarfism? They're very proportional. A Condor plastic. They're not proportion. Not at all. Alright, what else do I have for you? Nothing. We're done. Have a nice long weekend. And I will see you guys Tuesday evening.

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SUMMARY KEYWORDS

thyroid gland, tsh, hormones, calcium levels, hypothyroidism, release, acth, hyperthyroidism, adrenal cortex, produce, calcium, levels, stimulate, picture, gland, patient, goiter, iodine, called, metabolic rate

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Alright folks, here we go. Lots to talk about today. So let's remind ourselves what we're doing last Wednesday, we were talking about anterior pituitary hormones, we made our way all the way to growth hormone got through growth hormone, which means we just have these two right here, what I'm going to do is I'm going to talk about these two pretty much at the same time and draw one picture. And that picture is going to involve the hypothalamus. And these two hormones, these two releasing hormones. And then these two hormones, of course, and then the glands that these two hormones are going to regulate. So let's do that. So let's draw the hypothalamus. So you always do, it's going to be a rectangle. And then we're going to have the two hormones that are involved in a story, release in the hypothalamus, one of which is TRH. The other is CRH. And then they're, of course going to be released into those little teeny blood vessels that go directly from the hypothalamus, to the anterior pituitary, which is what this is. And I'll abbreviate it. And so these hormones are going to stimulate the anterior pituitary to then release TSH, thyroid stimulating hormone and ACTH adrenal cortical Tropen hormone now, and you know what, let's throw in as well, the thyroid gland and the adrenal gland. So this is going to be the thyroid gland, which is the target of TSH. And then the thyroid gland is going to release a couple of hormones T three and T four. I'll tell you what that abbreviation means what those abbreviations mean when we get to the thyroid gland, and we're going to do that shortly. And then we're going to have here the adrenal gland, but specifically the adrenal cortex. As you well know, the adrenal gland has a adrenal medulla and an adrenal cortex, you learned about that. We're going to talk a little bit about the adrenal medulla today, but right now, let's talk about the hormones that the adrenal cortex is going to release. It's going to be three different kinds. One minerale Oh, coracoid quarter chords, as you say, there's more than one glucocorticoids. And androgens. So those are three different kinds of hormones at the adrenal cortex is going to release that's under the control of ACTH. Now, what do I want to say about these hormones? TSH, ACTH. Let's just go to the notes. We'll just highlight a couple of things. So TSH, first one, okay, what stimulates it? Well, we just see TRH lhote thyroxin. By the way, that's t for Toyota, thyroid is T three, low levels of those two hormones that will make more sense when we get to the thyroid gland. What inhibits TSH? Well, of course, if you decrease TRH, you're going to inhibit it. Hi, t 43. Again, it will make more sense when we get to the thyroid gland stress. That does not mean that TSH is a stress hormone. Stress hormones are those hormones that are stimulated by stress. So stress causes a hormone to be released. It's considered a stress hormone. It stress inhibits the release of a hormone. It's not a stress hormone. It's not contributing to the stress response. And TSH does not. So I just want to make that clear. What are the effects of TSH hypersecretion and hypo secretion we're going to get into that when we get to the thyroid gland, so not a whole lot on TSH right now, a teeny bit more on ACTH. So what stimulates it? Of course, CRH does inflammation and stress stimulates it stressed us.

So ACTH is a stress hormone. Because it is released in response to stress and it's going to help us deal with the stress. We're going to talk more about that when we get to the adrenal gland, the adrenal cortex, low levels of glucose, we're going to talk about it when we get to the adrenal gland. This is something I want to highlight those sleep wake transition. highest levels of ACTH are right before you wake up the lowest levels of ACTH are during deep sleep. Does that sound familiar? Sounds a little bit like growth hormone except it's the complete opposite, right? The highest level of growth hormone is during when during sleep. lowest levels of growth hormone are when you wake up. This is still a cyclic hormone. It's just released in the complete opposite way that growth hormone is so I just wanted to highlight that to you.

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We'll talk a little bit more about that when we get to especially cortisol. Cortisol is a hormone that's a glucocorticoid. By the way, cortisol is aldosterone is a minerale cortical DHEA is the main androgen. And those are these three right here. So these three right here that we see, we'll get to them in detail when we get to the adrenal cortex. So again, ACTH is a hormone released in a cyclic way. And again, I'll explain why that is when we get to the adrenal cortex. There's one other thing I want to highlight with ACTH. And that's this. Not only does ACTA stimulate the adrenal cortex, but it's also going to stimulate melanocytes. That's in your notes. melanocytes are what kind of cells what do they do? What's their job to produce what melanin and they're in the skin, right. And so these melanocytes are going to be stimulated by ACTH, which means ACTH, when its levels go up, can darken your skin that's going to come into play during certain diseases that we're going to be talking about, it's going to help you diagnose certain diseases based on what happens to skin color. We'll see it when we get it get to it. And we're going to get to when we get to the adrenal gland, which isn't going to happen today. We're going to have to wait till Thursday to get to the adrenal cortex, we'll get to the adrenal medulla today, but not the adrenal cortex. That's really all I want to say about those two particular hormones right now, more on them later. So then let's move on. Let's talk about the thyroid gland. And by the way, looking at this picture, before we move forward, what kind of regulation do we have going on here? That's hormonal regulation all over the place TRH. Regulating TSH was regulate T three T four CRH, regulate ACTH, which regulates all these three types of hormones from the adrenal gland, a lot of hormonal regulation here. And we're going to continue to see hormonal regulation when we get into the thyroid gland. So let's talk about the thyroid gland. Now, we're going to start by talking about the structure of T three and T four. Now, up into this point, I haven't talked about the structure of any hormone. But I'm going to talk about the structure of these two particular hormones. And there's a reason for it. And you'll see, so when I draw T three and T four, what I do is, is I draw a box. So that's a certain ingredient to T four, and this is going to be t for The I stands for iodine, you need iodine to produce pyroxene, which is T four that you can see now why they call it T four, because it has four iodine molecules as part of its structure. And then T three is only going to have three iodides. As part of its structure, I'm still going to draw the box, the box is exactly the same ingredients for T four and T three the same exact molecules. The only difference between these two molecules, these two hormones is the number of iodine that they contain. This has a longer name, try i o dough. sigh Roni, otherwise known as T three, now have these two hormones T four is the one that the thyroid makes the most up about 90% is T four that the thyroid gland produces, which means that only about 10% is T three. But T three is kind of the big dog between these two, T three is approximately five times more potent, it's more biologically active, that's the one that's really going to be doing the work. That's the one that when it binds to the to the target cells, is really going to be telling those cells what to do. Now, something else that's important, these hormones are stored. So both T three and T four are stored in the thyroid gland. That is not common at all. All the other hormones that we've discussed up into this point, the hormones that we're going to discuss beyond this point, are produced, released, produced release produced release. Now these hormones are also

produced and released, but they're also stored in the gland itself. And we're looking at about three months worth of these particular hormones. So about three months worth What's that mean? That means right now, if everybody in this room stopped producing T three and T four, you wouldn't even know it for two to three months, because the thyroid gland would have enough to release and they'd be able to do their thing for about three months. Then at about three months sometime in March, you start to have symptoms of low T three and T four, which we will talk about. This is also going to come into play with some other things that we're going to be talking about shortly. What else

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most of the T three. And actually, before we go on, you might ask yourself, well, if this is the one that's doing most of the work, why the hell doesn't the thyroid gland produce more T three and T four this is the Most of the T three that's circulating in your body. This is how it gets formed. So this is T four again. And what's going to happen is a chemical reaction and a chemical reaction is called D, io, na zation, D D E means from in Latin, we're going to take an iodine from T four. And when you take an iodine away from T four, you are left with t three. So that's how most T three exists in the body.

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Now, where does this happen? This happens in the thyroid gland, but every other tissue in the body pretty much as well. So this occurs in the thyroid, not a whole lot of it. And most tissues of the body. So when t four gets released, the thyroid gland could have already turned it into T three, but once it gets to the tissues, whatever tissues those are, and it's pretty much most tissues in the body, the tissues themselves will take the T three or T four and make T three. And now we have a very biologically active form of these hormones that can do the job that they need to do. And what are those jobs. So now let's look at the functions of t four and T three, again, T three is going to be the one that's going to be doing most of the work here. And so let's look at function. So we just drew all this stuff here. Now let's talk about functions, regulates metabolism, your metabolic rate is in large part due to the levels of T three and T four. What else helps regulate your body temperature? We talked about body temperature last semester, we need to maintain it right around 98.6 degrees Fahrenheit, we talked about a number of ways that that occurs. Well, I didn't mention T three and T four last semester, but they're part of the story. Now how would they do that while they're regulating metabolic rate. During metabolism, what is a waste product and I say waste in quotes that is produced as a result of aerobic or anaerobic sorry, the respiration starts with an H. E. A. T heat, right? So when you control metabolic rate, you control the heat that's produced in the body. And if you control the heat that's produced in the body, you can control the temperature of the body. So T three and T four are going to help regulate body temperature by dictating what your metabolic rate is, what else helps produce protein. What else increases the glucose levels? Those two things right? There sound like something that growth hormone does, right? Growth Hormone facilitates protein production growth hormone increases blood glucose levels. That's something I want you to do. As we go through these hormones, as you go through these hormones, I want you to look at those hormones that do the same thing. And there's going to be many that do the same thing. There's a lot of redundancy in the body by the hormones work together. What else decrease cholesterol levels, that's a good thing. Again, these are not all of the functions of t three and T four. But they're the ones I'd like you to know. What's gonna stimulate T three and T four. We already know TSH does, we just drew it. First picture we do TSH stimulates T three and T four. What else? Well, if you have more of the one of the ingredients, you're going to use the ingredient the thyroid gland is going to use that ingredient to make more tea for especially in some T three low glucose levels. Yeah, of course. If this is normal

glucose, and your glucose levels are down here, what do you need to do? Negative feedback says you got to raise it is this a hormone that raises glucose. Of course, it's just playing on negative feedback. If your glucose levels are low, release more of a hormone that causes more glucose to be produced. Cold today, you go outside it's not very warm today. I guarantee you as soon as you walk outside, especially if you're not dressed warmly. You're going to release more T three and T four why need to raise your body temperature, you will increase your metabolic rate. So cold will stimulate T three and T four release again. It's just plain old negative feedback. And then what inhibits T three and T four coasts low level of TSH less iodine, high levels of glucose stress inhibits it via TSA. So if TSH is inhibited by stress, T three and T four is going to be inhibited. So our T three and T four stress hormones yes or no No, because they're inhibited by stress. So they are not going to contribute to the stress response. And when it comes to this stuff, increased blood iodine, decreased blood iodine, that would be what? Non hormonal regulation, right? Is iodine, a hormone, no glucose levels. That would be non hormone regulation, right. TSH would be hormonal regulation. Right? The Cold Response is neural, by the way. So it's regulated in all kinds of different ways, all the three ways that we discussed

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and acutely low glucose levels, that's not a programmed thing within the body, that would stimulate its release, or high glucose levels would inhibit its release, that would be acute regulation. So again, those things that we talked about in that first chapter, don't forget about them, keep on applying them to the things that we talked about in this chapter. And I'll keep on reminding you. So that's all the physiology. Now let's talk about the pathophysiology of the thyroid gland. What happens with hyper thyroidism? Too much T three and T four being produced? Well, what kind of things? Are we going to see? Excess sweating? Does that make sense? What's gonna happen if you have too much T three and T four, a higher metabolic rate, which means you're going to produce more white heat, which means your body temperature is going to go up. But what's one of the ways that you're cool your body temperature? You sweat. So you would sweat more? It possibly doesn't mean it's going to happen. It depends on the person. What else he didn't tolerance? Well, you're hot, T three and T four levels are high, your metabolic rate is high. So if it's hot outside, you're just going to feel even more miserable, you won't be able to tolerate the heat because your body temperature is already high. That's what that means. Unexplained Weight Loss. Why do you think you'd have unexplained weight loss? I don't have the word unexplained there. I'll explain what unexplained means and use a second why would you lose weight? Yeah, higher metabolic rate. But it would be unexplained what it means what it means you're losing weight, not trying. You go to the doc, they go to you one day. It's like I lost 20 pounds this month, I didn't even try. I didn't eat last I didn't exercise more. But I lost weight. Well, why? Because the metabolic rate was higher. Increased heart rate. So if you took their pulse pulse as a measure heart rate, it would be high, it would be elevated. Ventilation is all about breathing. They'd be fidgety, they'd have nervous as you sit there, you'd be evaluating them, and they wouldn't be able to stop moving their leg, for example. So that could be an indication of hyperthyroidism. The nervous system, metabolic rate is higher, more action potentials, they could actually be anxious. Sometimes when people are diagnosed with anxiety, it's actually hyperthyroidism. And so for a complete evaluation of somebody, you want to look at TSH levels, T three T four levels, because that could be the problem. Diarrhea, you've increased the metabolic rate within the digestive system, things just move through a bit too quickly. Except Thalamus, what's that protruding of the eyes, I'm actually going to show you a picture that she's not doing that on purpose. So it happens with hyperthyroidism, and it's the most common cause of what we see here is that pressure builds up behind the eyes and the eyes are literally just kind of being pushed out of the head. What you see to the right is her after treatment. And we will talk about treatments for hyperthyroidism. You can also have what's called a goiter. Tell me what your thyroid gland is, please.

Right here. And so a goiter is an enlarged thyroid gland. Now why would the thyroid gland be enlarged with hyperthyroidism? Well, let's think about that for a second. If you're making more T three and T four

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do you think you're going to store more T three and T four? Yes. And when you store more T three and T four what's going to happen your thyroid gland it's going to get bigger. That's what a goiter is an enlarged thyroid glands now look at the picture. So what we see here is I would say this is mild to moderate goiter right here. You go on the internet, you type in goiter, it'll look like there's like a softball, basketball sometimes in their neck. It's so severe. And what we see here on on the right hand side is well it's gone. Why? Because she got treatment. So something to look for when it comes to evaluating a patient. So that's hyperthyroidism. Now what are the causes of hyperthyroidism? I have a few of them here. One of them is Graves disease. One of them is a pituitary tumor and another one's a thyroid tumor. And so what we're going to do here is we're going to have patient a patient B patients C, and we're going to do our blood work. We're going to evaluate this patient based on signs and symptoms that we see as we evaluate them. And blood work. And that blood work who's going to be blood work done on the level of TSH, the level of T three and T four, and something else that I'm going to tell you about. So let's do that. Now let's do some clinical stuff. So patient, a patient being patient, or patient, A, B, and C. So patient a, a, say you're just got on your shift, it's seven in the morning. And patient a is on the books, and you see patient a, it's 730 in the morning, and you're evaluating patient a. Then after lunch, you see patient B, and around 415 430. Before you get to leave at seven o'clock, you see patients C. And patients A, B and C look exactly the same as you're evaluating. And so what kind of things might you see? Well, you're evaluating patient A, B, and C, and they tell you, you know what, Doc, or PA, I've lost a lot of weight over the last couple of months. And I sure as heck didn't try. And you look at them, and they're very fidgety. They just can't sit still. You do vitals on them. And they have a high pulse. And you notice, maybe they have a goiter. And they tell you know what, I'm sweating a little bit more lately. So they're sweating. So you look at all these things, and you suspect hyperthyroidism. Again, every single one of these patients has all of those. And so you suspect hyperthyroidism, you're probably right. But you have to confirm it, because it could be other things as well. And so how are you going to confirm it, you're going to do some tests, one of the tests you're going to do is blood work. So you're going to take the blood from the patient, you're going to send it to the lab, you're going to say lab give me the levels of TSH. And T for typically is T four, you don't do T three, but we'll put them together in this discussion. And you're also going to tell the lab to measure something else that I'm going to tell you about in just a little bit, but not yet. So now what we're going to do is we're going to have patient A, B and C and we're just going to put them in different columns. So we're going to put the anterior pituitary in the picture and the thyroid gland and the picture where to go back to the first chapter, when we talked about hormonal regulation, hypersecretion hypo secretion, and how you can tell if it's this gland or that gland, we're going to do that now. So patient a,

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patient B, and then patient C. Again, they look exactly the same. So now let's put the anterior pituitary in the picture. And here it is, we'll label it of course.

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And we know that the anterior pituitary is going to be releasing TSH. And it's going to release it on to the thyroid gland, which we will put here. We'll just make it a box, and we're going to be releasing T three and T four, I'm gonna make that arrow just a little shorter, give myself a teeny bit more room on the bottom. That's just something that we have to write down there, you'll see what so here's our thyroid gland again, releasing T three and T four. And we're going to draw the same thing in patient B and C as well. And so you are going to ask the lab once again for tcsh MT four you have to ask for TSH because it's regulating the thyroid gland. If you don't ask for TSH along with T four, you're not going to be able to come to a good conclusion as to what's happening with your patient. You won't be able to diagnose them properly. And again, I said that there's going to be one additional thing that you're going to ask the lab to look for. I'm just not going to tell you what it is yet. The suspense is killing you, I'm sure. Okay, so you do the blood work. And lo and behold, in patient a and in patient B, the levels of T three and T four Hi boom, you got it right. It's hyperthyroidism by definition high T three and T four is hyperthyroidism. You got it right. Based on your evaluation of the patient good for you. Now you tell me the high levels of T three and T four should feed back onto the anterior pituitary and do what to it stimulated or inhibited to bring the level of T three and T four down. What should it do? We should inhibit Correct. Negative feedback tells us we need to inhibit the anterior pituitary because the levels of T three and T four are high. So let's what should happen but I'm going to say that in patient a when you get the blood work back you're gonna see the TSH levels Actually let's just do in this direction are actually high. Is that the way the anterior pituitary should be behaving? Yes or No? No. It should have been inhibited, correct. The levels should be low. Where's our problem? pituitary is the problem. Is the thyroid gland the problem here? Is it doing what it's told? Absolutely it is. It's responding properly. It doesn't know any better. So what do you think we have here the pituitary, probably a tumor, probably. pituitary tumor caused him to to try to release too much TSH, causing the thyroid gland to release too much T three and T four. Patient B. Once again, as you told me, We should be inhibiting the anterior pituitary and the patient B. That's exactly what we see. Can we know that that's the case because TSH levels are low. So the pituitary gland in this particular case is behaving perfectly. What's not behaving perfectly thyroid gland? Probably cancer. So the tumor is probably there not probably it is there. What other option can we have here? What other differential diagnoses can we have? Looks like we have both of the scenarios. There's actually a third when it comes to the thyroid gland. And this is it. We're supposed to inhibit and that's exactly what's gonna happen. So then what Dr. R is going to be the difference between patients being impatient. See, I'm going to tell you, patients C has what's called Graves disease. Graves disease is where your immune system gets stupid. And in this particular case, with Graves disease, your immune system is going to produce a molecule that looks exactly like TSH. So with Graves disease, what's going to happen is is that the immune system produces T S H, what are called Auto antibodies. Auto antibodies couldn't be in the news anymore. If we tried lately over the last two years with COVID antibodies are proteins produced by your immune system. Normally antibodies are supposed to kill pathogens, like the COVID virus or Coronavirus, I should say, in this particular case, these antibodies are not going to do that. What these antibodies are going to do

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is stimulate the thyroid gland. So, in this particular case, there is absolutely nothing wrong with the anterior pituitary and there's nothing wrong with the thyroid gland. The problem is the immune system. The thyroid gland is doing exactly what it's told. And so to the anterior pituitary, they're behaving properly. So why don't you tell me the TSH auto body's auto antibodies I should say are what to the TSH receptors starts within a agonists. So we have TSH agonists. Those animals those auto antibodies bind to the TSH receptors just like TSH does. Now why does the immune system do this? beat the hell out of us. We don't know. If we did. Graves disease wouldn't exist anymore, we'd

stop it. But unfortunately, this is what happens. Now, that's the thing that I didn't tell you that you're going to tell the lab to measure. If you suspect hyperthyroidism, you will ask the lab for TSH levels, you will ask the lab for T four levels and we'll throw T three in there as well. And you will ask for TSH auto antibodies, how do you know they're there? The lab will tell you because the lab has an assay to measure those. The only time you're going to see it is with Graves disease. Now, Graves disease is actually the most common form of hyperthyroidism of three Graves disease is the one that you're going to see most often. It's not going to be a thyroid tumor or a pituitary tumor. They do happen. But Graves disease much more prevalent than patients say NP. Now, something else we're going to talk about now. And we're going to go back to our first lecture. primary condition, secondary condition. I'm not sure what that is. It's up on the screen. So I'm just going to kind of read you the definition and then we're going to compare pages A B and C. Like I'm supplying Marion secondary condition. So let's look at the definition of primary. This is the very last page of the first chapter. If a hormone is causing signs and symptoms and so we're just going to talk specifically about T three and T four right now. If a hormone is causing signs and symptoms? Well, what signs and symptoms? Well, let's talk about hypothyroidism. If the hormone T three and T four are causing signs and symptoms if you suspect that hormone causing the signs and symptoms, and let's go back to the definition. And there is something wrong with the gland that is producing that hormone, what's the gland that produces T three and T four, the thyroid gland, it is a primary condition. Now let's go the secondary condition. If they hormone in this case, T three and T four is causing signs and symptoms, and there's nothing wrong with the gland that is producing that hormone. But instead, something else is causing that gland in this case, the thyroid gland to be dysfunctional. It is a secondary condition. So now let's go to patients A, B and C. Patient a primary or secondary condition. Primary is Is there anything wrong with the thyroid gland? And patient? Nope. Is something causing the thyroid gland to produce too much? T three and T four? Yes. And what is that? The pituitary. So this is what is a secondary condition. So patient a has a secondary condition, patient B primary or secondary? Primary, because there's something wrong with the thyroid gland. So this primary Well, where does that leave us a patient see? What is it? Is there anything wrong with the thyroid gland? Patient see? Nope. Is something telling the thyroid gland to produce too much d3 nt four? Yep, it's not the anterior pituitary. But it's the autoantibodies. So patients see is also in secondary Graves disease is a secondary, hyper thyroidism. That's why I saved those two definitions for when we talked about something specific because I think it just helps it make sense. Right off the bat. Are we good with this? Yes. All right. Now what? Now? Let's go the other direction. Yes, sir. See that one more time? Patient a yes. Okay.

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primary condition pituitary gland at the same time is secondary.

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Not really. And the reason is, is that the high levels of TSH are not causing those signs and symptoms right there. Those signs and symptoms are caused directly by the high levels of T three and T four. So as I levels T three and T four, they're binding to the receptors, they're overstimulating these particular tissues of the body, causing these particular again, things that we see so yeah, so yeah, so it's primary, the thyroid nuts are secondary. Yeah. Now what? Let's go the other direction. Let's talk about hypothyroidism to little secretion of T three and T four. What kind of things will we see here? Well, now because your metabolic rate is going to be low, you're not going to produce as much heat in the body. So on a day like today, you'll be way miserable. You'll have cold intolerance cold is really going to bug you. Because it's going to be harder for you to raise your body temperature because

your metabolic rate is low because T three and T four levels are low, dry skin and hair. What the hell is that got to do with anything? Guys remember what sebum is, what sebum? Is the oil produced by the sebaceous glands which are in your skin, right? If you don't produce as much sebum your skin will be dry. Well, why the hell would you not produce as my sebum because you have low T three and T four and it decreases the metabolic rate of the sebaceous glands. You don't produce as much SIBO. So dry skin dry hair could be a sign of hypo thyroid. Now, it can happen for 18 million other reasons. You don't just look at somebody Oh, you have dry skin and dry here. You must have hypothyroidism. No. You have to look at other things as well. Like what high levels of cholesterol? Why the hell did that happen? Well, what does T three T four do? decreases your cholesterol levels. If you don't have this high level of T three and T four, you're not going to decrease them as much. And your cholesterol levels go up possible. When you look at the signs and symptoms under hyperthyroidism and hypothyroidism, it doesn't mean you have to have every single one of these, you might only have three or four and a patient. One might have a goiter, another one might might not. It depends on the person. So I want to make that completely clear. What else can happen. low heart rate, you're decreasing the metabolic rate of the heart. So the pulse would be low. Ventilation has to do with breathing. lethargy, just means you can't feel blah you might even feel kind of depressed. And so again, anxiety, overstimulation, the levels of T three and T four. He just kind of feel blah. So again, you might not have clinical depression, you might have hypothyroidism not as common but it can still happen. You're not going to have diarrhea could be the other direction you have constipation. Look at this one. This one might surprise you, a goiter with hypothyroidism, a goiter with hyperthyroidism. That should probably make sense, right? You make more T three and T four, you're gonna store more T three and T four. Why the hell would your goiter get bigger with hypothyroidism? I'll explain it to you in a little bit. Not yet. What would cause hypothyroidism levels A T three and T four a primary condition or a secondary condition. And so let's jot these down. Let's do patients D and E for this one, to be thorough. So now we have patient D and E. And we'll go back to the specific causes. Again, you see these two patients separately, but they're going to look exactly the same. You're going to have a low pulse. This person might feel lethargic, they just say, You know what, I just I just fit blah. I used to work, I don't work out anymore. I don't feel like really doing a whole lot. They might tell you they're constipated. Another one unexplained, I don't think I have it in your notes. Not weight loss. But what weight gain? They don't need anymore. They're not exercising less. And yet they're gaining weight. Why? Because their metabolic rate is low. So that could be something else that occurs. They could have a goiter. Okay, so again, we can go down the list and you know, put a whole bunch of these different things here. Let's just throw cholesterol in there. So they have high cholesterol higher than it was the last time that they were evaluated. So you look at these things and you suspect hypothyroidism. You might be right, you might be wrong. How do you confirm it, you do the blood work. And so you get the levels of TSH levels of T three and T four. And one other thing that I'm going to mention. So here's patient D.

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And here's patient E. anterior pituitary, of course. And the thyroid gland making T three and T four. Same thing here. Oops, did that wrong? There we go. Here's our TSH. Here's our TSH thyroid gland

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making T three and T four. And of course, because you're so smart, you got it right low levels T three and T four boom you nailed it hypothyroidism by definition. What should that do to the pituitary stimulator inhibit should stimulate. We're trying to raise T three and T four and TSH is a hormone that

can do it for us. So let's stimulate the anterior pituitary. But lo and behold, it's not what's going to happen. Level of TSH is low should be high. So obviously our problem is once again at the pituitary gland is that a problem with the thyroid gland? What could this be this could also be a tumor cancer does not always cause an increase in secretion, it can cause a decrease in secretion it depends on the cancer depends on what it's doing to the gland this cancer could be destroying the gland whereas the other one just causing the gland to pump out a whole bunch of TSH what one this one. So cancer can cause either a decrease or an increase. So we have a tumor over here. Now over here, we're gonna say that do Terry's listening. TSH levels are going up. The Pituitary is behaving properly. It's the thyroid gland is not behaving properly. Now, what could this be? It could actually be cancer, but most often it's not. I'm going to give you the most prevalent hypothyroidism. out there. It's called Hashimotos disease. Same doctor, the person that discovered it. This is also autoimmune. But unlike Graves disease, the antibodies that are produced here are antibodies that destroy these antibodies destroy the thyroid gland. So autoimmune destruction of the thyroid and if you destroy the thyroid, well, it can't make as much T three and T four. So patient D primary or secondary condition and secondary Hashimotos disease. Its primary because there's something wrong with the thyroid gland. What's wrong with it? It got eaten by your immune system. All right. Let's take a break. When we come back, we'll continue our story with hypothyroidism. We'll throw a goiter in here again with hypothyroidism and move on from there. Okay, folks, let's continue our story. And so what is our story? So right now we're talking about hypothyroidism What am I missing here? There we go. All right. Alright, so now we're good hypothyroidism. Okay, so let's talk about some causes here. So with the Primary Hypothyroidism, inability of the thyroid gland to produce T three and T four, that would make sense. There's something wrong with the thyroid gland. Now, why would it have the ability to make T three and T four? Well, one thing is that could have been born like that. Some congenital is what congenital means some congenital condition where the thyroid gland just doesn't have the ability to make T three and T four, you could have an iodine deficiency, you need iodine to make T three and T four could lead to a condition called cretinism cretinism, you're going to have mental retardation physical retardation of the child. And then of course, Hashimoto disease is one that I mentioned, because that's the most prevalent when it comes to hypothyroidism. And then, of course, secondary hypothyroidism, a decreased level of TSH. Now, you could have a decreased level of TRH. Let's go back to our first picture here. The level of TRH could be low, which would then cause a low level of TSH. If you have a hypothalamus problem here, it would actually be called a tertiary condition or a secondary condition. Why didn't I get into that? Because I just don't want it. When it comes to measuring the the releasing and the inhibiting hormones of the hypothalamus, those hormones are not released into the general circulation, you just released those little blood vessels that go through the hypothalamus to the pituitary. So you do bloodwork, you're not going to measure those got to do MRIs. It's just something that's not very common. But if you do have an anterior pituitary, for example, you, you more than likely will do an MRI to see the extent of the tumor, and then you will visualize the hypothalamus and I just don't want to get into it. Alright, so we're just going to keep it simple, secondary, primary, we're not going to worry about tertiary but I think the TRA story in there, just to kind of throw it in there to let you know that there could be a hypothalamus dysfunction that's occurring, but again, we're not going to get into it. Okay, so what else? This right here? Let's talk about this. Can you have a goiter with hypothyroidism? absolutely you can. I'm going to show you how. So that's going to be our next picture here. So a goiter with hypothyroidism, we're going to take iodine deficiency, for example.

42:48

So let's see how iodine deficiency can lead to a goiter. So number one iodine deficiency. You guys know where we get most of our iodine?

43:01

In our diet. Salt, it's attached to the sodium chloride you ever seen admired Morton's iodized salt, they stick iodine on it. Now, this is not typically a problem in the United States, because we are a very rich country, this is going to be a problem that is Iodine deficiency in poor countries where they do not have the means to stick iodine to the salt. And they're using more like sea salt, for example, sea salt, you get some, you know, salt from the sea that that's going to have iodine stuck to it. So anyway, iodine deficiency leads to a decrease in T three and T four. Now, when I talked about T three and T four, and when I drew it, what did I draw? Well, I draw, I drew a square. The square is the other ingredients, of T three and T four, iodine is not T three and T four is part of the structure of T three and T four. So if you have an iodine deficiency, it doesn't mean that the thyroid gland can't produce the square, they can still produce the square. But it's not T three and T four, it will not go to the T three T four receptors and bind to those receptors really d3 receptors, not gonna have any effect on the cells whatsoever. But the gland can produce the other ingredients. So let's make sure that we keep that in mind. So now let's do the anterior pituitary. We know what produces TSH and we're going to have a thyroid gland. That won't be producing a whole lot of T three and T four but it sure will be producing a lot of the squares. Why? Well if you have low levels of T three and T four we're supposed to stimulate the anterior pituitary which means we should have high levels of TSH and that's what will happen. And the high level of TSH is going to stimulate the heck out of the thyroid gland is going to tell the thyroid gland Would you just make more T three and T four and the thyroid gland said okay, I can do that. But it's going to be crappy T three and T four. Instead, it's just going to be making a bunch of squares. It's going to try but that's not T three and T four. If you did bloodwork the assays will not pick up the squares. It's got to be the squares with the eye on it. So the lab will come back and say hello levels of T three T four with high levels of TSH. But if the thyroid gland is being stimulated like crazy by the anterior pituitary and making a whole bunch of the squares, do you think that the thyroid gland is going to store those squares? It sure will. So they'll be stored which will lead to the goiter. Thyroid gland will get big whether the ingredients are full or not full, whether we have squares or squares with iodine on the gland will still get big. So you can have a goiter with hypothyroidism so this is a goiter with Primary Hypothyroidism that's how it can happen. Now, let's talk about treatments. How do you treat hyperthyroidism? How do you treat hypothyroidism? Let's talk about that. So treatments let's first talk about treatments for hyperthyroidism. I'm just going to give you a handful here we're not going to go through the exhaustive list of things don't have time. One treatment is anti thyroid meds that's really what they're called. These medications.

46:52

Oh, that's gonna suck if that doesn't work. Oh, no. Oh, that really sucks. I just lost all the pictures

47:05

all right. I'll deal with it. Don't worry, guys. Sorry, no, but crash recovery data that might allow you to retrieve some of your loss work. Would you like to try? Oh, yeah, sure. What?

47:23

Yes, there we are. Are they all there? Oh, yeah. Are they Oh, there they are. Okay, treatments.

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Maybe I was riding too fast. Alright, so hyperthyroidism. Treatments, anti thyroid meds. These medications literally just inhibit the thyroid gland from making T three and T four. That's how they work.

47:58

Another treatment is a thyroidectomy. That simply means remove the thyroid now doesn't mean remove all the thyroid. No, it doesn't remove me, you're going to try not to remove all of it. You're going to try to remove the part that's cancer is for example. Now could the cancer have just completely overtaken the thyroid? It could and you might have to remove the whole stinking thing. So anyway, thyroidectomy. And those are just going to be the two treatment options that we talked about hypothyroidism, I'll give you one. Somebody asked about it at the break Synthroid. That's artificial T four. So you're gonna give your patient T four, and then the T four can be turned into T three, but most of the tissues of the body. What's the dose? depends on the person and the dose change? Absolutely. It can. Somebody might be on a good dose for a couple years and all of a sudden it's not working anymore. You have to change the dose. And sometimes it takes months to get the dose right. So Synthroid is going to be the one that we discuss. Now, let me ask you this. If you have hyperthyroidism, and all of a sudden you get your thyroid gland taken out. Now what do you have? Now you have hypothyroidism you went from hyperthyroidism to hypothyroidism. And now you're on Synthroid and Synthroid is the most prevalent one. That's one that I mentioned, but there are others out there. But Synthroid, again, most prevalent medication use for hypothyroidism things like Hashimotos disease. So know those. Yes, sir.

49:29

clarification on primary conditions. So we talked about the iodine deficiency. We think of that, I guess, if I was looking at it as a secondary condition, because that's, there's nothing wrong with it is trying to reduce that.

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It is, but it has the inability to actually do it. And so, where the problem arises, is within the gland itself, it just simply doesn't have that ingredient. It's like the chef that doesn't have the salt To make the whatever the hell cake or whatever the hell chefs, chefs will make cakes, whatever whatever chefs make. So there'll be a problem with the chef. He doesn't have that ingredient. That makes sense. Okay. All right. Now what? Well, now we move forward. Let's talk about oh, calcitonin cross it. Yeah.

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I have a question. So up until this point, we haven't really heard much about the treatments. Is that like, we just need to know that treatment is the other ones were like tumors,

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for the most part. Yeah. And when it comes to treatments, their surgery, chemotherapy, those kinds of sometimes there isn't a treatment. A lot of times with pituitary tumors, there is no treatment. You just got to, and they're typically benign. That's something else that I want to point out. If you have a pituitary tumor, it's not cancerous, that just sits there causes all kinds of stinking problems. Okay, sometimes there are some drugs that can be given to not not chemotherapy, but other types of drugs that can that can address the problems. But for the most part, a lot of times aren't treated like acromegaly and that problem that there's just no treatment for it. You just got to let it go. And unfortunately, people died, usually in their 40s. Yeah. All right. Calcitonin not going to talk about it. Why? Because we don't know what the hell it does. books will tell you. websites will tell you they think they know what it does. We really don't know what this hormone does. We're not smart enough, yet. We'll figure it out someday, won't be on the exam. I can't tell you what it does, because we don't know what it does. parathyroid glands, you guys know where those are, from the thyroid gland itself. Typically, for some people can have as many as about a dozen. parathyroid hormone is what it produces. The function of parathyroid hormone, its main function is to regulate calcium levels and phosphate levels as well. And it does that in a few different ways. It's going to control phone production, bone resorption, it's going to control the kidneys, it's going to control what happens at the gut, I'm going to talk about those things. And that's what we're that's what we're here. So I'll put it in this picture that I'm about to draw. So what I'm really going to do here is I'm going to draw that in one picture. And so let's do that. Now, this particular line that I'm going to draw is blood calcium. It's not parathyroid hormone. So let me make sure that you understand that. So this is the setpoint line for blood calcium levels. Now I could have done blood phosphate, I just chose blood calcium. We need normal levels of blood calcium, and parathyroid hormone is that hormone that's going to play the biggest role in keeping your calcium levels where they're supposed to be. And they should be right here. Now, if something happens, that causes your calcium levels to dip, and why would that happen? It could be a number of reasons, you just don't get enough calcium in your diet, you don't get enough vitamin D or vitamin D deficient. There's a number of reasons, but it really doesn't matter. We have low calcium levels. So that's going to be number one, low blood calcium. Well, what's that going to do? Well, what that's going to do is it's going to stimulate the parathyroid gland to produce them release more parathyroid hormone. So this is going to increase the release up P T. H is going to be acute released, by the way, it's going to be non hormonal regulation. By the way, calcium is not a hormone, it's acutely released, because we have a low calcium level here. And what's going to happen as a result of that, a number of things. So number one, and number two, will cause number three, to happen. Now, what's going to be number three, well, number three, are all those ways that p th is controlled. Which then is going to lead to these changes in calcium. So one of the things that will happen during number three that is going to raise the calcium levels is we are going to increase bone resorption. Remember that from last semester. osteoclast become more active, destroy more osteo sites, and so doing the hydroxyapatite is destroyed and hydroxyapatite has calcium in it, and the calcium ends up in the blood. Okay, that 22nd spiel, right there was about 20 minutes of lecture last semester, if you remember. So we're going to break down more bone to get the calcium into the blood. It's one of the things that PT h is going to do and we actually talked about that last semester. What else is going to cause the kidneys and increase in calcium absorption it's actually called reabsorption, but that's alright, we'll just say absorption by the kidney, so the kidneys are going to take more calcium and transport it into the blood which will raise calcium levels. Same thing is going to happen in the gut and it's going to happen through vitamin d p th controls vitamin D and vitamin D allows your gut to absorb more calcium. So we're going to increase calcium absorbed option in the gut, what's the gut small intestine, for example. So the calcium that you eat, you get to absorb more

of it from your gut. And as you do that, you reach calcium levels back to where they're supposed to be. Well, let's say we have high calcium levels, we'll call that a, we won't go 123. So we don't get any mix up here. So here a, well, we have a decrease in blood calcium,

55:30

I'm sorry, increase, increase increase. So we have an increase in blood calcium be, well, the opposite is going to occur. Now we're going to decrease the release of PTA p th, is chronically released. And we're going to change the release of p th, because we're changing the concentration of calcium. And so as a result of that C is going to bring our calcium levels back to where they're supposed to be, it's going to be the complete opposite of what we see in three. We're going to now increase not bone resorption, but bone what production those osteoblasts. Remember that are going to take calcium from the blood because they need that calcium to produce Hydroxyapatite. Because the hydroxyapatite is needed to differentiate those osteoblasts and osteocytes. So we're going to produce more bone, that's going to lower our calcium levels in the blood. What else we're going to decrease calcium absorption. Again, it's called reabsorption. As you know what it's bugging me, let me put reabsorption there, you'll see what the difference is later on this semester when we get to the kidneys. So the kidneys are going to take calcium from the blood or not put as much calcium into the blood really is how it should be worded. And we're going to decrease calcium absorption

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in the gut. And we're going to lower our calcium levels back to where they're supposed to be.

57:00

And that's what we're supposed to be. So your calcium levels are dictated in most part by p th. And so that picture right there. It's every single one of these words that you see up on the screen. It's all of it, what stimulates and what inhibits and again, phosphates in the story as well. I just didn't talk about phosphate because calcium is a better story. Let's talk about hypersecretion hypersecretion. That's a picture of gut, kidneys bone. Okay, which is either number three, or seeing the picture that we just drew. Let's talk about hypersecretion. Osteoporosis, osteoporosis is what? Two little osteo sighs right. Hi, p th, what is p th D? Let's go back to our picture. p th causes what? When its levels go up resorption bone resorption can lead to osteoporosis. Last semester, when we talked about osteoporosis. I told you that it's an insidious disease takes years and years and years to develop. Most people with osteoporosis are above the age of 50. But then I told you last semester, that there are times when osteoporosis can happen very, very quickly. And at a younger age. Well, I'm giving you that time right now. If you start to pump out a whole bunch of PT, he will start to resorb all kinds of bone and you can develop osteoporosis quickly. And so that's what this is about right here. So can you develop osteoporosis? Yes, high calcium levels, if your pH levels go up, your calcium levels are going to go up. The direction in which pH goes is the direction in which calcium goes we just saw in the picture, we can see clearly that a pH goes up, calcium goes up. It's just here was physiologic. We were trying to raise calcium back up to normal but if you have a whole bunch of bthrm, you're gonna be doing a whole bunch of three. And so your calcium levels are gonna go up, up, up, up, up, up, up, up, up. Now what happens when your calcium levels are high? These kinds of things. Muscle weakness, Dr are we talking about last semester, we talked about how calcium is needed for muscles to contract. That's intracellular calcium. This is calcium in your blood, it's extracellular calcium,

lethargy. So and you don't have to know the mechanism because I'm not going to talk about it. If you want to talk about it in my office or over. over email, I'll be more than happy. high calcium levels decrease the number of action potentials that muscles produce, and that your nervous system produces less action potentials, muscle weakness, less action potentials nervous system, you feel blocked, you become lethargic, so revolves around calcium and its effects on action potential generation, high calcium, less action potential generation. High blood pressure has to do with your heart and your blood vessels. Your blood vessels are going to do this with high calcium levels. Because the smooth muscle wrapped around them. Contract they don't get weak, they actually get stronger, your heart will contract harder and faster. Hearts got muscle Smooth muscle is muscle. What the hell is the difference between that those muscles and your skeletal muscles? Well, we learned about last semester. Muscle art muscle uses extracellular calcium, not skeletal muscle. So that's the difference. It was the last picture we drew last semester. Do you remember that? She the last two pictures when we compare it and we contrast it smooth muscle, cardiac muscle, skeletal muscle, go back and look. So anyway, high blood pressure has to do with the heart, your blood vessels, what would cause it? Most of the time, it's cancer, it's to 10% We don't know what the hell's going on. So idiopathic secondary, just cross it off. It's not a very sexy story, I want to talk about it. So it's not going to be on the exam hypersecretion too little p th, one out, of course, your calcium levels are going to be low, high calcium levels, you are going to cause less action potentials to be generated. Really low calcium levels, you're going to have too many action potentials generated, which is why all of a sudden you're gonna have seizures. You could I mean, it's going to happen. Your muscles, skeletal muscles. They can be overstimulated causing spasms. We talked about that last semester. What would cause it a thyroidectomy. Again, when you do a thyroidectomy, you want to preserve as much of the thyroid as possible, and you sure as hell don't want to remove the parathyroid glands. Because sometimes it happens sometimes it has to happen because of the extent of the cancer. And sometimes it just kind of happens by accident. Fortunately, your immune system could destroy your parathyroid gland. cancer can cause destruction. You could have been born this way. Or it could be we don't know what the hell is going on. But for whatever reason, you're not releasing enough parathyroid hormone, idiopathic. Alright.

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On to the next gland, the adrenal gland, as you will know, because you learned it, there's an adrenal medulla. And there's an adrenal cortex, I already touched upon the adrenal cortex, that's the part of the gland is controlled by ACTH. The adrenal medulla is not controlled by ACTH is controlled directly by for something that we've already seen. So let me see if I have it here. I do. This picture right here, boom. adrenal gland, when we talked about the sympathetic, it's the adrenal medulla, that the sympathetic is controlling the adrenal medulla is actually considered part of the sympathetic nervous system. So it's under neural control, that our home water control center nonhormonal controls, well, it's all three. So let's go back to the picture here. So it's the inner part of the adrenal gland. So we're talking about right now. And the inner part of the adrenal gland produces two hormones, epinephrine, and norepinephrine, otherwise known as adrenaline, and noradrenaline, but we're not going to use adrenaline, noradrenaline, we're going to use the real words, epinephrine and norepinephrine. Most of it is epinephrine. On 80%, the other 20%, it's norepinephrine. We already talked about norepinephrine by the way, last semester doing. It was a neurotransmitter. Remember that? Right? It's also a hormone. It's not just a neurotransmitter. Now, let's talk about now there are there are nuances to these two different hormones. But we're just going to say that they do the same thing. That's what we're going to say we're just going to keep it simple. So these are the things that norepinephrine and epinephrine do when they bind to their respective receptors. They increase the metabolic rate, especially if skeletal and cardiac muscle. What else increase heart rate blood pressure, that's going to help us pump more blood. They increase your blood glucose levels, they increase your fatty acid

levels, they dilate your airways. If you look at all these things, right here, they are the things that you want to have happen during what fight or flight. I told you that the adrenal medulla is part of the sympathetic nervous system. And the sympathetic nervous system is responsible for what reflex, the fight or flight reflex. So these things are the things that you want to have happen. During times of danger, when you need to fight something, or you need to run away from, you're going to fight or run away from something you certainly want an increase in metabolic rate of your skeletal muscles. That's what's gonna help you to fight cardiac muscle, that's what's pumping the blood and increasing your heart rate. Blood pressure is gonna help you pump more blood what's in that blood? Glucose and fatty acids along with oxygen to produce what ATP through aerobic and anaerobic cellular respiration. If you don't have ATP, you're not fighting. You're not running away from anything. You're dead. dilating your airways, well, what's that gonna do? You got to get more oxygen into your lungs. That's where you get the oxygen you breathe it in, you got to breathe off the excess co2 from the body as well. So if you look at these functions here, it should all make sense to when it comes to these particular hormones being released. By the part of the adrenal gland that is considered to be part of the sympathetic nervous system, which we talked about in detail last semester. Now, what stimulates these hormones? Well, the sympathetic nervous system dies via what? Stress is certainly one of them again, fight or flight. So ignore epinephrine, norepinephrine, are they stress hormones, yes or no? Yes, they are part of the stress response. They are stimulated by stress. So they are stress hormones, unlike TSH, unlike T three and T four, but like growth hormone, for example. Like ADH, snow, the stress hormone, physical activity should make sense. walking, running, jogging, playing volleyball, lifting weights, whatever. You want to release more of these hormones wide because it does this stuff. And you need that stuff for physical activity. What else? Low blood pressure. We're not going to talk about it. Now. We're going to talk about it when we get to the cardiovascular chapter. So really, the first two are the first two are the two that I want you to know for this chapter. But you're going to see epinephrine and norepinephrine again. What inhibits these hormones? Pretty much nothing. What do I have here? I have low basal levels. What does that mean? It means under normal conditions, day to day, minute to minute, the levels are just low. And they're so low, that there's really nothing that's going to inhibit them. And if you do inhibit these hormones, you're not even going to know it. So really, there's no inhibitors of these hormones. What's going to happen if you release too much of these hormones? Well, we already know,

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if these hormones increase your blood pressure too much, you're gonna have high blood pressure, too much of them, your heart rate is going to be too high. Too much of them, you're going to have a really high metabolic rate, and you might have too much heat in the body, which would cause you then to sweat. That one of them. It is excess sweating, nervousness, fidget Enos really high levels of glucose. Yeah, because it increases your glucose levels. So again, everything revolves around function. If you know the functions of hormones, you're going to know what stimulates them, you're gonna know what inhibits them, you're gonna know what happens if too much is released or too little is released. Except for we here with this because when it comes to these hormones, because so little is released to begin with, releasing less of them, you're not going to notice the damn thing. Now, you'll notice it if you need them, and they're not getting released, well, you're certainly going to notice that. But again, day to day conditions, you really not going to notice that there's going to be anything going on if you have decreased levels of these particular hormones. That's all we're really gonna say about norepinephrine and epinephrine for now. Now, let's go on to the cortex. And so the adrenal cortex is the outer part of the cortex, or I'm sorry, the adrenal gland. And you know that they have three different layers that go Marissa Llosa, the physical Lada and the reticular parts. And we're going to take it from the outer part of the cortex, we're going to start with the zona glomerulosa. And the zona glomerulosa is what produces aldosterone that produces the mineralocorticoid ads. And the

main one we're going to talk about is aldosterone. So let's talk about aldosterone. And so what we're going to do here is I'm going to show you what regulates the release of aldosterone. And so in this picture, we're going to have the kidneys. So here's the kidneys. And I'm going to make it plural because almost everybody has to have them. On top of the kidneys, as you well know, is the adrenal gland. And that's going to be the adrenal cortex specifically. Although the adrenal medulla is there as well, but the story is adrenal cortex right now. And we're also going to put the pituitary gland in this picture because the pituitary gland as we just saw, is also controlling mineralocorticoid, but it has a minor role just with the mineralocorticoid. So let's put the pituitary gland here. Let's put it over here. And it releases ACTH. I'm gonna put the word minor next to ECT ACTH. Because again, it doesn't play a big role that is under physiological conditions, under some pathophysiological conditions that can play a very big role as we're going to see in the next lecture. So ACTH, as we already saw in the picture will stimulate the adrenal cortex to release aldosterone. If I have minor next to ACTH, that must be in there something major that's going to control the release of aldosterone and there is and it's produced by the kidneys. So the kidneys are going to produce something you're going to release fit into the blood and is called rennet. This is going to be the major player when it comes to the regulation of aldosterone. Renin is an enzyme, some books will tell you it's a hormone, some websites will tell you it's a hormone, and they would be wrong. It's an enzyme. Now, I'm going to show you where reading is, in your notes, I'm going to show you that there's some things that we're going to cross off for now. This stuff right here. We're going to talk about it in the cardiovascular chapter. So I promise you, we're not forgetting about this, it's just not super relevant right now, it will be later. So these are just a couple of chemical reactions that are occurring, that renin is going to catalyze, it's going to catalyze the first reaction, which is gonna lead to the second one, and so forth, so forth. So these two arrows that I'm going to draw, that's one of the chemical reactions. So we're gonna say that rennen is going to be the first thing that's released, which then leads to again, these arrows. So we'll just go here, one, two. So the arrows are the things we're crossing off that we'll learn about later. And so the last thing that's produced during those reactions is going to stimulate the adrenal cortex to then finally release, aldosterone, and we'll call that number three. And we can call ACTH. We'll just call it a because again, it's also a player, but it's not part of the 123 cascade, or the one two cascade, I should say. Now, when aldosterone is released, what is its target? Well, it's got a number of targets, but one of its main targets is actually on the page.

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It's the kidneys. And it's going to stimulate the kidneys. Stimulate the kidneys to do what naturally I'm going to call that for. And I'll also call it B because a was ACTH. So it's going to tell the kidneys to do something, actually, it's going to tell the kidneys to do a number of things. That's the blood. So it's blood vessels. And the kidneys are going to be told to do this, the main thing that the kidneys are going to be told to do is to transport sodium into the blood. And typically, wherever sodium goes, water is going to follow. So I'm not going to draw a solid line, I'm going to draw a dotted line. And the reason I did that is because aldosterone is not directly telling the kidneys to transport water into the blood. Like ADH does if you recall, sodium is going in, that's what telling the kid needs to do. And water is just simply following. What else does aldosterone tell the kidneys to do? It tells kidneys to take potassium out of the blood. It tells the kidneys to take hydrogen out of the blood. So it's telling the kidneys to transport three things. But four things total get transported because we have to add water into it. And I'm going to stop the story right there. So when we come back on Thursday, we are going to finish this chapter by starting with aldosterone. Let me say this before I lose it again.

Endo PM 1-20-22

Fri, 1/28 4:37PM 1:14:03

SUMMARY KEYWORDS

insulin, cortisol, cushing, acth, aldosterone, adrenal gland, blood, release, glucose, diabetes, high levels, inhibit, called, stimulate, people, talk, remember, levels, hormone, problem

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Shall we finish this chapter. So we just got done talking about the physiology about dosterone. And what causes this release, it's mainly rennen. minor role when it comes to ACTH. And we talked about then what aldosterone is going to tell the kid needs to do transport some sodium into the blood, water will follow and transport potassium hydrogen out of the blood. So now what? Well let's talk about then what's going to stimulate the release of aldosterone, then we'll talk about what inhibits the release of aldosterone. Now, what I have here is a line. And that is readiness released in response to these things over here that I have listed that I'm scrolling through decreased blood pressure, low sodium levels, high blood potassium levels, although we're really only going to concentrate on the blood pressure story and the sodium story, we're going to get into the potassium story in the hydrogen story when we get into Pasto Fitz, I want us to concentrate on how aldosterone his main role is to regulate blood pressure. And that's going to get repeated in the cardiovascular chapter, which is why I want to spend a little time on it here. So ready to release a response to these things. So what does that mean? It means this if you have a decrease in blood pressure, so let's go back to our picture. If blood pressure if this is normal blood pressure, my right hand, and our blood pressures down here, my left hand, what do we have to do? Negative feedback says what? We need to raise blood pressure, correct? Aldosterone can do that for us. How is it going to do that for us? It's going to stimulate the kidneys to transport more sodium into the blood, which will then cause more water to enter the blood. So then your blood volume goes up. Your hydrostatic pressure goes up. Remember that from last semester, Chapter Three when we did transport, hydrostatic pressure, as you increase hydrostatic pressure, or how do you increase hydrostatic pressure, there's a couple of ways the way that we learned that you increase the volume in a compartment. And if you remember from last semester, I told you that blood pressure is a type of hydrostatic pressure. So if your hydrostatic pressure is low blood pressure, do you need to raise it? How do you raise it, you add more water fluid to a compartment. And aldosterone can do that for us? Both water up into the blood, you increase blood volume. What else? The sodium levels are low in the blood? What do you need to do? Raise them? You know, dosterone do that for us? Of course it can because it tells the kidneys to transport sodium into the blood. So low sodium in the blood hyponatremia. Low blood pressure stimulates the release of aldosterone. But it does it through rennet. And so what does that mean? It means that low blood pressure stimulates renin, which then stimulates aldosterone, low sodium levels in the blood stimulates renin, which then stimulates aldosterone. That's what this line means here read and released in response to so it's running that's actually responding to these things, then causing our das rooms release. All right. Now, what else? What else stimulates aldosterone stress? Is that dosterone? a stress hormone? Yes or no? Yes, yes, because it is released in response to stress. Now, specifically, without dosterone, it's just psychological stress, not physical stress. And psychological stress would

be mental and emotional stress. And that's what I have the word psychological in there. And that is again, ACTH plays a minor role in this picture that we drew, I told you that ACTH does not play a big role. Physiologically, when it comes to stimulating the release of aldosterone from the adrenal gland.

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What's going to inhibit the release of aldosterone once again, it's working through rennet. So the two things that I have listed over here that are going to inhibit the release of aldosterone are actually inhibiting the release of renin, less renin, less aldosterone. And of course, if your blood pressure is high to begin with, you're certainly not going to want to release a hormone that raises your blood pressure, so you'll inhibited if you have high sodium levels, hypernatremia, you're certainly not going to want to release a hormone that raises sodium levels even more, you're going to want to inhibit it, playing on negative feedback. So that's the physiology of aldosterone. Now what now let's go to the pathophysiology. And now we're going to include the potassium and the hydrogen story in this story. Effects of aldosterone hypersecretion, so too much aldosterone, it's going to get back to our picture here. Too much aldosterone means you're going to have too much sodium transported into the blood hypernatremia. Too much aldosterone. Too much water in the blood. High blood pressure. Too much on dosterone. Too much potassium getting transported out of the blood. Your potassium levels in the blood will be low hypo Colonia too much aldosterone, too much H plus being transported out of the blood. So your H ion concentration will go down what happens to pH? Low H plus pH is high, right? There's an inverse relationship between h plus concentration and pH. So you'll have what's called an alkalosis with too much aldosterone. And again, I told you that that's pathophysiology and how we were going to visit the potassium and the hydrogen store. What would cause this aldosteronism. So when you hear the term on dosterone ism, what it's saying is there's too much aldosterone. And we have two kinds primary and secondary. So let's visit this what would the blood work look like? So let's compare and contrast these two. So primary aldosteronism. That over a little bit, versus secondary. And I have a specific disease name for primary, secondary aldosteronism on this side. And we're going to put all the players involved here, I'll throw the pituitary and even though ACTH plays a minor role, this is pathophysiology. So again, ACTH minor role, physiologically, patho, physiologically, the story can change. So primary aldosteronism will be the first one we draw over here. So I'm going to have this is not going to be anatomically correct. By the way, I'm going to separate the adrenal gland from the kidneys in this picture. I'm gonna put the adrenal gland down here. Both sides. And I'm going to have the kidneys over here. And I'm going to put the pituitary over here and I'll label everything. So anterior pituitary, kidneys, of course, the adrenal gland, but I'll put it there anyway. And so we have high levels of aldosterone. And so what should that do to the kidneys sit as stimulator? Should it inhibit? What should it do to the pituitary to the stimulator should it inhibit? We want to lower aldosterone. So we would do what to those two, we would inhibit, which is playing on negative feedback. And so we would inhibit the kidneys, we would inhibit the kids. And if that's the case, and it's going to be for this story, we should have low levels of renin. And we do with primary aldosteronism, we should have low levels of ACTH. And we do with primary aldosteronism. Those two things will try to lower the levels of aldosterone. But it's not going to work because we have a primary problem here. And the problem, the specific example that I have in the notes is a tumor. It's called cons syndrome or cons disease. And so we have a problem here.

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And so we have what's called cons. And so that's primary. And what would the blood work look like the blood work would look like high aldosterone, low renin and low ACTH. And now we'll tell you like that, where the problem lies. And the problem lies at the adrenal gland. If you understand negative

feedback, and hopefully you do secondary aldosteronism, there's not going to be anything wrong with the adrenal gland. But something is going to drive it to producing release too much aldosterone. And so we will put the kidneys and the pituitary in this picture as well. Now I am going to put the word or here, although I suppose it could be and I suppose both Pituitary and the kidneys can kind of go bad and cause this to occur. But we're just going to do or and just say it's either the kidneys or the pituitary, that's going to cause the problem here. So here's our anterior pituitary. So, as you already told me just a second ago, high levels of aldosterone should inhibit the kidneys should inhibit the pituitary. This is a secondary issue. And so we don't have an adrenal gland problem. We're going to have a kidney problem or we're going to have a pituitary problem. And so even though we're supposed to inhibit the kidneys, that is not what's going to happen here. And the kidneys are just going to keep on dumping renin. Or maybe we have a pituitary problem. And the pituitary just keeps on dumping a whole bunch of ACTH. And so given the blood work and the blood work can be high aldosterone, high renin or high aldosterone high ACTH. We know immediately that is a secondary issue. So the reason that the adrenal gland is releasing so much aldosterone is because there's just too much rennet or there's too much ACTH now Before you did the blood work, and you suspected that this person had aldosteronism, could you look at that person? Again, primary versus secondary? And let's say that you suspect either an anterior pituitary problem, or an adrenal gland problem. Would there be something about the person? Let's say that they had secondary aldosterone isn't that might be evident, given the blood work. ACTH being high. Remember what ACTH stimulates? Starts with an M melanocyte. Melanocytes remember that? ACTA stimulates melanocytes, we did that. I'll actually show you. I don't remember which lecture it was, though. Maybe it was this one. It was not that one. It was the next one.

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It was one that one. Was it the one before this? How many lessons have I had here?

11:04

I don't even remember. It's just the one. No, it was the last one. Sorry, I'm thinking I was going to be efficient about this and look at me waste time.

11:19

Price, where is it? Oh, hell, there it is. ACTH stimulates melanocytes we already talked about. And I told you that we could use this darkened skin as a means to diagnose a patient. So somebody with high ACTH levels might very well have darkened skin. So a secondary aldosteronism. If it's due to a pituitary issue, person could very well have darkened skin doesn't mean they're going to know it doesn't mean they will but they certainly could. Okay. So anyway, that's all dosterone is. Now what Well, now let's go the other direction, and let's talk about low levels of aldosterone. And if you have low levels of aldosterone, let's go back to our initial picture, then that would be this one, low levels of our dosterone less sodium in the blood hyponatremia less water in the blood, maybe your blood pressure is low, less potassium getting transported out of the blood, which means more potassium will be in the blood hyperkalemia less hydrogen being transported out of the blood, higher hydrogen concentration, lower pH patient would be in an acidosis. We'll talk about these things later on in the semester when we get to acid base balance. So what would cause that something called Addison's disease? Now, I am going to actually interject another term here that I have written later on in the

notes. I should have written it at this point. And it's called adrenal insufficiency. And I talked about it when we get the cortisol, but I'm going to talk about it right now. So there's primary adrenal insufficiency and the secondary adrenal insufficiency, we're just going to do primary for this. And so let's do this now. So this is too little out dosterone And what would cause it and so I'll write the term again, it's in your notes later in bold, primary adrenal insufficiency. And there's going to be a specific condition that I name in this specific condition is Addison's disease. So let's put our players in place here. So we're going to have our kidneys, anterior pituitary. And then, of course, the adrenal gland. And the adrenal gland is not producing and releasing enough aldosterone, so we have low levels of aldosterone. And again, this is a primary condition. So the problem is where obviously it's at the adrenal gland, right? We have low aldosterone, what should that do to the kidneys in the pituitary, we should stimulate that's just playing on negative feedback, we want to try to raise the level of aldosterone. So that's what should happen if the pituitary is working properly. If the kidneys are working properly, then we should have high levels of ACTH. And we should have high levels of rennet. And that's exactly what's going to happen with the primary issue, because those two things are working just fine. Unfortunately, those two things are not going to be able to increase the level of aldosterone because the adrenal gland is getting eaten by the immune system, because this individual has a condition called Addison's disease. So Addison's, and what Addison's is, is autoimmune destruction of the adrenal cortex. And so now the adrenal glands This in sufficient? Now, there's a term that's used out there that really sometimes has nothing to do with any type of pathology or pathophysiology is called adrenal fatigue. Have you ever heard of that? It doesn't exist. Okay, don't use the term adrenal fatigue. There's no such thing. All right, I just want to kind of throw that out there. There are certain professions that use that term. And they would be wrong. So anyway, that's this right here. And again, I interjected the the insufficiency term, which is later on in the notes, you'll see it. Oh, by the way, what else might this patient have given the ACTH levels? Besides high blood, their low blood pressure and all that darkened skin as possible? So I'll put that there can have darkened skin maybe doesn't mean they will.

16:00

But they certainly could. So you could have darkened skin with low levels of dosterone. You can have darkened skin with high levels of testosterone. It depends on what the issue is. All right. Now what, let's move on to the next part of the cortex. Let's talk about cortisol. glucocorticoids. Cortisol, what are the functions of cortisol? It is an anti inflammatory. And it does it in a few ways. The one that I have highlighted, the one that I don't have crossed off at the front of the room is inhibit series to prostaglandins. And so I will remind you of this, and I should get this one right off the bat, that would be this one. No, it wouldn't screw it up. Of course I did. Which one is it this one, this must be the one. It is. So let's go to our prostaglandins story, which is right there. If you recall, our series to prostaglandins are the ones that cause inflammation. And I told you that if you deem it if you decrease the production of prostaglandin to you decrease inflammation. I wasn't talking about cortisol at the time, I was talking about NSAIDs, like aspirin, naproxen, those kinds of things. And that's that stuff down here. Cortisol does the same thing. Cortisol is a COX inhibitor. And so it will diminish PG two, which is causing the inflammation in the body. So it's an anti inflammatory. It's also an anti inflammatory, as you can see, in a number of other ways, one of which is decreasing the number of white blood cells. We haven't talked about white blood cells yet, we will actually see it will, white blood cells are highly responsible for your immune system. So cortisol actually suppresses your immune system, which I will actually talk about when we get into the pathophysiology. As far as decreasing inflammation, the only thing you have to know, inhibits PG two. That's what I want you to know, because we had already discussed it. And then there's a third one here inhibits the release of histamine from mast cells. Again, these two Don't worry about him worry about the prostate gland in two, but we will talk about suppression of the immune system. When we get to the patho fits. What

else does cortisol do? blood vessel tone. So what does that mean? Well, last semester, when we did the autonomic nervous system, did we not talk about how smooth muscle is wrapped around blood vessels, right, and how we can control the tone of the blood vessels by controlling the tone of the smooth muscle. Cortisol plays a part in that if you increase the tone of smooth muscle, they contract harder, and blood vessels they constrict. If those smooth muscles don't contract as hard, because you've diminished, the amount of cortisol released, blood vessels will dilate. That will control blood flow. Obviously, it'll also control blood pressure. I'll tell you how, when we get to the cardiovascular system, it has to do with resistance. What else increases your glucose levels in the blood?

18:55

But it does it in a way that's not so great. It does it via gluconeogenesis, specifically by taking the amino acids and turning them into glucose. And that's something we talked about last semester. It was chapter one, and it was the last page of Chapter One. Do you remember you remember if not, that's okay. Unfortunately, a lot of the protein or a lot of the amino acids are derived from protein in the body and a lot of the protein in the body is in your muscle. Cortisol is a steroid. And typically when you hear the word steroid, you probably think well, that builds muscle. anabolic steroid does build muscle this steroid does a complete opposite. This steroid breaks muscle down, it's catabolic. That's unfortunate, but it does it to raise the glucose levels. What else? It is a stress hormone. This is released in response to stress and if we had to rank the stress hormones, this would be at the top of the list. When it comes to stress hormones, cortisol, it's kind of the big dog. So it is released in response to stress So what stimulates cortisol, stress? Inflammation, of course, it's an anti inflammatory, you have inflammation, you're going to release a hormone that diminishes inflammation. Of course you are, that's negative feedback. If your glucose levels are low, of course, you're going to release cortisol because cortisol raises glucose levels, of course, you will. sleep wake transition, this is actually something we already talked about with ACTH. So going back to ACTH, remember how that was released. When you get up in the morning, and its lowest levels or when you're in deep sleep, remember that and I compared it the growth hormone is the complete opposite of growth hormone. ACTH plays a huge role when it comes to cortisol, which is why that cyclic release of ACTH is going to be manifested through cortisol. So cortisol is a cyclic Lee released hormone is chronically released to it's acutely released, in response to glucose in response to inflammation in response to stress, acutely released significant injuries, sleep wake transition versus deep sleep. It's released in all three ways. What else, it's regulated in all three ways to it's regulated again, by the things that I just mentioned. So the things that we talked about in the first chapter, cortisol covers them all, how its regulated, how it's released. Low ACTH, of course, you're not going to have as much cortisol released. So now let's go on to the patho fits, let's talk about how too much or what's going to happen when too much is released, what's gonna happen when too little is released. And then we'll get into a couple of conditions. So if you have too much cortisol release, and obviously, your glucose levels are going to be really high. Because it is a hormone that causes the production of glucose. It could lead to if the levels are really high over a long period of time, to diabetes, type two diabetes, and we're going to talk about diabetes today. Diabetes mellitus is called adrenal diabetes. More on type two diabetes later increased production of fat. Well, where do I have fat over here? When it comes to function? I don't. But I do have glucose. So when you have excess glucose, what do we do to the glucose? We store it right? What's glucose stored? EFS? So the G, glycogen remember that? Do we have an infinite space for glycogen in our body? No, your muscles can only store so much glycogen, your liver can only store so much glycogen. And once those stores are full, that's it, you're not going to make more glycogen, you're not going to store glucose anymore. Well, let's say you still have high levels of glucose. Now what are you going to do to the glucose, you're going to store it in a different way, you're going to turn it into white fat. So why is it that when you have high levels of blue or cortisol that you start to produce in store more fat, because you don't have any more glycogen

stores to to distort the glucose. And when it comes to the fat, that is stored, a lot of it is stored in the face. A lot of it is stored in a tropical region, and kind of right below the neck and you're trapped. You're trapezius area. moonface. And this is actually terms that are used medically. And buffalo hump, I'm going to show you what those look like. I'm gonna show you pictures of people with with a condition called Cushing's. What else happens? muscle wasting, it tells you that this is a this is a steroid that destroys muscle. And why is it doing that? To take the protein, get amino acids out of it to raise our glucose levels. What else a Deema. This is something we talked about last semester, it was chapter three, when we were talking about transport. And we were talking about osmosis. Remember how much time we spent on osmosis last semester.

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That tells you a number of ways how we can because we have edema. And edema is just extra fluid or water in the interstitial space. And if you lost protein in the blood, the blood becomes what hypotonic. And the example I gave you last semester was because of starvation, member starvation. And I gave you the example of those starving kids that the show on TV that you can sponsor for, you know, five bucks a month. I mean, it was big bellies. And the big bellies were because of a Deema because they were starving. So they didn't have enough protein in the blood. less protein in the blood. Why? Because proteins get broke down all over the body. And so the blood becomes hypotonic and then water is transported out of the blood wire into the interstitial space. The demon what else? High blood pressure. Why that goes to this. High levels of cortisol causes your blood vessels to do this to constrict increases your blood pressure. How did that happen? I'll tell you in a few weeks when we get to the cardiovascular system. What else frequent infection your immune system is depressed By cortisol, and that goes back to it decreases the number of white blood cells, or at least that's the story we're gonna I mean, that is part of the story. That's the main part of the story. It helps us deal with stress, but unfortunately, it decreases inflammation. But unfortunately, it causes a bunch of bad stuff to happen. And so let's talk about a couple of conditions. One's primary. And one secondary. Yes. Skin. Oh, the skin. All right. Well, let's talk we'll talk about that as well. Oh, no, here it is. But those are more prominent face Pierce? No, I'll tell let's talk about the skin. So if it's in your notes, we'll talk about it. So your skin gets thinner, with high levels of cortisol. Now, why would that be? Goes back to protein being broken down. Thanks for pointing that out. Collagen is collagen a protein? Yes, where's collagen in your skin? In the dermal layer, remember that whole bunch of collagen in the dermal layer is also elastin in the dermal layer. Remember that the dermal layers as thick as part of your skin is not. When you start to break down protein, collagen, elastin, what's gonna happen in the skin, it's gonna get thin. So people with high levels of cortisol over long periods of time, their skin gets thinner. And so now I'm recalling my notes, talk about the face being read. So if the skin gets thinner, your blood vessels are going to be more prominent. And so people with high levels of cortisol for long periods of time, their face will look really, really red. They'll also get what are called stray, I'm going to show you a picture these you know what, let's just go to the picture. So these are people with Cushing's. And so what we have over here is this is a good picture of the moon face, this person and this person are the same person. She has Cushing's here, she doesn't have Cushing's over here. So this is showing the moon face. This is the camel hump I'm talking about right over here. Okay, it's just fat deposition. This guy right here is the same guy. And over time, he was developing Cushing's. He just thought he was gaining weight, I guess he was being evaluated by some physicians. And somehow I don't know how the hell they couldn't figure it out. But they did. Anyway, a long time with Cushing's Unfortunately, he's better now. She's better now I don't know about her. Hopefully, she's better as well. Here's kind of a cartoon pictures and things. And so there it says, red cheeks, and the red cheeks are all about thinner skin, and the blood vessels become more prominent. Here are the stray that I'm talking about right here. And those are prominent blood vessels in there, you know, mostly in the abdominal area, is where you'll find that what else it says thin skin over here. Oh,

another thing too, is that typically though, you'll have a lot of truncal fat. And you'll have skinny arms and skinny legs. Why? Because all the muscles getting broken down. Okay, these are severe forms of Cushing's, by the way, there's moderate, okay, there's, there's there's severe forms, there's there's all you know, different forms of, of these conditions that we talked about. But here again, I pick pictures where it's a bit more severe. So and when people have Cushing's it's almost it's like kind of blatantly obvious that they have Cushing's but you still have to do the blood work. And so let's talk about the blood work. And so what we're going to do now is that we're going to compare and contrast Cushing's syndrome to Cushing's disease. So we'll have two columns here. And we'll put Cushing syndrome on the left Cushing's disease on the right. So Cushing syndrome versus Cushing's disease and so the two players involved here, adrenal gland, anterior pituitary.

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And so here's our anterior pituitary, adrenal gland at the bottom. And of course, in both cases, we're going to have high levels of cortisol

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causing all the issues that we're discussing. High levels of cortisol, what should it do to the pituitary? Should it inhibit or should it stimulate should inhibit, we want to lower the levels of cortisol, we can do that with ACTH by inhibiting the anterior pituitary. And what's gonna happen with Cushing's Syndrome? Is that's exactly what's gonna happen with Cushing's Syndrome virus, we are going to inhibit the pituitary because there's nothing wrong with the pituitary. And that pituitary will desperately try to lower the levels of cortisol but it's not going to work. What kind of problem do we have here? primary or secondary? It's a primary issue. Cushing syndrome is primary. Most often we're talking about a tumor or the adrenal gland. Cushing's disease, on the other hand, is a secondary issue. We should inhibit, not going to work because we have a pituitary tumor. And we're pumping out a whole bunch of ACTH. And that's what's driving the adrenal gland to produce and release a whole bunch of cortisol of these two people. Would there be one thing that you might be able to tell when it comes to a diagnosis without even doing the blood work yet? person on the right person on the left? Which woman have darker skin? Versus on the right, correct Cushing's disease high levels of ACTH doesn't mean they will, but it certainly means they could. And if you do the blood work, Cushing syndrome, high cortisol, low ACTH Cushing's disease, high cortisol, high ACTH, that's what the blood work would look like. Now, there's something else in your notes. There's a third possibility here. The third possibility is due to prolonged cortical steroid use. And so what is a cortical steroid? Actually, let's just put it on a separate page here. So, corticosteroids actually, let me write this out Cushing's Syndrome could be due to prolonged corticosteroid use. Once a corticosteroid, a corticosteroid is a glucocorticoid. Yes. Okay, well, I mean, I'll get back to it. Okay, or at the break if you need to get it. So Cushing syndrome can be due to prolonged corticosteroid use what is a corticosteroid? A corticosteroid. I'll give you a couple of examples of a corticosteroid. corticosteroid. If you hear prednisone about cortisone, both of those are corticosteroids. They're steroids. But they're not anabolic steroids. Bodybuilders aren't taking this athletes aren't taking this because this is going to do what cortisol does. Use are going to bind to the cortisol receptor. And so these two are what to the cortisol receptor by the way, they're agonists correct. So they are agonists of the cortisol receptor, they are going to mimic cortisol. Now, not only prolonged use, but at high doses. If you take prednisone and maybe five milligrams of it a day, for years, you're not getting Cushing's. But if you take 60 milligrams of it a day, every day, for months and months and months and months and months and months and months and years, chances are you're going to develop Cushing's. Now, if

that's the case, why in the hell would people be prescribed these types of drugs? Well, they mimic cortisol. And there's two main reasons that people would be prescribed these drugs at high doses over long periods of time. By the way, you can be prescribed prednisone for a week, it just depends on what you had. Again, we're going high doses, long periods of time, prednisone for a week, doesn't matter if the dose is 60 milligrams, you're not getting Cushing's you have to be on it for a long time. So why would you take these why take why prescribe

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two main reasons. Number one, autoimmune conditions like lupus, for example, or rheumatoid arthritis. autoimmune conditions are conditions where your immune system gets stupid, and it does dumb things, like eats stuff in your body.

34:07

It's overactive. That's what an autoimmune condition is. It's an overactive immune system. Does cortisol suppress the immune system? Yes. So you take these to suppress the immune system of somebody with an autoimmune condition. So it suppresses the immune system. Now there could be a danger to that, what's the danger? Now they have a crappy immune system. And so now you got to be very, very careful with things very simple things, especially now with freakin COVID. You have somebody with COVID and they're on a steroid. You got to be very, very careful. So that's one of the things inflammation, some inflammatory process that's going on in their body. Cortisol these steroids are powerful anti inflammatories. And so that's another reason. There's certainly a risk, though what's the side effect develop Cushing's. And so you have to kind of weigh the good and the bad. And so sometimes these autoimmune conditions can be very, very progressive lupus can kill you. Rheumatoid arthritis sucks to have. And so it might be better to develop the problems that we see with high levels of cortisol and not die from lupus, or not be completely debilitated from the rheumatoid arthritis to where you can't even grab a pencil anymore. You're in just so much stinking pain, all the time. Whereas sometimes as people develop these side effects from high levels of, of these corticosteroids to say screw it, I'm done. titrate me off. I don't want to do this anymore. Because I mean, you are going to be miserable with this bus, you can develop high blood pressure, right? diabetes, type two diabetes. So there can be some really, really bad side effects. So that's something that you will someday discuss with your patients. This is the side these are the side effects. These are the benefits. What do we want to do? Yes? Would that be a side effect due to cortisone injections? No. So you can get like a cordless cortisone injection in a joint? That stain right there. Yeah. And plus, didn't last that long. We're talking long, long, long, long, long time with this kind of thing. All right. So are we good with that? So that took care of all that, yes. Cushing's like for the pictures, is that talked about the syndrome or the disease? Oh, for the pictures, the defense that people are going to look just like that, whether it's Cushing's Syndrome or Cushing's disease. Now there could be like, maybe darkened skin here, there. And that would be what Cushing's disease? I'm not really seeing much of it here in any of these pictures. Although she might have, I don't know, well, that looks kind of natural to her. So probably not. You can see the redness in her face, by the way, right. So that's that redness in the face as well because of the thin skin in the prominent blood vessels. Okay, now what? Let's go the other direction, low levels of cortisol. Here's the primary adrenal insufficiency and your notes for the first time, but I've already introduced it to you, and then secondary adrenal insufficiency. And so let's do this very quick. And then I'm going to give you a break, so that we have low levels of cortisol, primary and secondary adrenal insufficiency and we'll just go through it quickly. Secondary, and we'll see what the bloodwork is going to look like. Although

we already know because we've done these so many times. Primary, we're going to have a problem at the adrenal gland. Secondary, we're gonna have a problem with the pituitary. So there's are the bits there, there's a pituitary. And again, we have low levels of cortisol. Now at the levels get really, really low, it's going to be difficult for the body to deal with stress and inflammation. And on a day to day basis, even if you don't do much at all, your body is going through a tremendous amount of stress and producing an awful lot of inflammation. So this can become life threatening when you have really low levels of cortisol. If it's really low, it's a little bit low, not so much. But anyway, if we have low levels of cortisol, what should we do to these to the pituitary stimulator inhibit, we should stimulate. And so what should happen is if there's nothing wrong with the pituitary gland is we should have high levels of ACTH and we do with the primary adrenal insufficiency, or that should be insufficiency, not sufficiency. But it's not going to work. Why we're going to go back to Addison's disease. So Addison's, Addison's can affect aldosterone. Addison's can affect cortisol.

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So the immune system will eat both the outer layers of the adrenal cortex. And so our problem is with of course, the adrenal gland over here, what should have happened is we should have raised a level of ACTH but it's not happening. So our problem is here with a secondary adrenal insufficiency and now we have low cortisol levels because of yet alright. Okay, folks, let us take a break. When we come back, we will finish up this chapter. Alright folks, here we go. So now what are we going to do? We're going to do the last part of the adrenal gland and we're going to talk about their ridiculous region. Androgens. Not a whole bunch of antigens are going to be released from the adrenal gland. And really when we talk about this, we're only going to be talking about females because in males, it's not going to make one bit of difference. And this is the reason so when it comes to these androgens, there's two kinds dehydroepiandrosterone, otherwise known as DHEA, and androl. Steam Daya. DHEA is the main androgen released by the adrenal gland DHEA in males and females gets converted into testosterone. And in females will also get converted to estrogen and roasting die on gets converted to testosterone, both male and female. The reason we're not going to talk about males is because males have a lot of testosterone produced by the testes. And the levels are so high that if the adrenal gland just stopped making DHEA and androstane die on males would not know it. Because their testosterone levels would be so high, it would be completely masked. Females however, do not have testes. And so their source of testosterone is in large part from the adrenal gland by converting DHEA and androstane down into testosterone. And so that's why it matters in females and males and less the males don't have testes are now working that it would matter in males. But that's a very rare thing. When it comes to the function of DHEA and roasting dye and once you get it gets converted into testosterone. And that's what I have here effects were only significant females, it plays a large part in libido and we already talked about what libido is its sex drive, what stimulates it ACTH what inhibited low ACTH we're not gonna spend a lot of time on this, by the way, effects of adrenal hypersecretion or antigen hypersecretion. Two things that I want you to know. And again, it has to do with females, well, they're going to have a high sex drive, probably not necessarily masculinizing effects as well. Meaning why? deeper voice maybe bigger muscles, facial hair, those kinds of things. masculinizing effects, again, because that's what testosterone does. Caused by and again, we don't we're not going to worry about really secondary sex characteristics, short stature, ambiguous units, we're not going to worry about that stuff. And as always, I'll send out you know, the the thing where I cross out the stuff that you don't have to know for exam, just as I did last semester, I'll do it for this one as well prompts. What would cause this? Well, it's a negative feedback thing due to actually not the androgens, but due to low levels of aldosterone and low levels of cortisol. adrenal insufficiency primary adrenal insufficiency, so I'll write this up really quick for you guys. So it's called adrenal genital syndrome.

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So here's the story. The story is this. You have an adrenal gland that is not producing and releasing very much cortisol or aldosterone. And what could cause that? Certainly Addison's disease, right? Primary adrenal insufficiency. And so we could have Addison's here. Oops, I don't want to put it on that side, you know, didn't matter. So Addison's is causing that to occur. Well, if we have low levels of cortisol, we have low levels of an ER of aldosterone. Of course, we already know what's going to happen here with the anterior pituitary, by the way, there's nothing wrong with the anterior pituitary is both of these should stimulate the heck out of the anterior pituitary. And if that's the case, and the pituitary is working properly, and it is you're going to stimulate the heck out of the adrenal gland. Well, it's not going to help with cortisol, it's not going to help without dosterone. But the third section of the adrenal cortex is intact. And it's going to get stimulated a lot. And so we'll go number one, cortisol, aldosterone, you know, like number one should be Addison's disease because that's what's going to cause our issues here. So number one is Addison's number two, that will result in low cortisol, low aldosterone number three, we're going to stimulate the heck out of the pituitary number four, we're going to have high levels of ACTH. That's going to lead to high levels of the androgens leading to adrenal genital syndrome hypersecretion. And that's obviously then five plants plain old negative feedback. Low levels of DHEA. decreased libido and females again, in males. We're not going to know it again, because we have high levels of testosterone. So that's it. No more adrenal gland. Now what? Last but not least, let's talk about the pancreas. The pancreas is both an endocrine organ An exocrine organ, we're going to see the pancreas again, with the digestive system as extra confections very important exocrine functions when we get to the digestive system, when it comes to the pancreas, its endocrine activity is going to be all about the production of two hormones. And so what we're going to do here is that's the pancreas. And I'm going to draw an imaginary line, I don't know right about there. And I'm going to put beta cells here, and I'm going to put alpha cells here and that smaller section, we're looking at about 70% of these cells are going to be beta cells. And about 30% of these cells are going to be alpha cells. Now there's the same type of cells, there, what are called islets of Langerhans, I'm going to show you where that is in the notes. It's in bold. Right here, so islets of Langerhans. And there's two kinds, there's beta, and there are alpha. And each of these cells is going to be producing and releasing a specific hormone, the beta cells, the more abundant cells are going to be producing and releasing insulin, the alpha cells, glucagon, we're not going to spend hardly any time on glucagon, but we're going to spend a lot of time on insulin. And so let's talk about insulin. Now. What does insulin do when it's released, where's it going to go gonna go to certain cells in the body. And so I'm just going to draw a cell here.

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And I'll label it cell. And we'll put the pancreas up here. And I'll actually even put beta in there, releasing insulin. And I'll just say that that's number one, their release of insulin going to be dumped into the blood, of course it is.

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It's going to make its way to this cell and stimulate it. And then that cell is going to be told what to do stuff is going to happen inside the cell, the cell is going to do what it's supposed to do. And what is it going to do? Well, we have to put the blood in this picture. So it's a blood vessel. This is the main thing that insulin does. And it's what we're going to be concentrating on for most of the discussion

about insulin. And that is this. Number three, it's going to tell the cell transport glucose out of the blood and into the cell. And I'm going to make that number three. And that's the main function of insulin to allow glucose to be removed from the blood. A couple of other things as well. Oh, and by the way, you need insulin to do this, you need insulin, to transport glucose out of the blood must have it. These other two things, you do not necessarily need insulin, and one of them is to transport amino acids out of the blood into the cell. That's also number three, insulin not needed. And another is transport fatty acids out of the blood and into the cell. Insulin not needed for either of those not necessary. But insulin still facilitates both of those transport processes. Because the glucose story is the main story. That's the story that we're gonna revolve around. So let's talk about when it comes to insulin. What it's doing so what we just did there is draw this, so we just do this functions right here. And so will facilitate protein synthesis. It'll facilitate the storage of fat. But again, glucose is our main story. What stimulates insulin, high blood glucose. That's the main stimulus and so on, we're going to concentrate on what inhibits low blood glucose. And by the way, stress inhibits insulin. Is insulin a stress hormone? Yes or no? It is not. Right. So let's do this. You're going to put a couple of other hormones in this picture as well. Not just a couple, but quite a few, not just insulin. So this line right here is not insulin, it is blood glucose levels. And you should have a normal range when it comes to blood glucose. But let's say that your blood glucose levels go up. Now, why would blood glucose levels go up? You just say some food. You just say some carbs. Pizza. Whatever. Twizzlers? Whatever. Pasta mama Tucci pasta. So you're going to stimulate the release of insulin Of course you are because you're going to want to remove that glucose from the blood. And as you do Your blood glucose levels go back down to where they're supposed to be. And So insulin is going to do this for us, thankfully. Now, what happens when your blood glucose levels are really low? Let's say you didn't eat all day, and you're hypoglycemic. What does it do to insulin? Well, it actually inhibits insulin. So you stimulate insulin there. I'm just going to draw that a little bit lower. You'll see why. So we're going to inhibit insulin down here. So what stimulates insulin, high glucose levels, what inhibits insulin, low blood glucose levels, of course. That'll help get our blood glucose levels back up. But there are other things that are going to actually really raise our blood glucose levels, a bunch of other hormones, and I'm going to list them now. And hopefully, in the order that we learned them. Does growth hormone raise our hands or glucose levels? Yes. How about TSH stimulating T three and T four, this T three and T four stimulate the, or increase our blood glucose? Yes. How about norepinephrine? And epinephrine? Yep. How about ACTH via core cortisol just got done talking about it. Then this last one, we had yet to speak about glucagon. And really, that's all I need you to know about it. It's just a few other things in the notes. But this is really what I want you to know about it. That glucagon again, raises your blood glucose. So what does this show us? It shows us that we have a whole mess of hormones that can raise your blood glucose, but we only have one that can lower it. And that's how important insulin is. Without insulin, you can't lower your blood glucose levels. And if you don't do that, you're screwed. You're actually dead. And then take that long.

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Now what? So that's the physiology of insulin, and how is regulating your blood glucose levels? Now, let's talk about this. And in order to do this, we're going to compare and contrast type one diabetes to type two diabetes and diabetes mellitus, not the other one, which was in syphilis, remember that in syphilis has to do with what hormone but I make a D, H, right? diabetes insipidus, low levels of ADH. Remember, that was the first hormone we talked about. Or maybe it was the second hormone we talked about when we got off of the hypothalamus anyway. So let's talk about hyper secretion of insulin and hypo secretion of insulin, we're going to talk about it at the same time. So we're gonna compare and contrast type one, type two diabetes. So we're going to take all these words right here, and we're going to draw a little chart to college, and talk a little bit more about it. So type one diabetes, we'll put on the left hand side, type two, we'll put on the right hand side. So type one

diabetes. Again, this diabetes mellitus and type two diabetes. And again, that's diabetes mellitus. Now, before I move forward, we're going to talk about the prevalence of each of these. You you're looking at about 5% of the total cases of people with diabetes have type one. And roughly obviously, then 95% of the people who have diabetes have type two now, not too long ago was 10% versus 90%. The reason it's now 5% versus 95%, is it because the people with type one diabetes, or there's less people with type one diabetes is because there's more people with type two diabetes, that's the reason that the percentages have changed. As you get even further apart, more and more people are getting Type Two Diabetes, unfortunately. Now type one diabetes, cause what is the cause type one diabetes, the cause is autoimmune. So this is an autoimmune disease, destruction of the beta cells, no beta cells, no insulin. And so this is hypo secretion and you can't get any more hypersecretion than a zero. And so because of this, you have no insulin. So type one diabetes is a Hypo secretion issue. Now Oh, and by the way, you can get it at any time in your life. Most of the time, it happens earlier on in your life, but it doesn't mean you Can't get it when you're 2025 or so 30. You can get it at any time. It's like any autoimmune condition. You're not born with it. All right? How do you treat it? Well, there's only one treatment, you need insulin, you don't take insulin. And there are a number of ways to take insulin. There are shots and is very fast acting, insulin is fast acting and as long acting and has all kinds of different insulin. But fortunately, technology has now developed what are called insulin pumps. And those insulin pumps are just continuously infusing insulin, you still have to kind of adhere to a certain lifestyle, not change it too much. But if you do, then you have the injections. So if you want to like an extra piece of cake, maybe you take an injection of like a very fast acting insulin, we're not going to get into that kind of detail. You need insulin, if you don't take it, you're going to die, unfortunately, and you're not going to last very long. And by the way, they can be treated to the point to where the person can lead a completely normal and very, very long life. Okay, so technology has come a long way. Because you need insulin is called insulin dependent.

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Diabetes. It also used to be called juvenile onset diabetes, because again, most people that get it, the onset is at a young age. And it used to be the type two diabetes was called adult onset. Well, they don't use those terms anymore. And the reason is, is because type two diabetes is being found in really young people. Teenagers are getting type two diabetes, and actually some, you know, eight 910 year olds are getting Type Two Diabetes, that was unheard of, even 20 years ago. So what's causing type two diabetes? What is the cause? The cause is insulin resistance. That's the cause. Now, what is insulin resistance? Let's go to our first picture. So our first picture here is insulin, targets a cell. Obviously, it has to bind to the insulin receptor, or it's not going to work. And so what happens is, is that the receptor, the insulin receptor, there's either not well, not either, it's both there's not in many of them. And their structure has changed. And if that's the case, insulin is not going to bind to that very, very well, you're not going to have the same effect on the cell, so the cells will be resistant. Now, why the hell did that happen? The reason it happens is because we have a hypersecretion problem with type two diabetes. So much insulin is released. So much insulin is bombarding the cells that they're just done. They're sick and tired of binding the insulin, and they're just done. And so they will respond to it properly. Why would that happen? Most often, not always, typically, not always. Typically, due to a lifestyle problem, due to a poor lifestyle. Well, what does that mean? You don't exercise, you're not active. Exercise has been shown 18 and 18 gazillion studies. To increase the sensitivity of the insulin receptor, not decrease it, it does the opposite of resistance, during exercise, is you don't even have to exercise, you don't have to workout for five years to get the effects. You have to work out for five minutes, and you get the effect, it happens immediately. And then it stays with you. And especially if you're working out all the time, more sensitive, more sensitive, more sensitive. And so we want we want our cells to be very sensitive to insulin. Again, I told you before that the cells are being bombarded by lots of insulin. So for diet, eating the wrong kinds of foods that

keep on causing the release of insulin, especially really, really hard carbohydrate. But insulin is also released in response to amino acids and fatty so so just really, in the end, just eating too much thinking food. That's what's doing it because when you eat food, you stimulate the release of insulin and if you eat too much food all the time. This is what you get. Now, I said typically, that doesn't mean always. You can do everything right. You can exercise. You don't have to be overweight. You can live a proper lifestyle and you can still get it but that is incredibly rare. Okay, there's got to be some type of a predisposition for that to occur. That hardly ever occurs. The problem is almost always poor lifestyle. Unfortunately, The fortunate thing is, is that it can be managed. The fortunate thing is, is that when we have a type two diabetic diagnosis, before that there's something called pre diabetes. And how does it get diagnosed by the way? bloodwork, you take a fasting glucose. And what's that mean is that you don't need for 12 hours, you go, you get your blood work done, it gets sent to the lab, and they look for the level of a fasting glucose level, it should be below 108. Between 70 and 100 is normal. If it's between 100 and just under 120, then you're a pre diabetic, it doesn't mean you have diabetes, it means you're on your way. How do you think you could treat that and reverse it? Change your lifestyle, start to exercise, get, get more active, lose weight. That's all you have to do. I actually had a former student just call me about a week ago, didn't call me contacted me about a week ago. Like my student years ago, and she came up to me and she said, My dad is a type two diabetic, what can I do to help. And I said, You got to tell him to be more active, you

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got to tell him exercise, you got to tell him to lose weight. He lost 45 pounds. He's off his meds, all the students walk in, he's walking, and he lost 45 pounds. He's off of his meds. He's in his 60s. So it's not rocket science. I think when people hear you got to exercise, they think that you have to run a marathon, where you got to be some athlete, you got to you know, go into the gym, you know, five days ago, he had to do that. Walk around the block every single day. It doesn't take much. He does a little bit less food every single day. That's typically all you have to do to really change things turn things around. So when it comes to treatment, what's the treatment? The better lifestyle. Okay, as you put treatments, better lifestyle. Now, you might need meds. What kind of meds while they're in the notes, Metformin is one of them. I'm not too concerned about you know what it is, you could need insulin to depends on how severe it is. And it could be that if you're not taking care of it later on, you stop producing insulin, the beta cells were done. And now you're a type one diabetic, and now you're insulin dependent. So that could very well happen. But that's a severe form of it. But if patients are compliant, typically if the prognosis is pretty good, but there will come a point to where there's no return whatsoever. And there's not a darn thing that you can do about it. So what's so bad about you might think, well, big deal, like glucose in the blood. What do you do? Well, here's the problem. Problems with failing to treat diabetes mellitus, and it's the hyperglycemia, it's a high level of glucose and glucose levels are high, because you just can't transport it out of the blood. Glucose is just a carb doctor, or how bad can that be? These are the problems problems with untreated diabetes, and this could be type one or type two, you might not be compliant take your insulin the way this is supposed to. Typically, compliance though, goes with type two. And there's way more people with type two problems arise from really high levels of blood glucose constant. And that's again, hyper glycaemia. Now again, what do you do with just glucose? This is what I want you to think about when you have high levels of glucose. Now glucose at normal levels below 100, between 70 and 100. Not a problem whatsoever. But once the levels are high all the time, I want you to think of glucose as glass, jagged, glass, circulating through your circulatory system to your blood vessels. And so what this is going to do is it's going to cause inflammation and damage to your blood vessels. What's that gonna do? Well, it's gonna diminish blood flow to your tissues. What's that gonna do? Damage kill your tissues. Tissues become what's called necrotic. necrotic is a term used when tissues die, because they lose blood flow. Now this isn't the only reason it would happen. But now you have damaged tissue. Well,

what can that lead to eating gazillion things? People With diabetes go blind, why? blood flow to the eye sucks. You go blind kidney failure. So this can lead to the Oregon Oregon failure, kidney failure, blindness amputations, amputation amputation, what? Yeah. If your toes are dead, you have to cut them off. If your foot is dead, you have to cut it off. If your leg is dead, you have to cut it off. People who do not treat their diabetes properly will get their limbs amputated because their tissue is necrotic. It's dead. And so we got to cut it off. Organs are failing. They're dying. Oh, and by the way, cardiovascular disease too. So when you inflamed your blood vessels

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that leads to cardiovascular disease. People do not equate diabetes to cardiovascular disease type. Diabetes is one of the leading causes of cardiovascular disease creates blood clots because you're inflaming the tissue that can create atherosclerosis plaque formation. It's terrible disease. And it's an epidemic. We have an epidemic, we have a pandemic going on right now. We had a pandemic before the pandemic. It was this. You've heard me go on my soapbox stuff about, you know, lifestyle and preventative medicine. I'm not going to do that again. I promise I don't have time. So anyway, bad. Something else that can happen here. It's called diabetic ketoacidosis. So what we just talked about was that right there, so that right there is all this one last thing. Diabetic Ketoacidosis what is it? This is life threatening. So diabetic ketoacidosis, although they do call it diabetic ketoacidosis. But you don't necessarily have to be a diabetic to develop this. But this is what has to happen here with diabetic ketoacidosis. You have to fulfill two conditions. Two things occur. Number one, you get an increase in blood glucose. And you get an increase in blood ketones. People think that ketoacidosis is just high ketones. It's not and I've been asked what Dr. Or what about a ketogenic diet, ketogenic diet does not cause diabetic ketoacidosis. Because on a ketogenic diet, your blood glucose levels are actually really low. Your ketone levels are high, what's your blood glucose levels are really low, you have to have both of these happen for this particular scenario to take place. So let's talk about it. Why would this happen? So how does it occur? How does it occur? This is how it's going to occur. It starts it's initiated by

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really, really, really, really low insulin or no insulin. That's what's going to start it off. And so now let the story begin. So we're going to draw diabetic ketoacidosis. And again, this is life thread.

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So here's the cell, and it's specifically actually a liver cell. Because liver cells are the only cells in the body that can produce ketones. We learned that last semester at the end of chapter one. So here's a liver cell. Although other cells are going to be affected, ketoacidosis is developed because of what I'm going to show you in the liver cell. And so number one, really low insulin or no insulin. Now, I said as I was writing diabetic ketoacidosis that it doesn't necessarily have to be a diabetic that this happens to but most people who have diabetic ketoacidosis are diabetics most often most often a type one. Without insulin, their pump breaks down. They ran out, I don't know they've the power outage and they can't, whatever just they don't they don't have insulin, there can be scenarios where type one does not have insulin, or really, really, really high stress. And that can be anybody in this room could be me with a tremendous amount of stress distress inhibit insulin. Yes, it does already showed you that. That's one of the end of it. So if you have high stress, you might have little to no insulin

circulating in your body because it's inhibited by stress. So those are two things that can cause the butt stuff in. It's a type one without insulin, where you going to see this? Which is why they call it diabetic ketoacidosis. So what's going to happen here? Well

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they happen.

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You're not getting any insulin to the cell. Well, if that's the case, what's the cell not going to be able to do? That That ain't happening. But it's necessary for insulin, the car was necessary for the glucose transport, it's needed insulin. So because of this, number three, you cannot use glucose to make ATP. Why? Because you don't have any. The liver cells don't have glucose. Alright? That's okay. Well, not really. Because now what this liver cell is going to be forced to do is use fatty acids exclusively, to make ATP. Now. We learned last semester, that at rest, and most of the time, we are using fatty acids to make ATP but not a lot, because we don't have to, because we get so much ATP from fatty acids. But if you all of a sudden have to use them exclusively use fatty acids exclusively to make make my liver cell a little bigger to make ATP. When you do that, I don't know if you remember this from last semester. Beta oxidation, remember that in the mitochondria? Beta oxidation, we make ketones that his liver cells do.

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And so we produce a lot of ketones. So we're gonna have really high levels of ketones. And by the way, if you can't transport glucose out of the blood, what's going to happen to blood glucose

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is going to go up? Do you think the cell is going to keep all those ketones? It better not or is going to die. So six, we're going to transport a bunch of ketones into the blood by ketones, like glucose, diabetic ketoacidosis, is can kill somebody. One because the inflammation going on by the glucose number two ketones, diabetic, Kido, acidosis are acidic. And a pH is too low. And you're gonna start to denature proteins. And now you're screwed. And if this doesn't get fixed, this patient is going to die. How do you think you can fix it so easy? Give him insulin. So I got to do if this patient presented to the ER, and this just kind of started, they would seem drunk. Guys like ketone smell like, you know what acetone is? We make it that's a ketone in the body of the three ketones. Willams. Acetone, what's acetone smell, it smells like alcohol on it. This patient is going to come all loopy. The acidosis is going to make him loopy. And you're going to smell their breath. You're gonna say they're drunk. Put them in the corner. Let him sleep it off. Your patients dying. Don't assume anything. When you take care of people. You might get sued. You might kill somebody. All you got to do is give them insulin, and they'll be fine. In no time. There was one last thing that I can't talk about because it's 621. It's okay. It's this right here. But I will just we'll do it at the beginning of lecture on Thursday. I won't be here on Tuesday secret Well, these are all the stress hormones. I just tell you a little story about that won't take but a few minutes. Alright folks,

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Fri, 1/28 4:38PM 1:12:35

SUMMARY KEYWORDS

sperm, lh, happen, corpus luteum, estrogen, follicle, menstrual cycle, fsh, progesterone, hypothalamus, hormones, cells, called, uterine wall, meiosis, picture, arrows, embryo, draw, put

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Okay, folks, before we begin, the exam, obviously is on Tuesday, right? 60 questions on the exam, most of the questions are going to come from chapter two, because while we spent the most time on Chapter Two, I am going to be generous, I'm going to give you the entire lab time to take that exam. And so that's a minute and a half per question. That's a lot of time for those questions. And so you'll be able to take your time as you take that exam, please get help if you need it. The material tonight is going to be the last material that's going to be included on that exam. So let's start let's talk about the male reproductive system, and then we'll do the female. First thing we're going to do, we're going to talk about how males produce their gamete. And that's going to be the sperm. And we know that it happens in the walls of the seminiferous tubule. I'm pretty sure that lecture receiver talked about that with you. And I know you guys don't want to seminiferous tubules are. And so what we have here is just a simple cross section of the seminiferous tubule. Most of that is wall right there. And then that lighter pink shade stuff in the middle, that's the fluid. So what I'm about to draw on the screen is the darker shaded pink is going to be the wall of the seminiferous tubules. And all these little purple circles are the cells that we're going to put on the screen in just a second as we go through spermatogenesis. So let's draw all those words that you see right there. We're going to start with the stem cell and that stem cells called Get spermatogonia. So I'll write that here. Now this spermatic, Odium is going to go through division, and division and division and division and division, mitosis, I'll put next to its stem cell, just to remind you, and I'm just going to draw that. And so it's going to go through mitosis. And as we know, when you go through mitosis, you're gonna get two identical cells. And that's what we're going to get, however, we're going to change the story just a teeny bit. So I'll put an under spermatic conium. Over here. On the other side, what I'm going to do is we're going to put a self on a primary spermatocytes, because we are going to get another spermatic gonia. But one of these cells is going to differentiate into what's called a primary super medicine, I'm just going to skip that step. It's okay. And I'll put mitosis over there. And so this is going to be a different cell, a primary spermatocytes. And I'll write it next to it. And again, let's remind ourselves that this is mitosis, we're going to see meiosis in just a second. So that's the primary spermatocytes. Now, what I'm going to do is I'm going to put a dotted line right underneath that's romanticized. And what that dotted line is going to represent is puberty. You know, right puberty right there. So what we're seeing up into this point is happening continuously doesn't stop. I mean, it won't, for a very, very long time up into this person, this, this male is very old. But then once we reach puberty, what's going to happen is, is that we're going to be allowed to move forward from this step, we're not going to move forward from this step until we hit puberty. And the reason is, because our testosterone levels are going to go up. And so I'll put that over here at puberty, we're going to get an increase in testosterone. And until we get an increase in testosterone, we're not going to be able to produce sperm. And so now that we've hit puberty, let's see what's going to happen. Now we're going to go

through meiosis. And so what this slide is going to represent is meiosis one, I don't know if you remember meiosis, there's two cell divisions, there's meiosis one, and there's meiosis two, I'm not going to get into the details of meiosis is not important. But I will put here meiosis one. And later, meiosis one is a cell division. So we're going to get two cells from this. And both of those cells are going to be called secondary spermatocytes. So I'm going to draw two cells now. So here's one of them. And here's the second. So those two cells used to be that one primary spermatocytes. And so now we have secondary spermatocytes. Both of those cells. Gotta make it plural. Then we're going to go through a second meiotic division, each of those cells is and so that's meiosis right there. That's meiosis right there. And it's called meiosis two.

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Now in your notes, you're gonna see diploid haploid. And I don't care. I don't care. I just threw him in there just to throw them in there. I just need you to know what cell becomes what what division is happening, where, why it's happening. Well, we have an increase in testosterone. Each of those secondary spermatocytes is going to divide give us two cells. Now we're going to have a total of four cells. Sure you remember that with meiosis, one cell becomes four cells. So here are the four cells. There's two from that one, secondary spermatocytes. And here's two from the other secondary spermatocytes. We call those spermatids. And again, this is happening in the walls of the seminiferous tubules. Let's not forget that. So those are spermatids. Now what we're done dividing, now these spermatids are going to mature. And so that's what I'll put over here. So that's going to mature that's going to mature so is that one, so is that one and I'm just going to put the word mature there. It has nothing to do with cell division. And each of these spermatids is going to become a sperm. And so I'll just draw sperm as best as he can draw sperm. So I'll put a little flagellum on there. acrosome all the stuff that you learned about with lecture, Siebert. And so we have sperm for sperm, one primary spermatocytes Then what's going to happen is, is that the sperm just going to be plopped right into the fluid. And then they're going to go, they're supposed to go and actually see her talk to you guys about the journey of sperm. So there you go. We're actually going to come back to this picture a couple of times, as we talked about some other things in the lecture today. So why do we make sperm recently make sperm is so we can fertilize a secondary o site and a female so that we can perpetuate the species. That's the reason that we produce sperm. So we have to deliver it into the female reproductive track. So that's the next thing we're going to be discussing. How do we do that. And so now we get to talk about Erection and ejaculation. So in order to do that, the sperm has got to be in semen. Semen is going to be the vehicle you guys talked about semen with Lex receiver, so you know what semen is comprised of, we're not going to worry about it here. And so let's first talk about erection. How does this happen? And so we're going to go through a little flowchart here, the same thing with ejaculation. So erection here. And so let the story began. And the story is going to begin the story is going to begin with stimulation, I think we probably know what that is. I'll put it in quotes. Now what's that going to do, what that's going to do is going to stimulate, activate or increase the tone of the parasympathetic nervous system. So I'm just going to draw an arrow with the positive sign, indicating that we're going to be stimulating the parasympathetic nervous system and I'm going to write parasympathetic nervous system here just parasympathetic. And so actually even number everything. So number one, stimulation number two, stimulate the parasympathetic nervous system. Now as we know from last semester, when we studied the autonomic nervous system, we know that the parasympathetic nervous system has the pre postganglionic neurons, and then certain neurotransmitters are released. And one of them we know for sure is acetylcholine, at least I hope we remember that from last semester, because I hammered you guys with it. But that's not the only neurotransmitter that the parasympathetic nervous system can release, I didn't give you the entire story last semester. Acetylcholine is going to be one. And then we're going to have another one that's a gaseous neurotransmitter that we actually also talked about last semester, nitric oxide. So what I'm

going to do now is I'm going to draw a red tube. And you guys know what a red tube is, when I draw a red tube, it's going to be blood vessels that I'm going to label it. So blood vessels have erectile tissue, I'll just keep it simple like that. Now in the notes, you might see the word sinusoids. I guess you guys didn't talk about sinusoids. And so just don't worry about it just blood vessel. sinusoids are kind of special blood vessels, you don't have to worry about it just blood vessels. And we know that these blood vessels are wrapped by smooth muscle, we know that we learned last semester. And that's going to be the target of these neurotransmitters. And so we're going to control the tone of those smooth muscle cells was sent is going to control the tone of these blood vessels. And we're going to do that well because we're going to release these true neurotransmitters and if you remember from last semesters, little chicken's feet represent chemical synapses. And so we're going to have our neurotransmitters released onto those smooth muscle cells. And I'll label what each of these neurotransmitters is this one over here, acetylcholine, this one over here. Nitric oxide and if you remember from last semester,

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what's going to happen now is is that the smooth Basile is going to relax. And when smooth muscle relaxes that's wrapped around the tube, what's going to happen to the tube? It's going to dilate, it's going to get bigger. And so number three is the release of these neurotransmitters. Number four, is we're going to get dilation, dilation of those blood vessels. And when you get dilation of a blood vessel, certainly you're going to increase blood flow. And so number five as a result of the dilation of the blood vessels, we're going to get an increase in blood flow. That blood is going to get trapped. It's going to increase pressure. And numbers six, well, we have our direction. And so that's really all I need you to know. Again, the word sinusoid in the notes, don't worry about it. So what's next, the mission, the jack Ulation. So let's talk about those things. So we're going to continue our little flowchart here. And we're going to start with number seven now. And so number seven, we'll just call it continued stimulation, or just stimulation. And so now what is that going to do? Well, we're gonna start now we're going to stimulate another division of the autonomic. Now the sympathetic nervous system is going to come into play. And so once again, an arrow with the plus sign number eight. And I'm going to put sympathetic here. Now what the sympathetics are going to be responsible for emission, we'll talk about what emission is. So I'm going to just write emission over here, and then I'm going to explain to you what it is. So what is the mission emission is the mixing of the sperm with the semen, that's part of the mission. Mission is also taking the semen which now has sperm in it and discharging it into the prostatic urethra. And you guys know what that is, right? That's the little tube that's going right in the middle of the prostate gland. If we discharge fluid into a tube, what's gonna happen to the to go stretch, so we're going to stretch the prostatic urethra. And all of that is happening with the mission. All right, and the sympathetic nervous system is responsible for that. And so now, as a result of that, when we stretch the prosthetic urethra, it has connected to it sensory nerves. So that urethra has these receptors these autonomic receptors are called Mecanim receptors. I talked about him last semester. And when they detect stretch, they stimulate the sensory nerves and the sensory nerves are going to take that information to a certain part of the spinal cord. And so that's going to be the next thing I write down here. So sensory nerves which are made up of axons, of course, conduct action potentials, to the ejaculatory reflex center of the sacral spinal cord. We put that all in one line of the sacral spinal cord. If you go back to your notes from last semester in the autonomic nervous system, I talked about reflex centers and one of the reflex centers they talked about was ejaculatory reflex center in the spinal cord, go back and look, it's there. I'm showing you another autonomic reflex. This is a third one of this chapter or not this chapter but this section because lactation involved in autonomic reflexes, remember that and so to childbirth, go ahead and look at the notes it's there. So another autonomic reflex here. And so now we're going to go to 11 now and we'll put it up here because we kind of ran out of room over there. So now number 11. So

now the reflex center is just a bunch of neurons we know that and those neurons are now going to respond with motor output but the motor outputs going to be somatic and so now we're going to have motor I should put somatic motor somatic motor nerves got its motor so without somatic controls what by the way motor what are we going to what are we going to control somatic nervous system? What kind of muscle skeletal muscle so somatic motor nerves

14:34

conduct EPS action potentials to skeletal muscle at the base of the penis Of course he's skeletal muscles are going to contract and so number 12 Those skeletal muscles contract rhythmically. And when they contract, what they're going to be doing is that they're going to be squeezing the tubes, increasing the pressure within the tubes and forcing the semen out. So 13 There you go. We've just delivered our spur. So what's involved here when it comes to erection, and a jack Ulation inhibition is included in this story. erection is parasympathetic. And then sympathetic is going to be involved with the mission, the somatic nervous system is going to be involved directly with the ejaculation. The sympathetic is still involved because it must be given that what's happening here on the left hand side last semester, if you remember when we were talking about the functions of the autonomic nervous system, the parasympathetic and the sympathetic, I had you guys know, you know, the sympathetic desist parasympathetic does that we talked about point and shoot, remember that? We're gonna talk, we're just gonna mention it again. But I got my friends Stewie to help me with it this year, or this semester. So again, point A direction. P, they both start with a P, that's an easy way to remember it. Last semester, I just talked about shooting ejaculation. Sympathetic starts with so to shoot. But last semester, I didn't talk about the somatic nervous system, because we were just discussing the autonomic at the time. So we are just adding something here. So parasympathetic reaction, sympathetic and somatic ejaculation and a mission as well, again, because the mission is very important to the story. All right, yes.

16:51

Number one, you know, when you started after, like, you know, pretty much fromages Then that process occurs.

17:02

You talking about this? Number one, I hear it a lot. And are you tying it in somehow to this? Yeah. Yeah, the sperm are already made. So once the sperm has already made, then it does the stipulation for? Well, I mean, you don't necessarily have to have sperm for any of this to happen. You don't have to have sperm in your semen. But we're just assuming that it's there. But this this is not necessary for any of this. No, not at all. Not at all. We're just assuming that you everything's working fine. And everything is good. Yep. Thank you. You're welcome. Okay, so we take care of that. Now what, let's talk about some hormones, hormones that we've already discussed, by the way. And so what we're going to do is, is that we're going to take those hormones. And we're going to draw a picture, a picture that we're familiar with. But I'm going to put a couple of extra things in this picture that we haven't yet seen, but you have actually seen them with Siebert, I'm just going to add a little bit more to it myself. So we're going to start with the hypothalamus. And I'll write that right here. And the hypothalamus, of course, releases a bunch of releasing and inhibiting hormones, one of which is GnRH. And so that's going to be part of this story. So there's our gonadotropin releasing hormone.

And we know that that particular hormone targets the anterior pituitary, and it controls the uniting hormone and follicle stimulating hormone, we know that. And so here's the anterior pituitary. And I'll label it and it in response to GnRH is going to release luteinizing hormone and follicle stimulating hormone what we learned about those hormones. And we know the luteinizing hormone causes testosterone production and release in the testes. And we know that follicle stimulating hormone causes spermatogenesis. We learned it already. But we're going to repeat it. And what this is going to represent are the testes. So I'm going to make it plural. So that's both testes. Now, I'm going to put an imaginary line right there. This is not really the way it is anatomically but visually, it'll work for us. Over here on the left hand side, I'm going to put the word Leydig, or lighting, lighting cells, interstitial cells of lighting, you guys know about those because you learned about them with lecture receiver. And then the other cell that we need to talk about here are the sertoli cells. The target of LH late excels which is why I'm going to draw the arrow there. FSH target of FSH sertoli cells will lady cells do produce a release testosterone. So how does that leech cause testosterone production and release? Does it be the late excels? And so then I'm going to write the word testosterone here. So our testes produce and release testosterone insulating cells that do it in response to LH. What about FSH? What is it going to tell the sertoli cells to do make sperm. So there's spermatogenesis, which is the first thing that we talked about today. So now let's add to our story in the first picture. So let's go back to the first picture.

20:26

And let's do this. And I'm going to put over here sertoli cells. And I will put over here Leydig cells. And then what I'm going to do is I'm going to point to the Leydig cells.

20:47

Because they are controlled by regulated by LH, I'm going to point to the sertoli cells. I'm going to put the hormone FSH and by the way, these sertoli cells are intermingled with all of these cells that you see here up on the screen. Now I need to do one more thing to make this story complete on this page. And that is why I do that. It's a testosterone that is going to be stimulating those sertoli cells along with FSH. So in order to make the next picture, complete this one right here, I have to draw an arrow from the hormone testosterone to the sertoli cells. So everybody works together so that we can make sperm. Now the story is not quite complete, because what I have to do here is the sertoli cells actually make a hormone Leydig cells make a hormone testosterone sertoli cells make a hormone is called inhibit. And inhibits target is the anterior pituitary, and it specifically inhibits FSH. Now why would it do that? It's just negative feedback. Just so we don't release too much FSH, that's what that's about. So it's just a negative feedback inhibition. One last thing I want to put here up on the screen with testosterone, when it comes to testosterone, not only is its job to stimulate the sertoli cells to produce sperm, but it's also responsible for secondary sex characteristics in a male. And so I'll put that here. And I'll show you where that is in the notes, secondary sex characteristics. Now, what does that mean? secondary sex characteristics are when a little boy becomes a little man. So he's going through puberty, and his muscles start to get bigger, his voice gets deeper on the reason is your vocal cords are mostly skeletal muscle, and so they get thicker. And that's why your voice gets deeper. And a number of other things occur as well increase in sebaceous gland activity, bones get thicker, you have growth spurts, all these things again, that are happening at puberty to cause that, again, little boy to become a little man. And so where is that in the notes? It's right here. So just aspirin allows production of sperm just showed you causes development, a secondary sex characteristics, here, some of them. And so all of that is part of that picture. And that's it for the male

reproductive system, fairly simple. female reproductive system, different story a little bit more complicated. We're going to tackle it the exact same way. So I just showed you how the male gamete is produced sperm. To start, we're going to do the same thing when it comes to female, we're going to come back to this picture a number of times. So there's a lot of words in this particular in this particular picture. And so it's going to be more vast picture than what we saw with spermatogenesis. So we're going to now do oogenesis. We're going to make some parallels between spermatogenesis and oogenesis. We're going to see some similarities. But we're going to see more differences between the two. And I'll point them out as we go along. So that this story begin now. So this is now oogenesis. At birth, and so that's what I'm going to put over here. So this little girl is just born at birth, that little girl is going to have roughly 1 million follicles. They're called primordial follicles. I'm going to label it over here to the right, 1 million. I'm just going to say oh sites, because that's really in the end, what's going to matter. Oh site per ovary. There's two ovaries, so roughly 2 million o sites. And each of those o sites could be fertilized by a sperm. So that's the those are the eggs. And so over here to the right, I'm going to draw the O sites but they're going to be housed by what's called a follicle. So this is a follicle is called a primordial follicle and again, the little girl is born with these So this is not a cell that I'm drawing here, it's not a cell. It's going to house a cell, the O site is the cell. And so I'm going to put the O site and the follicles, so that's not a nucleus. That's the O site. And I point to it. And again, and there's a, there's a million primordial follicles per ovary as well, because each and every Oh site is housed by a primordial follicle. And the follicles job is to nourish that oh site, protect the O site, and help it develop. That's the job of the follicle.

25:36

Now, as this little girl is 1-234-567-8910, somewhere around 11, she's going to start losing an awful lot of those follicles. And all sites actually, she's going to lose more than half of them. And so what I'll do here is, is that I'm going to draw a line from here to here. And I'm going to draw another follicle but it's going to have a different name now. And so here we have another follicle. And there's still the O site, in the follicle. And so at puberty so roughly no 1011 12 Somewhere in there. Now we're down to about 400,000 Oh sites, again, along with the follicles per ovary. So she lost over half of her follicles and over Oh sites. Why these are the best ones. It's survival of the fittest. What happened to the other ones, they're gone. These are the best ones out of the bunch. It's okay, she lost over half. These are what she could live to be 5000 years old. She's not using all these. She has plenty. She's got almost a million. It's again 400,000 per ovary, this two ovaries. So there is still plenty. Now what are these called? This is now called not a primordial follicle is called a primary follicle. So I'll put it here. And that is now called a primary Oh site. That's something else I'm going to add over here at the top. It's about meiosis. When it comes to a male, you get enough testosterone at puberty and off to the races you go, you're just gonna start making sperm sperm spread, you can make sperm, if you as long as you have enough testosterone in the mail, you're gonna make sperm, you can be 120 years old. And if you start strong, those are high enough, you got to keep on making sperm, female, completely different story. She's born with all the OSI she's ever going to have in her entire life. Males, we keep on making females, she's made over it. When it comes to meiosis, this oh, say here, this first oocyte here, meiosis one has started. But then it stops starts. But then it stops. I'm going to show you how you complete meiosis one. But it hasn't happened yet. So now she's reached puberty. What that means is that she's going to start to have menstrual cycles. And this is part of the menstrual cycle. And when we go over the menstrual cycle in detail, I'm going to come back to this picture a number of times. And so now because she's reached puberty, and our very first menstrual cycle is going to happen. And from that point forward until of course, she reaches menopause where she won't have anymore. This is how it's going to work. So at the beginning of each month, so at the beginning of each month roughly about roughly a dozen follicles, primary follicles, and the O sites, they're always together, start to mature. But only one of them is going to survive. We're not getting

into twins, we're not going we're not doing that stuff. We're just going to assume no twins, just one follicle makes it one oh site makes it so roughly a dozen of them start to mature, but only one survives. And so that's what this line is going to represent. Is that. So what are we going to get in the end, we're going to skip a step. Because what's going to happen is during this maturation process, we're going to get a secondary follicle with a secondary Oh sided and it becomes what I'm going to draw here, which is what's in your notes. It's a super duper secondary follicle. Let's call it graph E and folic So I'm going to, I'm going to write that down. So this is a follicle.

30:06

And it's a graphene follicle. It's supposed to have two A's, by the way, with a secondary o site. And so there's our secondary o site because it is mature.

30:26

Now, I told you that meiosis one started, but then it stopped. This is what's going to complete meiosis, the secondary o site getting to this maturation point. So the meiosis one is going to be completed. And so I'm going to write that here. So over here, meiosis one is completed.

30:46

So meiosis one completes meiosis two starts, but then stops. And I'm going to tell you what allows meiosis to to be completed.

31:08

So far, so good. I'm going to add something here. When it comes to the maturation from primary follicle, the graphene follicle is hormonally driven and want to hormones are going to be involved with this luteinizing hormone and follicle stimulating hormone. And so I'm going to put LH and FSH right there. And actually, we're going to come back to it because well, you'll see. So far, so good. So now we have a graphene follicle with a secondary Oh site. And that's secondary Oh, site is D O site that can be fertilized by sperm. And so let's get into that. So we have to go through some more steps I ran out of room. So now let's go to the next page. And so over here, I'm going to write at day 14. Now, what the hell does that mean? Well, we are assuming again, this is part of the menstrual cycle, we are assuming a 28. Oops, a 28 day menstrual cycle. Of course, menstrual cycles can vary widely, but on average is 28 days, right? The middle of 28 is 14. So at day 14, this is what's going to happen. So I'm going to draw the graphene follicle again. And what I'm going to do when I draw this graphene follicle is going to put a hole in it. So there's a hole in the graphene followed, because it's going to rupture. And when it ruptures, that secondary oocyte is going to leave. And so at day 14, graphene follicle ruptures.

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And the Oh site is a secondary Oh site is released. It also leaves the ovary. And actually, you know what I'm going to I'm going to reword that a little bit. I'm going to say that the O site, and I'm going to put secondary aside, just to remind you, secondary o site ruptures from the graphene follicle and not

put secundario aside, just to remind you, secondary o site ruptures from the graphene follicle and not only the graphene follicle but the ovary, I do assume that you guys know that this is happening in an ovary, right? So what I'm drawing up here is occurring within the ovary itself one of the two. And so again, not only is that going to leave the graphene follicle, it's leaving the ovary. In where is it going? Well, you guys already know. It's going into the fallopian tube. Why does it want to get fertilized? That's why. And so now we had the secondary Oh side over here, and one of two things are gonna happen. It's either gonna get fertilized, or it's not going to get fertilized. And by the way, that secondary o site is viable for about 24 hours. What does that mean? It means it's not very patient at all. If the sperm don't get there in time, it doesn't care. It's going to leave, it's going to disintegrate, it's going to be gone. And that's it. So a female can get pregnant one day of the month. And that's it. Now we do know that sperm can remain viable within their female reproductive tract for what days to almost a week just depends. That matter how long the sperm are there. It matters. I mean, it matters because there's a greater chance that the sperm are there for a longer period of time. So if they had sex at let's say on a Tuesday, and let's say she ovulate on by the way to Scott ovulation right, let me add that here. That's called ovulation if she ovulate on a Thursday, two days later, the sperm are still alive. She can get pregnant on that Thursday. Okay, so the bottleneck is the secondary Oh site. It's not the sperm. Again, one of two things can happen, you can add to get fertilized or not fertilized. And so let's put that in the picture. So oh site. No fertilization is this arrow is going to be so no fertilization, the sperm didn't make it in time, or there was no sperm. So fertilization, what's going to happen? It's going to die. Oh, sigh just goes away. It's done. What if there is fertilization, and that's what this arrow will be.

35:39

And so this is with fertilization? Well, we're going to get a single cell. And that single cell, actually, I'm just going to write it. That single cell is called a zygote.

35:56

So when the sperm and the secondary Oh sigh combined with each other and the DNA come together, we have a zygote. That's one cell. As soon as that cell divides as soon as it goes from one cell to two cells. We call it an embryo. And then those two cells divided become four and then eight, and then 16, and 32. And exponentially. Well, the thing grows and grows and grows and grows and grows. And what does it become? Well, as soon as it's two cells, it's an embryo. And up to about two months, it's an embryo. Beyond two months, it's a fetus. So what happens with fertilization? Well, that happens with fertilization. Meanwhile, what happens to the graphene follicle that goes through a transformation, it becomes something else. And what does it become? It's gonna become something called a corpus luteum. And that's going to be an important thing to talk about after it's been introduced here. So that's a corpus luteum. The job of a corpus luteum is to produce a lot of estrogen and progesterone. So I'm going to put that here.

37:11

And by the way, when it comes to the graphene follicle and the O site rupturing from it, that's driven hormonally as well. And so what drives ovulation what drives ovulation? is once again LH and FSH, a lot of it. Put four arrows here. LH, FSH, mainly LH and we call those surges FA FSH surge, LH search. I'll put some arrows over here, when we get into the menstrual cycle. So we're going to come back to this we're going to come back to this picture a number of times because once again, it is part of the

menstrual cycle. When it comes to the corpus luteum, it is always going to come about the Grampian follicle is always going to turn into the corpus luteum whether it is fertilization or not. So this is fertilization or no fertilization. fertilization or no fertilization, it doesn't matter. This is going to happen regardless, you're always going to have a corpus luteum always. Now, if this fertilization is going to stick around, if there's not fertilization, it's not going to stick around. And we're going to talk about that. But not yet. Let's take a break. When we come back, our story shall continue. Alright, folks, here we go. We'll finish up. So now what are we going to do? Well, let us talk about this corpus luteum. A little bit, let's talk about fertilization and no fertilization and compare and contrast. A couple of things here. So I will put here on the left hand side fertilization, so it has occurred. And over here on the right hand side, no fertilization. Now if we have fertilization, obviously, we're going to have an embryo. If we don't, obviously, we don't have an embryo. So let's draw the embryo here. So there's our embryo and down here, we're going to draw corpus luteum. Now, as I said, the corpus luteum, his job is to produce a lot of estrogen and progesterone. And so I'm going to put both of those home runs in the picture now. And the reason is to keep that embryo alive, that progesterone and estrogen is gonna thicken the uterine wall is going to allow for implantation, it's going to allow that uterine wall to be nourished, it's going to be very well vascularized, the embryo is going to be within that area, and that within that uterine wall. And so those two hormones are vital for that embryo to remain viable, so keeps it alive

40:24

the embryo is going to do something for the corpus luteum. So the corpus luteum produces those two hormones, the embryo is going to produce its own hormone. And hormone is going to be producing is called Human chorionic gonadotropin, otherwise known as H, C, G. And this hormones target is the corpus luteum. And it keeps it alive. So they're keeping each other alive, they need each other. keeps it alive. They live symbiotically with each other. Until the umbilical cord is formed at around two months, then they don't have this kind of symbiotic relationship anymore, because well, it's the umbilical cord that's going to keep at that point, then the fetus alive, because beyond two months is considered a fetus. What about no fertilization? Well, if we have no fertilization, we have no embryo. We have no embryo, we have no hCG. If we have no HCG, corpus luteum is going to die. There's no need to have a corpus luteum. There's nothing to keep alive, there's no embryo. So eventually, that corpus luteum is going to go away, it's it's still present, it'll take a little bit of time for it to die. But it's going to because it has nothing to keep it alive. So please notice, one other thing, too, is this HCG here, that's what you detect in a pregnancy test, it is going to be excreted by the kidneys, it's going to end up in your urine, you're gonna pee on a stick, or whatever. And there's gonna be a reaction and they're gonna detect whether or not HCG is president used to be that it would take even like 10 years ago, almost a week, for the level of ACB HCG to be high enough for those tests to detect it. Now, the testes are incredibly sensitive. And within like a day now, you can detect levels of HCG in the urine that would indicate whether or not somebody is pregnant. And so where is this by the way, in the notes, let me show you. So this, this Oh, one of the thing to pull her body, I don't talk about it. And the reason I don't talk about is because they have absolutely no function whatsoever and they disintegrate. So don't worry about polar body. They're never around. But everything else you see here, every single word here, those two pages plus the third one I just drew drew plus this over here. So there's our human chorionic gonadotropin, by the way, there are specific cells of the embryo, they're called trophoblast. cells that produce hCG. I don't care really. pregnancy tests detect HCG in urine. And that's where that is. All right. Now, estrogen really quick. And progesterone, just a couple of other things. We're going to see it again. And when we talk about the menstrual cycle, so helps regulate the menstrual cycle, house mag really regulate the menstrual cycle, both of them do. And I just added a couple of things here. Progesterone affects the mammary glands during puberty, so helps with the development of the mammary glands. Estrogen, responsible for the development of

sex and secondary sex characteristics. So testosterone in males, turns a little boy into a little man, estrogen, little girl into a little woman. Increased deposition. So these are the kinds of things that estrogen is going to do. They also are that they will Yeah, this day, stimulate enlargement of accessory reproductive organs. Those are things that you talked about with X receiver. So that's some additional stuff with estrogen and progesterone. But we're going to see additional things on top of this when it comes to the menstrual cycle. And so let's talk about the menstrual cycle. There are three phases to the menstrual cycle. And we're just going to take them one at a time and these are going to be in chronological order as to how they occur. We are going to assume a 28 day cycle again, it could vary widely. But 28 is average. And it's a nice even number. And so we're going to use it. And so now let's go through the menstrual cycle. Mensis. So days, one through five is what we're going to assume. And so what's happening here is this. We're going to have really, really, really low level cuz I don't want to know if I want to go quite three, I'll go three arrows. It's not that terribly low, so estrogen, and really, really low levels of progesterone.

45:13

Now why on earth would our estrogen levels and our progesterone levels be so low? And this is the exact same thing we're going to see at the very end of the menstrual cycle, but I'll just give you the punchline right now. The corpus luteum is dead. If you have a menstrual cycle, you have to assume that you're not pregnant. You're not pregnant. There's no embryo, there's no embryo, there's no HCG, there's no HCG, there's no corpus luteum. If there's no corpus luteum Well, you're not producing a whole lot of estrogen, progesterone. You can't have a menstrual cycle. If you're pregnant, it's not possible. Now, can you bleed during pregnancy? And maybe does it mimic a menstrual cycle? It does, but it's not a menstrual cycle. Anyway, so I'm gonna put in parentheses here, why the corpus luteum is dead. disintegrated. And when that happens, all of a sudden your estrogen levels plummet, your progesterone levels plummet before this. They were high. And we're going to see that when we get further into the menstrual cycle. So now what especially because of the low level of estrogen, we are going to get constriction of the blood vessels of the uterine wall. So constriction have uterine wall, blood vessels. And these blood vessels are responsible for maintaining the uterine wall, and the uterine wall is much thicker now, then would have that it was at another point in time. And why is the uterine wall thick, because it was preparing for an embryo to implant in it. But it didn't happen. Because no pregnancy occurred, no fertilization occurred. So there's extra tissue in that uterine wall. And now there's less blood going to that uterine wall. And so because of that, the extra tissue is going to die. And that's by design, this isn't by accident. And so constriction of uterine wall blood vessels causes the death of extra uterine wall tissue. Well, now what's gonna happen? Well, if it's dead, it's not going to stick around, is going to rip away from the wall. And if you rip tissue away from tissue, what's going to happen? You're going to bleed. This is why you believe you're going to get rid of the extra tissue, and there's going to be some bleeding. And so the wall sloughs off in bleeding occurs. How much just depends. So that's one thing that's happening during menses, what else is happening during menses, now, we're going to visit some other hormones. So estrogen or progesterone to hormones, now three more, and that we're very familiar with, by the way. And so at day one, what we're going to do here is we're going to put our hypothalamus which as we know, very, very well by now produces and releases GnRH. Its target is the anterior pituitary, of course it is. And so here's our anterior pituitary. Which release is of course, in response to GnRH, LH and FSH, we know that. And this starts on day one. So the level of LH and FSH just slowly start to creep up. And what I'm going to do here is I'll just put one arrow, one arrow, it's not very high at this point, it's going to slopes up slowly. This happens in the first very first day, each month of a menstrual cycle, what's that sound like to you? What kind of release first day every single month, it's programmed, cyclic. And because of that, that cyclic and because of that, that cyclic release program. That's it for masses. That's

what's going on from days one through five. Now we're going to go to the next stage of the menstrual cycle, and the next stage of the menstrual cycle is the proliferative phase. And so let's go to the next page. So here is our proliferative phase. And this is going to be assumed to be stay six through 13.

50:13

So what's going to happen here GnRH is going to continue to go up a little bit. LH and FSH are going to continue to go up a little bit. And so instead of one arrow, I'm going to make it two arrows. And so I'm going to put them right in the middle. So we had two arrows up when it comes to LH, we're going to have two arrows up when it comes to FSH. And there's two things we're going to concern ourselves with when it comes to LH and FSH and what that's going to do. And so we're going to go left and right with this, we'll be here. We're going to say that we're going to be stimulating the primary follicles to mature into eventually a graphene follicle not going to make it plural because only one survives these primary follicles, how many about how many? About a dozen? Right? I am repeating this? So how many arrows should I put next to LH and FSH over here to to match these two. This is the proliferative phase causing the primary follicles to eventually one of them mature into a Grampian follicle. So two arrows up primary follicle to graft I told you this is part of all Genesis, or I'm sorry, part of the menstrual cycle, all tied together perfectly. What else? Let's go to the right. So we're gonna affect another hormone here. And the hormones estrogen. So LH and FSH rising is going to cause an increase in estrogen. And at this point, the level of estrogen is really low. How do we know that? Well, because I showed you over here during menses it's really stinking low. Now it's gonna start to rise slowly. Why? Because these two hormones are going to cause it to rise slow. And it's going to just trickle up, Trickle Up, Trickle Up, Trickle Up, trickle up, until it hits this magical point. And all of a sudden, it is going to shoot up to the sky. And the estrogen levels are going to be really high. And what causes that estrogen causes that? Not LH and FSH, LH and FSH did their job, they raised estrogen a little bit. And estrogen just keeps on going up. And then all of a sudden, estrogen says, You know what, let's go. And estrogen starts to stimulate itself up, up, up, up, up, up, up, up up, what's that called? What kind of feedback is that called? Positive feedback. So I'm giving you another positive feedback mechanism. First one was oxytocin. Second one was prolactin, oxytocin with with lactation, and childbirth, prolactin with lactation. Now I'm giving you another one. This is all about positive feedback. And by the way, that's called a surge as well. So that's an estrogen search. And what that estrogen surge is going to do is caused the LH and FSH search. And so now oops, wrong one. Yes. is going to cause LH and FSH to skyrocket. So three arrows. And so that's a search. That's a search. I'm actually going to show you a pretty picture of this. So I'm pilot.

54:15

It's a great picture. This picture is using every stinking book I've ever seen. So what we see in this picture on the top in green and orange, those are the levels of LH and FSH. And then what we have over here is the hypothalamus. GnRH getting released to the anterior pituitary. So that's what this little bit over here, but in green is LH and the level. So the y axis here is the level over here, this is time and the time is 28 days. Over here in this little area here, this is oogenesis. You see rupture of the secondary o site from the graphs you can follow right there. So we see all ovulation the blue one here that's the level of estrogen through the month purple one level of progesterone through the month, and then down here. That's the uterine wall. And we can see how it's getting thicker and thicker and thicker and thicker, until of course it sloughs off that Mensis. pretty picture. And so let's look at our estrogen surge. It's happening right here. So it doesn't show very well we hear how we have a slow increase in LH and FSH. It's actually a crappy stuff, the best resolution, there's actually a

little slight dip over here. Don't worry about it doesn't matter for us. Just go by what I say. Here's our estrogen. It's really low over here. Progesterone is really low over here. Why does it there's no corpus luteum. And then the estrogen is going to slowly climb until it reaches this magical spot and then boom, off to the races that goes. Estrogen is rising quickly before LH and FSH are rising quickly. So the estrogen surge is responsible for the FSH and LH search. Okay, the resolution of this one isn't fantastic there, there are better ones. I grabbed this one because this came from a book that we used to use in the course that nobody ever bought. And so we just don't. That was the book that I suggested in the syllabus, from McGraw Hill. But literally, I think a student has bought the thing and yours because it's wildly expensive anyway. But it's a nice colorful picture to kind of give you an idea of what's happening in a cyclic manner during the month and you look at it, just you can just see the cycle happening, surge, no surge up, down, up down, just very, very specific points in time during the month. So anyway, go back to the proliferative phase. And what we just drew is all this stuff, right there. And as that estrogen goes up, it's also helping to thicken the uterine wall, progesterone is going to start to go up a little bit, it helps thicken the uterine wall. And we get our again, LH and FSH search. But ovulation hasn't happened yet. Ovulation doesn't happen until day 14. And we've made it up to day 13. So LH and FSH are going to go up just a little bit more. So in the next picture, which is going to be the secretory phase and the luteal phase. I'm gonna put four arrows up, just as I did over here. And so now let's do the next phase. And that's going to be days 14 to 28. So the luteal phase or secretory phase, whatever you want to call it, both are correct. And so that's going to be days 14, to 28. Of course, on day 14, ovulation is going to occur. Now there's going to be three different phases within this phase, there's going to be an early phase, a middle phase and a late phase. So we're just gonna have three columns. So we're going to put early over here and just a hard line, middle over here, hard line.

58:01

And then laid over here. Now early, what happens? Well, we continue with our search, it's going to go up just a little bit more. So just one more arrow up.

58:17

And we'll just we'll call them surge again. And what that's going to lead to is, of course, ovulation. And so the secondary cause is going to be graphing follicle is going to rupture from the ovary itself is going to sit in the fallopian tube 24 hours, and it's going to hope to get fertilized. And that's early. I mean, that's that's, that's what marks the luteal phase is ovulation. When that happens. That's the luteal phase. We still have a corpus luteum. Of course we do. We're going to I should say not of course, we still have it. We're going to have it because the graphene follicle is still around here. But then it'll immediately turn into the corpus luteum whether we have fertilization or not. So I will put corpus luteum here. And what does the corpus luteum do? Well, it produces a lot of estrogen or progesterone. And so estrogen and progesterone. Now it's certainly doing that, as I already said, to help maintain an embryo. It does it for other reasons as well. But I'll actually add that one down here. Well, I won't, I won't, because there's not obviously there's another pregnancy so I'm not going to add it but do know that that's one of the that's the main reason estrogen levels are going up is to maintain an embryo to thicken the uterine wall and I will put that so we're going to thicken the uterine wall, which is part of maintaining that embryo because the embryo is going to implant into that uterine wall happens for another reason Isn't that is the high levels of estrogen and progesterone in there it is to inhibit LH and FSH. Well, Dr. R, I thought you just said over here in the proliferative phase and estrogen stimulates LH and FSH ducts, but only when the levels are three arrows up. If the

levels are, if the levels of estrogen are one arrow up you're not going to stimulate LH and FSH, you needed three arrows up to stimulate these particular hormones. If it's only one arrow up when it comes to the level of estrogen, it's not going to do crap Lola, to LH and FSH, it's actually going to be influenced by LH and FSH. If it's two arrows up. It's actually going to inhibit LH and FSH. This is something that I didn't mention. But when it comes to the concentration of certain molecules, it can change the way that they affect other molecules. Okay, why didn't I get into that kind of detail? Because it's an undergrad course, when you guys go to graduate school or medical school, you'll get into that kind of detail. I'm giving you a taste of it right now. Why would we want to inhibit LH and FSH? We don't want to ovulate again. We already did it. The assumption is that when we ovulated that is the body is that it got fertilized. That's the assumption that's made. This is how your traditional birth control pills work. By the way, with estrogen, those birth control that is traditional traditional birth control pills have estrogen progesterone, the estrogen levels that are high and that birth control pill, inhibit inhibit ovulation, the higher progesterone thickens the mucus of the cervix so that the sperm can swim through it. And so that's what prevents pregnancies. We're not going to get into it, I know that you do see, I have contraception on the last page. And are you 46 the abortion pill that we're not, I'm not going to cover. So we're gonna cross that stuff out. So we're actually almost done tonight. So anyway, corpus luteum is still around, we're increasing level of estrogen, progesterone, these are the two things that I need you to know when it comes to estrogen and progesterone levels going up and what the function is late. And again, we're assuming though, pregnancy. corpus luteum. is dead. Because there's no HCG because there's no embryo. And at the corpus luteum dies. Well, now what

1:02:51

we need to do, let me remind myself, I had three of them. So really low levels of estrogen, the estrogen levels are going to plummet.

1:03:09

And so the progesterone levels. And we're going to start all over again. That's how we end.

1:03:18

That's how we started. And the cycle just starts over month after month after month after month after month, unless of course, there's fertilization, there's a pregnancy, well, then we're not going to have any menstrual cycles. So there you go. That's the menstrual cycle and those things that I would like you to know. Now what last but not least, these are the only things we're going to be talking about, see what I have an extra there, you're not going to have to know any of that stuff. I used to teach it. I don't teach it anymore. So birth control pills, no more emergency contraception? Are you 4060 abortion pill, I don't talk about it anymore. So it won't be on the exam. This stuff will be on the exam though. Once monarchy is the very first menstrual cycle. That's what it is. What's the matter Ria, we already know, a failure to have a menstrual cycle if you're of age to have a menstrual cycle. Now I know that there's some extra stuff here on the screen. But these are things that we already knew this should have been in the notes. It just didn't make it in time to the publishers. But we already knew this because we learned it in the endocrine chapter hypersecretion are hypo secretion of LH and FSH causes amenorrhea. So when I first introduced the term amenorrhea I can you know clearly see why. If we go to the pretty picture, look what has to happen to LH and FSH. They have to be timed

perfectly. And if they're high all the time, or low all the time. This ain't happening. Prolactin, really high levels of prolactin remember that? lactational amenorrhea Correct. High levels of protein I can inhibit GnRH you inhibit GnRH you're gonna hit inhibit LH and FSH hypo secretion again, we already learned what else really low body fat why estrogen and progesterone are hormones made of fat? What do we call those? By the way? There's three kinds of hormones. Protein, I mean, and steroid steroid hormones, steroid hormones are made of fat. If you don't have enough fat, you're not going to make enough estrogen and progesterone. Well, how do you think that's going to work out with the menstrual cycle, you're certainly not going to have an estrogen surge, which means you're not going to have an LH FSH search, you're not going to thicken the uterine wall very well. So you won't have a menstrual cycle. Progesterone again, that's also a steroid hormone. Stress, anxiety. What the hell's that about? How does the menstrual cycle start? What do we release? What's the first hormone that gets released in the menstrual cycle that must be released and there's not happening GnRH that starts the whole thing off. So hypothalamus, you guys learned about the hypothalamus last semester, right? And it's involved in like, stress, anxiety. So now if you're stressed and anxious, hypothalamus not being might not release GnRH. If you don't release GnRH, you're not going to increase LH and FSH. If you don't increase LH and FSH, this might not happen. You might not get the Grampian follicle, you're not you might not get estrogen to surge, which means that LH and FSH aren't going to surge, which means ovulation is not going to none of this is going to happen. Because the hypothalamus is compromised because of the stress that somebody is under. And so that's what that's all about stress and anxiety, some change in your lifestyle. Or Tucci exam on Tuesday? Who knows? Yeah.

1:07:00

Like sometimes they say that, like female athletes start their period later. So would that be because of the low body,

1:07:06

low body fat, and if you're an athlete, you're probably stressing the hell out of the body, right. So when you think of stress, you think anxiety, but it can be physical stress, too. Okay, if you're working out six days, seven days a week and running 10 miles or doing whatever, you know, soccer player, whatever, you know, fighter, that's stressful to the body. So that stress is not just mental and emotional stress that that can be physical stress as well. And if you're an athlete and a female, you probably have lower body fat depending on what sport you're participating in. And so it can be multifaceted when it comes to that absolutely. menopause, talked about last semester, when we talked about the bones right? Talked about how with menopause, you got an increased risk of developing osteoporosis. Remember that because of the low level, low level of estrogen and progesterone, that we're not going to get into, we don't need to so successfully the menstrual cycle right around the age of 50. Those ovaries just don't work the way that they used to, which means we have decreased levels of progesterone and estrogen. And so let's just do this really quick. And it'll be the last thing we do looks like you might get out of here just a little bit early tonight. So with menopause, no more menstrual cycles. And so it presents with a number of symptoms. And so with menopause, there's a decrease in progesterone lot of it, or a big decrease, I should say. And there's a big decrease in estrogen now, especially estrogen when it comes to the story. The decrease in estrogen causes dysfunction. And we're going to go back to the hypothalamus again, of the hypothalamus. And as you know, from last semester, when X receiver taught you and I touched upon it a little bit. It's involved in in the response to stress and anxiety, especially emotional stress, mental stress. And so now that we have something and it's also by the way, and I mentioned this last

semester, that's the thermostat of your body as well. So our body temperature is normally what around 98 degrees and 8.6 37 degrees Fahrenheit, right? Why? Because the hypothalamus says that's what's gonna that's going to be that the hypothalamus is the regulator, it's gonna determine the setpoint. So if you have a screwed up hypothalamus, that thermostat might be doing all kinds of crazy stuff. Like thermostat in my house right now. 69 degrees. If I go home, and I press that up button, to 72 or 73, my house is going to be 72 or 73 degrees, or if I want it to be 66, which is about what it is when I go to sleep. That's when it's gonna be because of the thermostat. If the hypothalamus is doing that, body temperature is gonna go up and down, up and down, and up and down, up and down. You get hot flashes. Now it's called hot flashes. But she can get cold, or she can get hot. And it can change fairly quickly. Because again, the hypothalamus is kind of screwed up, it's not functioning properly because it needs a high enough level of estrogen. These other symptoms that you see emotional disturbance and irritability, it goes back to, again, the job of the hypothalamus. And if it's not functioning properly, well, then emotions might not be able to be regulated the way that they normally are. Fatigue as well. Can this be managed? What can this person do if she wanted? Hormone therapy? Just as we talked about last semester, hormone therapy? How much let me just ask you the second be an example. Just for fun. Let me see if you remember, during menopause? How much more bone do you lose every every year? Does it how much does it go up? You guys remember how many times three, get a three fold increase in bone loss. Remember that during menopause, a bone loss and then it kind of levels out and what we call old age. And we talked about how you can just wipe that out wipe that three fold increase in bone loss by doing what just replaced the hormones that are lost. It's the same thing here. So let me do this. But no idea how that happened. Okay. Causes dysfunction of the hypothalamus responsible for the symptoms in large part.

1:11:43

menopausal symptoms, which we just talked about. Do you treat hormone therapy can be treated with hormone therapy doesn't mean you have to, because then you run some risk of increasing or you increase the risk of some cancers that just saw the decision that would be made by us physicians and PDAs and whatever you got to be someday and your patient can be treated with hormone therapy and that's that all done. So again, test on Tuesday, most of the questions are going to come from again that second chapter because that was the biggest chapter

Practice Exam 1

1. How could one help minimize inflammation in the body?
    a. Synthesize more series 2 prostaglandins
    b. Synthesize less series 1 prostaglandins
    c. Ingest arachidonic acid
    d. Ingest omega-3 fatty acids

2. Epinephrine is most often released from the adrenal gland in what manner?
    a. Chronic
    b. Acute
    c. Cyclic

3. Synthroid, which is an example of a drug that mimics the action of thyroid hormone, would be considered a(n) _____ of thyroid receptors.
    a. Agonist
    b. Blocker
    c. Antagonist
    d. Second messenger
    e. Autocrinie

4. Why can most protein hormones circulate freely (i.e. not carried) in the blood?
    a. They are very large
    b. They are very small
    c. They are water soluble
    d. They exhibit endocrine activity
    e. They have long half-lives

5. What neurohormone can be involved with a positive feedback mechanism?
    a. ADH
    b. Prolactin
    c. GHRH
    d. Oxytocin
    e. Somatotropin

6. What does hyposecretion of ADH cause?
    a. SIADH
    b. Diabetes insipidus
    c. Retention of water
    d. Hypernatremia
    e. Two of the above

7. All of the following hormones can increase blood glucose except one. Which one is it?
    a. Aldosterone
    b. Glucagon
    c. Cortisol
    d. Thyroid hormone
    e. Growth hormone

8. What can hypersecretion of adrenocorticotropic hormone lead to?
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    c. Cushing's disease
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9. What negative feedback loops is correct?
    a. Increased level of cortisol inhibits ACTH release
    b. Decreased level of cortisol inhibits CRH release
    c. Increased level of ACTH stimulates FSH and LH release
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10. Hypoglycemia, stress, deep sleep and exercise all stimulate what hormone?
    a. Corticotropin releasing hormone
    b. Cortisol
    c. Glucagon
    d. Growth hormone
    e. DHEA

11. What can be caused by a pituitary tumor?
    a. Hypersecretion of TSH
    b. Hypersecretion of T_3 and T_4
    c. Cushing's disease
    d. Pituitary dwarfism
    e. All of the above

12. What is associated with untreated type 1 diabetes?
    a. Low blood glucose
    b. Glycogenolysis
    c. Hyperglycemia
    d. Gluconeogenesis
    e. Increase protein synthesis

13. A decrease in LH levels can lead to male sterility.
    a. True
    b. False

14. What could a decrease in T_3 and T_4 along with an increase in TSH indicate?
    a. Hashimoto's disease
    b. A problem with the anterior pituitary
    c. Iodine deficiency
    d. A problem with the hypothalamus
    e. Two of the above

Use the following to answer the next four questions.

a. increases (directly or indirectly)

b. decreases (directly or indirectly)

c. has little or no effect

15. Decreased normal resting level of epinephrine _____ on heart rate.

16. Low level of cortisol _____ the release of ACTH.

17. Increased somatostatin _____ the release of hormone from the beta cells of the pancreas.

18. What is associated with hypernatremia and high blood pressure?
    a. Cushing's disease
    b. Cushing's syndrome
    c. Addison's disease
    d. Aldosteronism
    e. Hashimoto's disease

19. What disease can have the following symptoms: enlarged heart, elevated growth hormone, type 2 diabetes mellitus, elevated fatty acids.
    a. Hyperthyroidism
    b. Gynecomastia
    c. Acromegaly
    d. Pituitary dwarfism
    e. Addison's disease

Use the following to answer the next two questions.

a. increased, increased, increased

b. decreased, decreased, decreased

c. increased, increased, decreased

d. decreased, increased, increased

e. decreased, decreased increased

20. With hypothyroidism due to a lack of iodine, TRH is ___ TSH is ___ and T_3 and T_4 are ___.

21. With SIADH, plasma Na^+ concentration is ___ ADH is ___ and blood pressure is ___.

22. The zona reticularis produces and secretes more testosterone than DHEA.
    a. True
    b. False

23. Primary hyperparathyroidism causes osteoporosis and a decrease in blood calcium.
    a. True
    b. False

24. What has lost negative feedback control given the following: high renin and high aldosterone
    a. Adrenal cortex
    b. Pancreas
    c. Kidneys
    d. Thyroid gland
    e. Posterior pituitary

25. What has lost negative feedback control given the following: high FSH and low testosterone
    a. Adrenal medulla
    b. Pancreas
    c. Thyroid gland
    d. Testes
    e. Anterior pituitary

26. What could inhibition of Leydig cells during gestation cause?
    a. Cryptorchidism
    b. Spermatogenesis
    c. Retardation of male reproductive organs
    d. Emission
    e. Two of the above

27. During spermatogenesis, testosterone is absolutely required for meiosis I to occur.
    a. True
    b. False

28. Ovarian follicels are fully developed at birth.
    a. True
    b. False

29. What is the neurotransmitter that causes penile erection?
    a. Nitric oxide
    b. Epinephrine
    c. Norepinephrine
    d. Acetylcholine
    e. Two of the above

30. What initiates the final meiotic steps of an ovulated secondary oocyte?
    a. FSH
    b. LH
    c. Menses
    d. Secondary oocyte fertilization
    e. Estrogen

31. What initiates the beginning of the menstrual cycle (i.e. menses)?
    a. Decrease in FSH
    b. Decrease in LH
    c. Decrease in estrogen and progesterone
    d. Disintegrated corpus luteum
    e. Two of the above

32. Estrogen level rapidly declines after ovulation.
    a. True
    b. False

33. Who would most likely fail to ovulate?
    a. Female with amenorrhea
    b. Female during menopause
    c. Female with low GnRH
    d. Female with low LH
    e. All of the above

1. D
2. B
3. A
4. C
5. D
6. E (b and d)
7. A
8. C
9. E (a and d)
10. D
11. E
12. C
13. A
14. E (a and c)
15. C
16. A
17. B
18. D
19. C
20. C
21. D
22. B
23. B
24. C
25. D
26. E (a and c)
27. A
28. B
29. E (a and d)
30. D
31. E (c and d)
32. A
33. E

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    a. True
    b. False

14. What could a decrease in T_3 and T_4 along with an increase in TSH indicate?
    a. Hashimoto's disease
    b. A problem with the anterior pituitary
    c. Iodine deficiency
    d. A problem with the hypothalamus
    e. Two of the above

Use the following to answer the next four questions.

a. increases (directly or indirectly)

b. decreases (directly or indirectly)

c. has little or no effect

15. Decreased normal resting level of epinephrine ____C_____ on heart rate.

16. Low level of cortisol ____A_____ the release of ACTH.

17. Increased somatostatin ____B____ the release of hormone from the beta cells of the pancreas.

18. What is associated with hypernatremia and high blood pressure?
    a. Cushing's disease
    b. Cushing's syndrome
    c. Addison's disease
    d. Aldosteronism
    e. Hashimoto's disease

19. What disease can have the following symptoms: enlarged heart, elevated growth hormone, type 2 diabetes mellitus, elevated fatty acids.
    a. Hyperthyroidism
    b. Gynecomastia
    c. Acromegaly
    d. Pituitary dwarfism
    e. Addison's disease

Use the following to answer the next two questions.

a. increased, increased, increased

b. decreased, decreased, decreased

c. increased, increased, decreased

d. decreased, increased, increased

e. decreased, decreased increased

20. With hypothyroidism due to a lack of iodine, TRH is _C_ TSH is _C_ and T_3 and T_4 are _C_.

21. With SIADH, plasma Na^+ concentration is _D_ ADH is _D_ and blood pressure is _D_.

22. The zona reticularis produces and secretes more testosterone than DHEA.
    a. True
    b. False

23. Primary hyperparathyroidism causes osteoporosis and a decrease in blood calcium.
    a. True
    b. False

24. What has lost negative feedback control given the following: high renin and high aldosterone
    a. Adrenal cortex
    b. Pancreas
    c. Kidneys
    d. Thyroid gland
    e. Posterior pituitary

25. What has lost negative feedback control given the following: high FSH and low testosterone
    a. Adrenal medulla
    b. Pancreas
    c. Thyroid gland
    d. Testes
    e. Anterior pituitary

26. What could inhibition of Leydig cells during gestation cause?
    a. Cryptorchidism
    b. Spermatogenesis
    c. Retardation of male reproductive organs
    d. Emission
    e. Two of the above

27. During spermatogenesis, testosterone is absolutely required for meiosis I to occur.
    a. True
    b. False

28. Ovarian follicels are fully developed at birth.
    a. True
    b. False

29. What is the neurotransmitter that causes penile erection?
    a. Nitric oxide
    b. Epinephrine
    c. Norepinephrine
    d. Acetylcholine
    e. Two of the above

30. What initiates the final meiotic steps of an ovulated secondary oocyte?
    a. FSH
    b. LH
    c. Menses
    d. Secondary oocyte fertilization
    e. Estrogen

31. What initiates the beginning of the menstrual cycle (i.e. menses)?
    a. Decrease in FSH
    b. Decrease in LH
    c. Decrease in estrogen and progesterone
    d. Disintegrated corpus luteum
    e. Two of the above

32. Estrogen level rapidly declines after ovulation.
    a. True
    b. False

33. Who would most likely fail to ovulate?
    a. Female with amenorrhea
    b. Female during menopause
    c. Female with low GnRH
    d. Female with low LH
    e. All of the above