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Doctors 2021 - رَوح - Medicine - Mu

Physiology Sheet

Zona granulosa and Fasciculata

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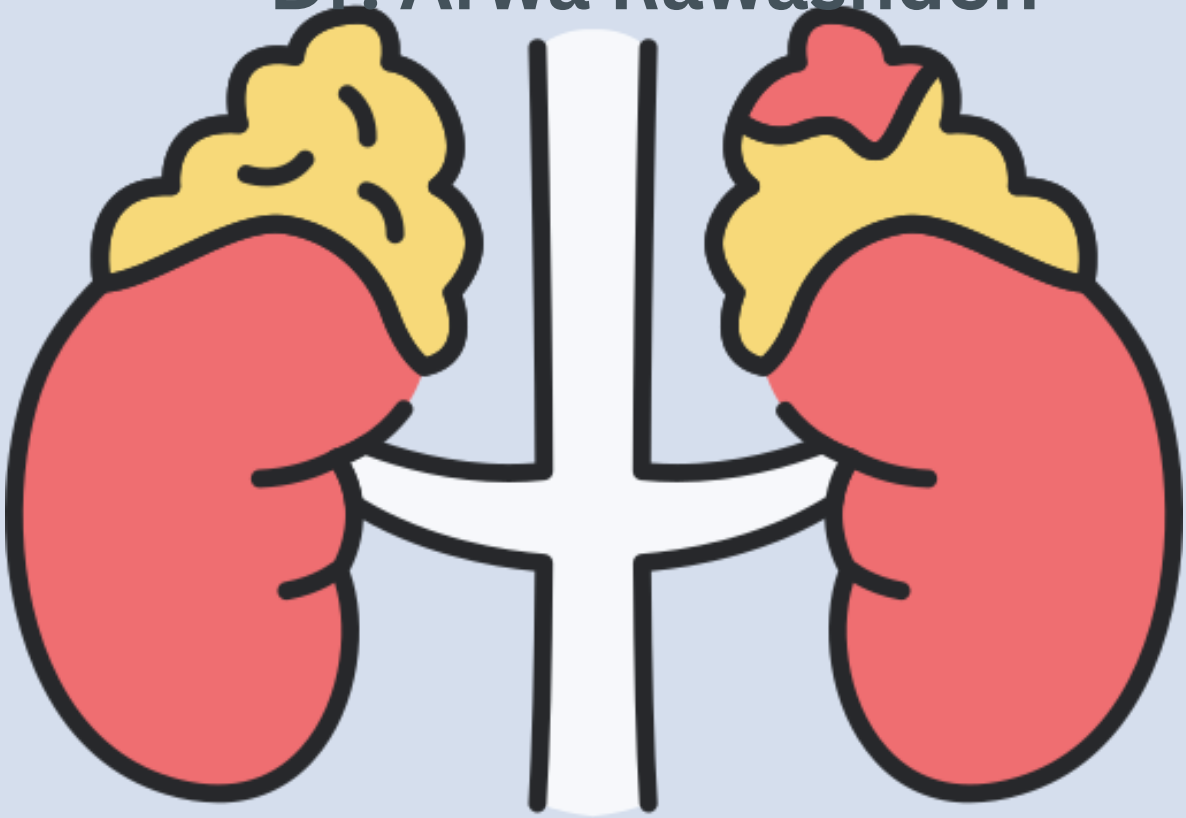

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For nenja nerd vedio

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For osmosis vedio

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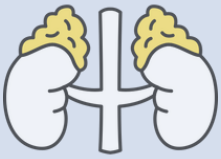
Adrenal Gland



- Top of the kidney (suprarenal gland)
- Pyramid gland
- cortex

granulosa mineralocorticoids aldosterone mainly controlling Na+

fasciculata(biggestLayer) glucocorticoids --> cortisol

Reticularis androgens (weak androgens)

Adrenal medulla (neural tissue) catecholamines

-—> Mechanism of secretion of Zona granulosa

this mechanism is known as renin- angiotensin-aldosteron mechanism

Low blood pressure (strong stimulus for aldosteron secretion)

In the kidney ,Renin(Juxtaglomerular cells)

Liver plasma protein enzyme (angiotensinogen)

Renin(enzyme) convert angiotensinogen, angiotensin one

Lung angiotensin converted enzyme(A.C.E) , angiotensin one into two

low blood pressure --> reduced blood pressure in kidneys --> secretes renin --> renin cleaves angiotensinogen which is secreted by the liver to --> angiotensin I --> angiotensin I is metabolized by ACE from the lung to angiotensin II

Angiotensin two binds to G receptor coupled protein, G stimulatory protein –> convert GDP into GTP activate adenlytae cyclase which convert ATP into cAmp activates Protein kinase P.K.A (the strongest stimulus)

Paraventricular nucleus corticotropic releasing hormone (CRH) is from hypothalamus travels to anterior pituitary releases ACTH adrenocorticotropic hormone (weakest stimuli in stress conditions) , the same pathway of angiotensin two

بالامتحان رح نعتبر انه ال ACTH ماله علاقه بال aldosteron

Steroid hormone synthesis

Cholesterol –> pregnenolone –> progesterone by 21- hydroxylase –> 11-deoxy corticosterone –> corticosterone –> Aldosterone (the second stimulus)

- P.K.A phosphorylating each enzyme in each step (of formation of aldosterone)
- Low sodium (hyponatremia) or high potassium (hyperkalemia) level in the blood is also a stimulatory signal of aldosteron formation (weak to moderate stimulus for aldosterone secretion 2nd stimulus)

➡Inhibitors

- Blood pressure high ⬆ -->causes production of atrial natriuretic peptide

Atrial natriuretic peptide (strongest) activate G inhibitory pathway which result in K efflux lead to --> (hyperpolarization) , alter the enzymatic activity (inhibition)

➡Effect of aldosterone

Bind to Trans cortin (corticosteroid binding globulin) or albumin reach distal convoluted tubules, enter Inside the cell bind with receptor activate gene sequence–>transcription mRNA translation proteins Very slow action

📍 water soluble hormones have more rapid onser of action than lipipd soluble hormones as lipid soluble hormones play their role on gene level

📍 ?aldosterone is lipid soluble

📌? Overall effect

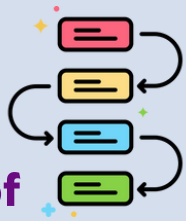
Plug three different types of protein into the cell membrane

1. Sodium potassium pump establish gradient 3Na - out / 2 k (in utilizing ATP)
2. More pumps for sodium in the luminal membrane from the filtrates into the blood

In Blood ⟶ Na (increase)
k (decrease)

3.Potassium from the blood excreted through distal convoluted tubules

- Increase blood volume --> due to Na retention (osmolarity)
- Increase blood pressure -> (the final outcome)



another mechanism for increasing blood pressure is by the action of angiotensin I, which causes vasoconstriction and increase in total preipheral resistance --> high b.p

sodium get into the cell from distal convulated tubule

📍Na—> move into the cell —> go to blood —> ⬆ Na level in the blood (the origin stimulus here is low sodium level) we fix the problem

📍K —> exereted and go with urine —> ⬇ low potassium level in the blood

when Na increase in the blood, the water follows the sodium—> ⬆ the volume of the blood —> ⬆ blood pressure

📌the stimulus here is low blood pressure (Angiotensin) we fix the problem

🔴ANP is inhibiting aldosterone from being released—> the stimulus of ANP is high blood percent

Hyperaldosteronism

OSMOSIS

- Primary: Conn's syndrome (adenoma) of zona granulosa , genetic, idiopathic .

Low renin high aldosterone --> renin is decreased due to negative feedback

- Secondary: chronic low of blood pressure

(congestion of heart disease), cirrhosis. High aldosterone and Renin

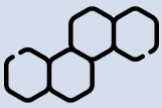
Insufficiency of adrenal gland

--> there is no negatice feedback effect of aldosterone on renin as the cause of hyperaldosteronism (high blood pressure) is still active or not treated

cause : hypokalemia, hypernatremia, hypertention, alkalosis

- Addison's disease low cortisol (manifestation)
  - Autoimmune or tumor
  - Hyperkalemia
  - Dark pigmentation alpha melanocyte
  - Opposite of cortisol excess

Mechanism of secretion of cortisol



- Paraventricular nucleus (of hypothalamus) secret corticotropin releasing hormone CRH —> hypophyseal system to anterior pituitary gland which secrete in the blood adrenocorticotropic hormone (strongest stimulator) stimulate the zona faseculata

- ACTH bind to G protein coupled receptors —> activate G stimulatory protein bind GTP which stimulate Adenylate cyclase convert ATP to c. AMP activte protein kinase A P.K.A (phosphorylating different kinds of protein)

Steroid hormones

- Cholesterol is the basic unit to make steroid hormones not DNA or mRNA or proteins
- Cholesterol —> pregnenolone —> progesterone —> 17- hydroxy progesterone by 21- Hydroxylase 11- -> deoxy cortisol —> cortisol

P.K.A phosphorylating different enzymes involved within enzymatic reaction stimulate

Effect of cortisol

cortisol prepares the body for fight or flight

- 25% of cortisol bind to albumin
- 75% bind to cortico steroid binding globulin (trans Courtin)

➡ on muscles and bones: Muscle and bone (Protein catabolism) 5-6mm (normal thickness of compact bone) 40 mg prednisone /day /year. 1/5 of contact bone (the thickness of compact bone, becomes only about 1mm after conteneues cortisol adminestration)

- Binds intracellular receptors activate —> specific gene. ..->make:
- Proteases break the peptide bond (for protein in muscle)
- Releasing Amino acids into blood
- Amino acids travel to liver

➡on Adipocyte

Triglycerides will be broken into glycerol and fatty acids(glycerol to liver, Fatty acid chains utilized by muscles or redistributed in different part of the body)

➡on liver (hyperglycemia); increase insulin from pancreas due to gluconeogenesis activation from glycerol and amino acids.

📌Gluconeogenesis Glycerol, amino acids, lactic acids (from skeletal muscles), fatty acids and converted to glucose

📌Glycogenesis (converting glucose into glycogen) Direct effect of cotisol

📌Glycogenolysis (breaking glycogen into glucose) by stimulating adrenergic receptors in the liver -->increases their sensitivity to released epineohrine and nor-epinephrine indirect effect nor epinephren causing

➡Tunica media of Smooth muscle(increase the sensitivity of adrenergic receptor)

Sensitivity of adrenergic receptors amplify the effect of norepinephrine (cortisol increases the sensitivity of adrenergic receptors)

📍 the over all effect (vasocontraction and increase blood pressure)

➡in immune system

📍inhibiting of :

📌Basophiles (histamine (very potent in allergic), leukotriene,

📌prostaglandins)

Lymphocytes (interleukins, cytokines) Monocytes (interleukins, Cytokines)

Secretion of cortisol

Hypoglycemia --> enhances secretion of cortisol and leads to :

1. Glycogenolysis (indirectly) glycogenolysis which increases sensitivity to NE
2. Gluconeogenesis
3. Glycogenesis (direct)

secretion of cortisol occures primarly during stress while epinephrine is released during active stress

long term stress (chronic stress) >1 or 2min Trauma or starvation or emotional leads to enhance secretion of cortisol which leads to:

1. Vasocontraction (increase blood pressure)
2. Protein catabolism
3. Depression of immune system

High cortisol causes

- Negative feedback effect on hypothalamus (CRH) lead to ⬇
- Negative feedback effect on anterior pituitary gland (ACTH)

Low cortisol

- High CRH and ACTH

Cushing Excess cortisol

normal laboratory test for cortisol level reveals that blood and urine conc. is <2mg/dl

normal

1. 1mg Dexamethasone suppression 2mg blood Urine free 24hrs <2mg
2. 2mg dex positive >2mg
3.

ACTH low adrenal tumor (benign or malignant) ACTH high pituitary or Ectopic

4. 8mg DEXA

Pituitary suppression Cushing disease

Ectopic not suppress tumors

how to test cortisol levels

1- 1mg of dexamethasane is administered to patient

--> if both blood and urine cortisol levels are still <2mg

--> the patient is normal

OR

--> if blood cortisol levels become MORE THAN 2mg

--> the patient requires more investigation

2- 2mg of dexamethasane is administered to patient --> by defult blood conc. is MORE THAN 2mg

–> ACTH is tested

--. low --> means that there is adrenal tumor

--> high --> means tha it is either pituitary or ectopic caused require further test --> 8 mg of dexamethasone administered to patients

–> IF ACTH id suppressed then its a pituitary TUMOR

--> if ACTH is not suppressed it means that there is an ectopic cause

يا ولدي..

تعلّم فقهَ الاستدراك، فلا تجلِس شاكياً على ما فاتَ منك، نادِماً على ما فرّطت، فتقعدَ مع القاعدين ترثي حالك..

وإنّما كن معَ المُستدرِكين، من فقِهوا قيمةَ أوقاتهم، وعِظَم المسؤولية التي على عاتقهم، فشمَّروا عن ساعدِ الجِدِّ، واستعانوا بالله على أنفسهم وعلى العقباتِ في طريقهم..

من يستغفرون على ما مضى من تَقصيرهم، لكنّهم يعاهدونَ الله كلَّ يومٍ على المضيّ قُدُماً في استدراكٍ يُرضي الله عنهم..

تعلّم أن تُتبعَ كلّ سيئةٍ حسَنة، وكلّ تقصيرٍ إنجاز، وكلّ ذبولٍ بذرة، وكلّ خمولٍ عمل، وليَكن رفيقك في كلّ ذلك تذلّلاً لله وشكراً، بأن وهبكَ إشراقَ يومٍ جديد من عمرك، تستدركُ فيه ما فات

- في كلّ منّا فارس.