Autonomic nervous system

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Introduction:

The autonomic nervous system is the portion of the nervous system that controls most visceral functions of the body, and it is distinct from the motor somatic nervous system, which innervates skeletal muscle. This system helps to control arterial pressure, gastrointestinal motility, heart rate, gastrointestinal secretion, urinary bladder emptying, sweating, body temperature, and many other activities. Some of these activities are controlled almost entirely and some only partially by the autonomic nervous system. (maybe with the help of hormones for example)

The ANS has two subdivisions:

sympathetic and parasympathetic.

Characteristics of the ANS:

1-Speed of onset: ANS can make changes in the activity of the organs it innervates within seconds (3-5), because you must respond in a fast way if you are facing a danger.

2-Automatic nature: you can't control the activity of the organs that are innervated by the ANS, they are being controlled without your conscious using involuntary muscles, but the urination process is an exception, because it has some voluntary muscles.

3- Tonic activity: The ANS fires continuous impulses to target organs at very low rate. The basal rate of firing is called “sympathetic tone” and “parasympathetic tone”. These tones establish basal rate of contractile activity in smooth muscle cells, and secretory activity of glandular tissues. The activity of these effector cells can be changed as a result of an increase or a decrease in the activity of any divisions of the ANS.

شو هاذ التخبيص اللي مكتوب؟

عارف هسا بشرحو

The ANS normally is always active even if there is no stimulation so the sympathetic and parasympathetic systems are continually active, and the basal rates of activity are known, respectively, as sympathetic tone and parasympathetic tone.

Basal rate, in biology, is: **the rate of continuous supply of some chemical or process**.

Simply it is the amount of nerve impulses needed to sustain the involuntary activities of the body including maintaining muscle tone, body temperature and proper functioning of the heart, lungs and gastrointestinal tract.

The value of tone is that it allows a single nervous system to both increase and decrease the activity of a stimulated organ. For instance, sympathetic tone normally keeps almost all the systemic arterioles (الشرايين الجهازية) constricted (متضيق)to about one-half their maximum diameter. By increasing the degree of sympathetic stimulation above normal, these vessels can be constricted even more; conversely, by decreasing the stimulation below normal, the arterioles can be dilated(متوسع). If it were not for the continual background sympathetic tone, the sympathetic system could cause only vasoconstriction, never vasodilation.

In other words, the ANS sends consistent intermediate signals that are not extremely weak nor extreme and may be graded (increased or decreased) in response to several stimulations

So for example even now when you are reading this, your ANS is working and its keeping your body temperature normal and keeping your heart rate at normal range even though there is no stimulation.

هاذ شرح لنقطة رقم 3

Important terminology:

1 – Ganglion: Nerve cell cluster, where neurons are typically linked by synapses. Also, it`s the border line between preganglionic and postganglionic and where they synapse.

2 – Preganglionic = presynaptic = first neuron: the neuron which extends from Central nervous system to its synapse with second neuron.

3 – Postganglionic = postsynaptic= second neuron: the neuron that extends from the ganglion to the effectors (usually organs).

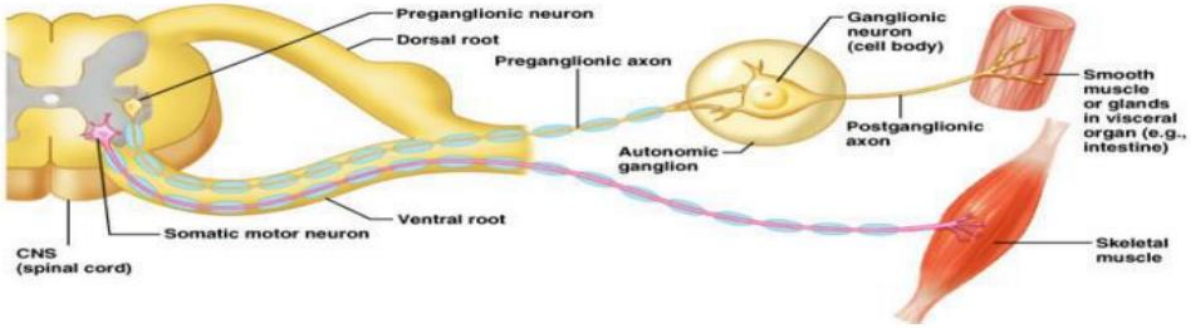
4 – Paravertebral ganglion: ganglion that presents near the vertebral column, found in sympathetic nervous system only.

5 – Prevertebral ganglion: ganglion that presents apart from the vertebral column near the effectors (usually organs in abdomen), found in sympathetic nervous system only, and they are only three: - celiac ganglion. - superior mesenteric ganglion. - inferior mesenteric ganglion

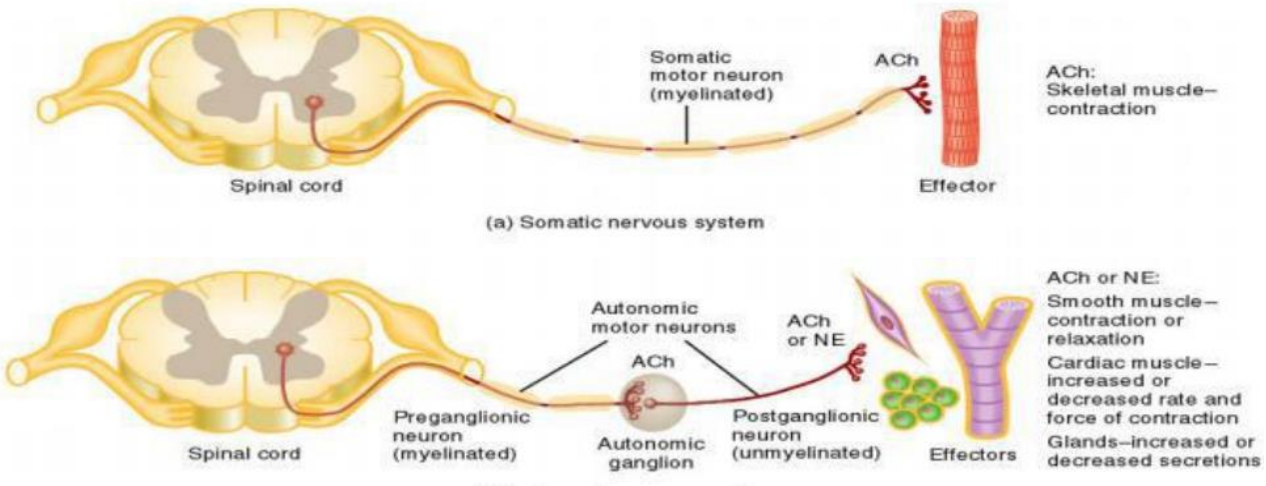
6 – Terminal ganglion: ganglia that are found in the effected organ, only found in parasympathetic nervous system.

احفظهم منيح من هسا كل الشرح اللي بعد هيك بيعتمد عليهم

السعادة تنصرف عنَّا في أكثر الأحيان ليكون تلهفنا عليها واحتياجنا لها سعادة على وجه آخر.

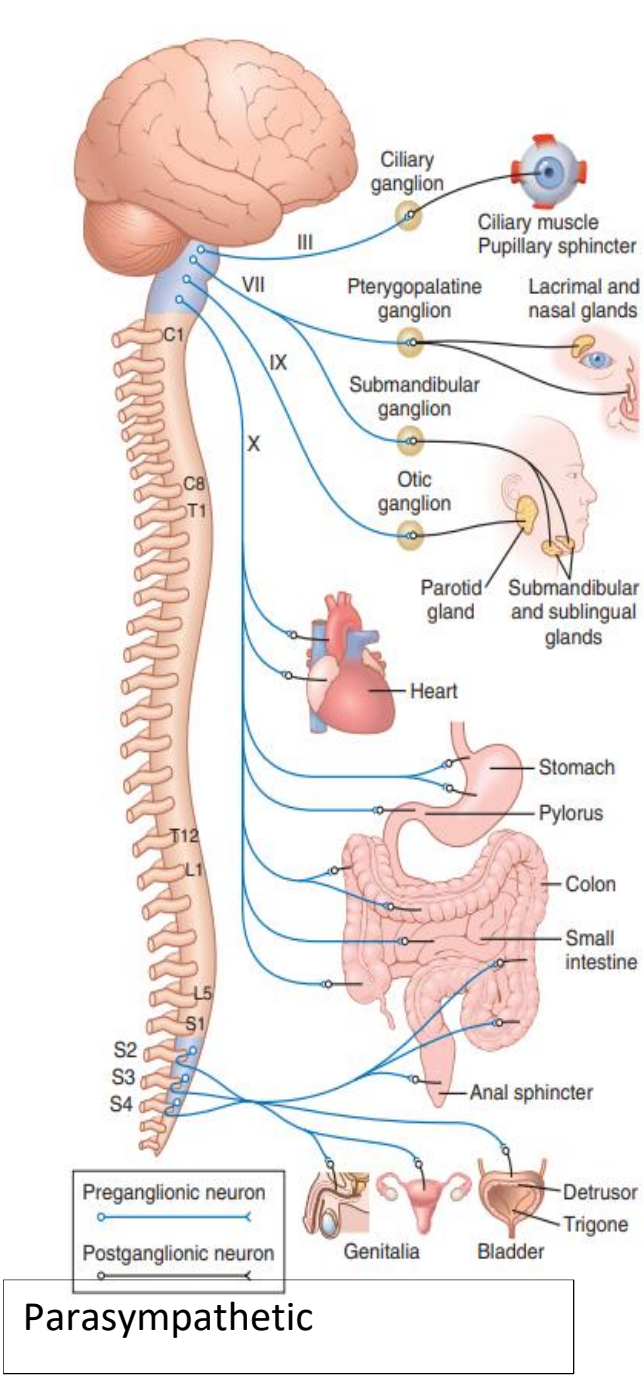


As you can see in the picture The ANS neurons are different from skeletal motor nerves in the following way: Each sympathetic pathway from the cord to the stimulated tissue is composed of two neurons, a preganglionic neuron and a postganglionic neuron, in contrast to only a single neuron in the skeletal motor pathway.

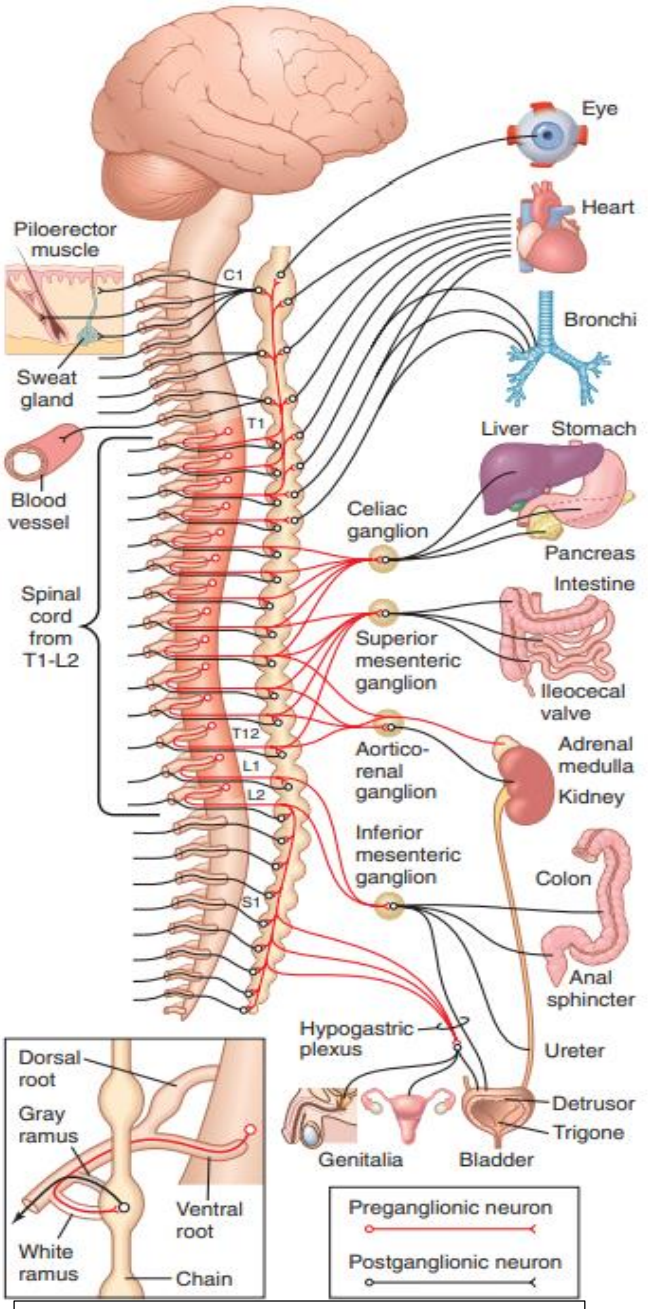


This represent ganglion where we have the synapses between the postganglionic and preganglionic neurons (in autonomic), and the cell bodies of the postganglionic neurons are located in ganglion which is different from the somatic system. The somatic system h cell body at the level of spinal cord (like the motor neuron), long axon towards the effector structure, which is skeletal muscles, and the synapse between the terminal of somatic neuron and effected structure (neuro-muscular junction). We don't have ganglion along the somatic nerve system but we have along autonomic.

The picture is from the book it is better than the one in the slides and it is not for memorizing



Parasympathetic



sympathetic

Physiological anatomy:

Two neurons carry impulses of the ANS from the CNS to the effector organs.

The first is known as preganglionic neuron, the cell body is located in the CNS The fibers of preganglionic are small and myelinated, and usually end within a ganglion where they synapse with the second neuron called postsynaptic neuron. The second neuron (postsynaptic) carries impulses to target organ.

DIVISIONS OF ANS: There are two divisions of the ANS: sympathetic and parasympathetic autonomic nervous systems.

Sympathetic nervous system: The cell bodies of preganglionic neurons lie in lateral gray of spinal cord at segmental levels of T1 through L3. Axons leaves spinal cord via ventral roots, then leave ventral root via white rami communicants to enter a vertebral ganglion of the sympathetic chain at the same segmental level. The preganglionic axon then can: * Synapse with postganglionic cells at the same segmental level. * Turn cranial or caudal and synapse with sympathetic postganglionic neuron at higher or lower segmental level. Synapse may occur at more than one postganglionic neuron.

The picture bellow is number coded each number represents the corresponding step

شو هاذ التخبيص؟ هسا بتفهم

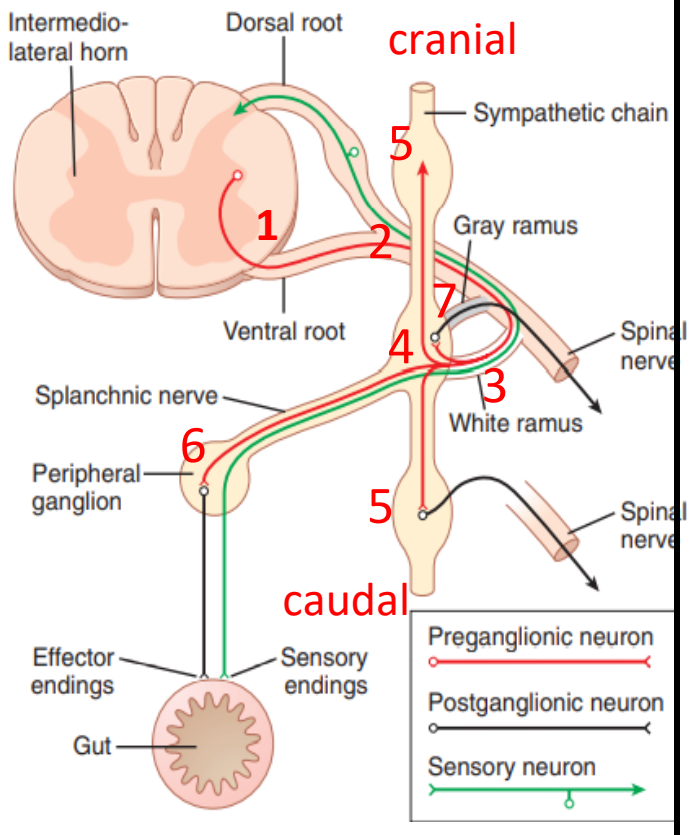
First of all, lets know the origin of the sympathetic division it's from the spinal cords of T1 to L3 thus it is called "thoracolumber"

1-The sympathetic neuron exits from the spinal cord from the ventral (dorsal root) as you see in the picture in red

2-Then it joins with the dorsal root to form the spinal nerve 3- then immediately the preganglionic sympathetic nerve fibers leave the spinal nerve and pass through a white ramus into one of the ganglia of the sympathetic chain.

The fibers then can take one of the following three courses:

(4)-they can synapse with postganglionic sympathetic neurons in the ganglion that they enter (same segmental level;



(5) they can pass upward (turn cranial) or downward (turn caudal) in the chain and synapse in one of the other(paravertebral) ganglia of the chain; or

(6) they can pass for variable distances through the chain and then through one of the sympathetic nerves radiating outward from the chain, finally synapsing in a peripheral(prevertebral) sympathetic ganglion.

(7) After synapse with neurons at paravertebral ganglia, axons of postganglionic (second) neurons leave ganglia via gray rami communicants to return to the corresponding spinal nerve.

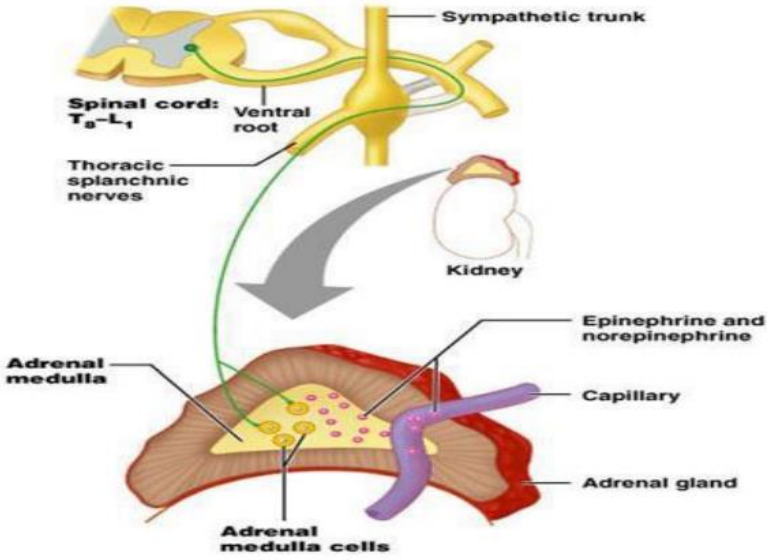
A side note from Zaid: the preganglionic neurons are myelinated and appear white and they pass from the white ramus that is why it is called white, while the postganglionic are unmyelinated and appear grey and they pass from the grey ramus thus it is called grey.

Some preganglionic fibers that enter ganglia pass without any synapse at the paravertebral ganglia and continue to some ganglia located in the abdomen known as prevertebral ganglia (as explained above in step number 6), where they have the synapse with the second neuron. There are three unpaired prevertebral ganglia: celiac, superior mesenteric and inferior mesenteric ganglia. * Some preganglionic fibers pass without synapse in paravertebral ganglia and celiac ganglion. These fibers continue to adrenal gland where they synapse onto chromaffin cells. These cells liberate epinephrine into blood stream. As you see in the pictures bellow

Adrenal medulla= adrenal gland = suprarenal gland:

mainly secretes epinephrine (adrenaline).And norepinephrine (noradrenaline)

*****one neuron passes to the effector without any synapse (Synapse in gland)*****



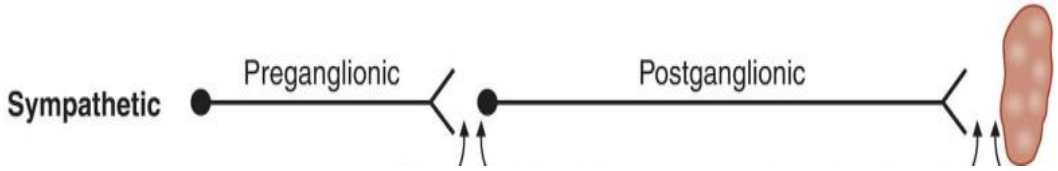
Convergence and divergence in sympathetic: In convergence: the cell body of the postganglionic neuron can receive signals from more than one preganglionic neuron. In divergence the preganglionic neuron can synapse with more than one postganglionic neuron.

Synaptic organization of sympathetic ganglia: Individual postsynaptic neuron in vertebral ganglia can receive signals from many preganglionic fibers (convergence) and one preganglionic neuron can relay impulse to many postganglionic neurons at different segmental levels (divergence). This organization of the sympathetic system induces widespread effects on target cells innervated by sympathetic postganglionic fibers.

The question is why do we want to have one preganglionic synapsing with more than one postganglionic, and vice versa? Because in "fight or flight" reactions, the body wants to accelerate the reactions of the targeted effectors, and they give a push to these reactions.

Simply the sympathetic division has a very wide effect on wildly distributed effectors like the blood vessels so we need to many postganglionic neurons to stimulate these effectors.

The preganglionic fibers of the sympathetic division are short while the postganglionic are long as you can see in the picture in page6(غلب حالك وارجعلها الله يرضى عليك)



this makes the divergence and convergence processes easier.

While it is the opposite in parasympathetic division. Long "pre" and short "post"

Parasympathetic nervous system: The preganglionic fibers arise in appropriate cranial nuclei and in segments S3 and S4 (sometimes S2, S5 also). These fibers leave the CNS in the III, VII, IX, and X (vagus) nerves for fibers of cranial origin and in pelvic nerve for fibers of sacral origin. The preganglionic fibers are long and go all the way to the effector organ where they synapse with the second postganglionic neuron located within the tissue of the effector organ or to a ganglion located very close to the effector organ. The axons of postsynaptic neurons are short.

You didn't understand half of it you just read, I know☺.

Simply let's start with the origin of the parasympathetic, it is from the cranial and sacral spinal cords so it is called **"craniosacral"** * a small note not included in the handout but good to know:(About 75 percent of all parasympathetic nerve fibers are in the vagus nerves (cranial nerve X)/ The sacral parasympathetic fibers are in the pelvic nerves, which pass through the spinal nerve sacral plexus on each side of the cord*.(in short: the sacral fibers are present in the pelvis as part of the sacral plexus)

The parasympathetic system, like the sympathetic system, has both preganglionic and postganglionic neurons but here the preganglionic fibers are long and go all the way to the effector organ where they synapse with the second postganglionic neuron located within the tissue of the effector organ or to a ganglion located very close to the effector organ. The axons of postsynaptic neurons are short.

Synaptic organization of parasympathetic nervous system:

In parasympathetic there is no or little branching of preganglionic fibers (divergence). The ratio of pre to post ganglionic neurons is 1:1 or 1:2. As a result of this arrangement, the parasympathetic actions tend to be more discrete and confined to the innervated organ.

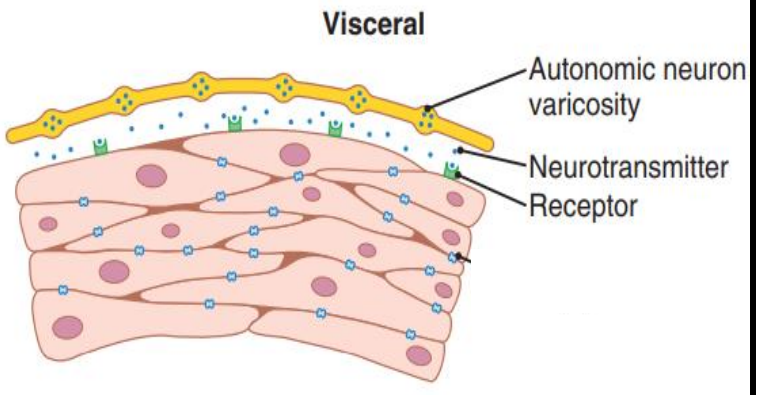
So simply The responses to parasympathetic stimulation are localized responses (you can say it effects a specific organ) while the sympathetic are more generalize responses. (the effector is widely distributed throughout the body like blood vessels)

Organization of the autonomic neuroeffector junction: The terminals of autonomic nerve fibers are unlike terminals of the somatic motor fibers (skeletal neuromuscular junction). The autonomic terminals are highly branched forming extensive network of fibers beaded with small swellings or varicosities. These varicosities are sites from where transmitter is released. The receptors on effector cells are scattered widely over the innervated organ. Unlike skeletal muscle, there is no specialized receptive region at the effector cell. The effect of ANS on these cells can be stimulatory or inhibitory. This effect depends on transmitter type, receptor subtype and changes in functional proteins induced in cell by binding of transmitter to its receptor.

What is said above is that the terminals of the autonomic nervous system are branched to form networks of fibers these fibers are full of swellings along their length called varicosities. it is the site from where neurotransmitters are released.

We have learned before that neurotransmitters are released at the terminals of neurons right? Well here this is not the case because many of the parasympathetic nerve fibers and almost all the sympathetic fibers merely touch the effector cells of the organs that they innervate so where these fibers pass over or near the cells to be stimulated, they usually have swellings called varicosities; it is in these varicosities that the transmitters vesicles are synthesized and stored. And this picture can help you understand the concept.

The receptors for these neurotransmitters are scattered widely over the innervated organ so there is no specialized receptive region as you can see in the pic. Unlike the skeletal muscles where there is a specific specialized receptive region.



The upcoming part needs a lot of حفظ Soooo حيلك شدلّي.

Functions of the ANS:

There are some changes in the external or internal environment that effects homeostasis so the body tries to adapt and control these changes by the ANS. Examples of changes are:

1- Light: constriction of the pupil to bright light (miosis), and dilation of pupil to low light (mydriases).

2 - Temperature: cutaneous vasodilation and sweating in a warm environment, and vasoconstriction in cold.

3- Stress: The ANS (mainly the sympathetic and the adrenal medulla) mediates the immediate response (fight or flight response) to threatening stimuli.

All of these changes are controlled by the ANS

EXAMPLE OF ADAPTING WITH CHANGES IN EXTERNAL ENVIRONMENT:

Imagine that you are in a forest and you face a bear, so you have two choices: fight or run (flight). In the previous scenario, the body is going to increase the breathing to supply the muscles (heightening the rate of CO2 and O2 exchange). Also speeds up metabolic reactions to generate more nutrients to supply the whole body. (carbs and other energy-rich macromolecules breakdown will increase).

Generally, “fight or flight” reactions are group of reactions that take place in body response to face the terrifying things or for getting stressed (harmful stimulus), in addition they push the body to speed up its metabolic processes, as well as the following reactions:

Sympathetic (fight or flight) Example of adaptation to external stimuli (Fight and Flight Reaction)

1– increasing heart rate and force of contractions; to deliver more blood to cells.

2 – mydriasis, (dilation of pupils).

3 – pallor (pale of fear): paling of the face or the skin; happens as a result of decreasing the amount of blood that goes to the skin. In “fight and flight” reactions the blood circulation is redistributed (higher amount of blood is directed to muscles, lower amount is directed to skin and unnecessary tissues in the response. (vasodilation for muscles blood vessels and vasoconstriction for unnecessary tissues’ vessels).

4 – goose pimpling: contraction of the smooth muscles that are found in the root of the hair which causes hair erection.

5 – cold sweat (it is cold because of the low amount of blood that is delivered to the skin)

6 – dry mouth: inactivation of salivary glands THE BODY is trying to shut down all unnecessary tissues by vasoconstriction the blood vessels that supplies these tissues.

* On the other hand during ordinary situation the parasympathetic division conserves and restores, it:

• Slows heartbeat • Decreases respiratory rate • Stimulates digestion • Removes waste • Stores energy.

This is a general idea of what the ANS does. now let’s speak about each division

In detail

Effects of sympathetic stimulation (fight or flight reactions):

As we said before this division effect tissues that are widely distributed throughout the body like blood vessels and sweat glands and smooth muscle cells of hair follicles these structures are only enervated by the sympathetic division.

1- Blood pressure: There are many things involved in regulating blood pressure (vessels, hormones, function of the heart itself and the body fluid) We'll focus on diameter of vessels … Sympathetic is the major controller of the diameter of vessels by vasodilation it decreases blood pressure. (remember pressure equals force/area so when we increase the diameter of blood vessels we increase the cross sectional area so we decrease the pressure).

Dr. Mohammed Hussein sends his regards☺

2-Body temperature: by the sympathetic effects on cutaneous blood vessels and sweat glands. it is related to vasodilation and vasoconstriction

-Dilation: we redirect the blood flow away from the skin so we lose temperature

And we activate sweat glands to lose temperature as well.

- Constriction: we redirect the blood flow toward the skin +no sweating.

3-Cardiovascular system: effects on vessels will result in redistribution of blood by enhancing blood flow to skeletal muscle and reducing blood flow to skin and mesentery.

4-Effects on heart: it increases heart rate, and causes more powerful contraction (increasing the force of contraction) which leads to more blood distribution to our tissues, more oxidation of these tissues and increasing "cardiac output": is the amount that can be measured (volume of blood pumped per minute)

5-Respiratory system: causes relaxation of bronchial muscle which result in bronchodilation, getting more oxygen to the smooth muscles, we get more airflow for the lungs and better oxygenation for the blood.

6-Digestive system: inhibition of motility and secretion, one of the aspects is dry mouth.

7-Metabolic effects: metabolic is the chemical processes that the body uses to convert organics to energy, by (SNS): mobilization of glucose (creation and breakdown), Increased lipolysis (lipid breakage), and Increased metabolic rate.

Effects of Parasympathetic stimulation (rest and digest reactions):

1-Gastrointestinal system: increases motility and secretory activity.

2-Glands: increases secretory activity (but remember sweat glands are under sympathetic control).

3- Heart: decreases rate of contraction (bradycardia). At conductive tissue! We have slow depolarization contraction potential by increasing parasympathetic stimulation, the rate of slow depolarization become more slow, so in this case the number of action potential generated per minute will be less. While by sympathetic stimulation we are increasing the rate of slow depolarization and we got more frequent generation of action potential. *To express these effects in another way, sympathetic stimulation increases the effectiveness of the heart as a pump, as required during heavy exercise, whereas parasympathetic stimulation decreases heart pumping, allowing the heart to rest between bouts of strenuous activity*.

4-Pupil: control pupil diameter by papillary light reflex (miosis) → (regulates the amount of light falling on retina).

5.Accommodation of the lens for near vision.

6.Voiding the urinary bladder (micturition).

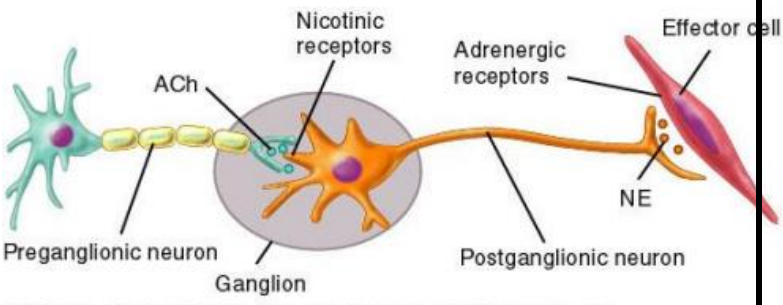
Notes from 3dwi☺:the doctor said during the lecture that the SNS is acting over the cardiac muscle to increase the force of contraction while the PSNS has no effect on the force of contraction, it is acting only on the conductive tissue not the cardiac muscle itself and I don't think that this is the case but the doctor said this so I wrote it here. Also the SNS and PSNS act on the eye on two different aspects they can either cause dilation or constriction of pupil to adjust to bright light (constriction of pupil) or low light (dilation of pupil). or they can affect the lenses of the eyes, the PSNS is the main controller here, it adjusts the convexity of the lens to help you focus on close subjects (near vision).

MOLECULAR BASIS OF PHYSIOLOGICAL ACTIONS OF THE ANS:

Transmitters: At ganglion: preganglionic neurons of both sympathetic and parasympathetic release acetylcholine (Ach).

But at the effector organ the case is different; At effector organs:

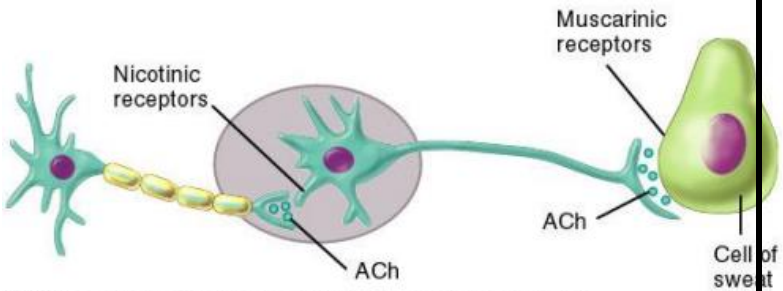
First, at ganglion: preganglionic neurons of both sympathetic and parasympathetic release Acetylcholine (Ach) and causing activation of the second neuron (the postganglionic neuron).



(a) Sympathetic division–innervation to most effector tissues

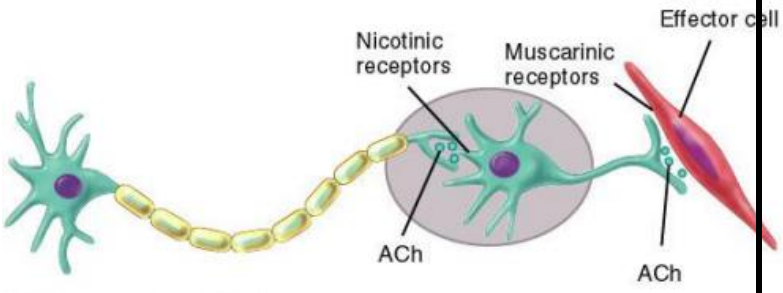
Now, while the second neuron is active.

the parasympathetic postganglionic neurons release also Acetylcholine to the effector cells.



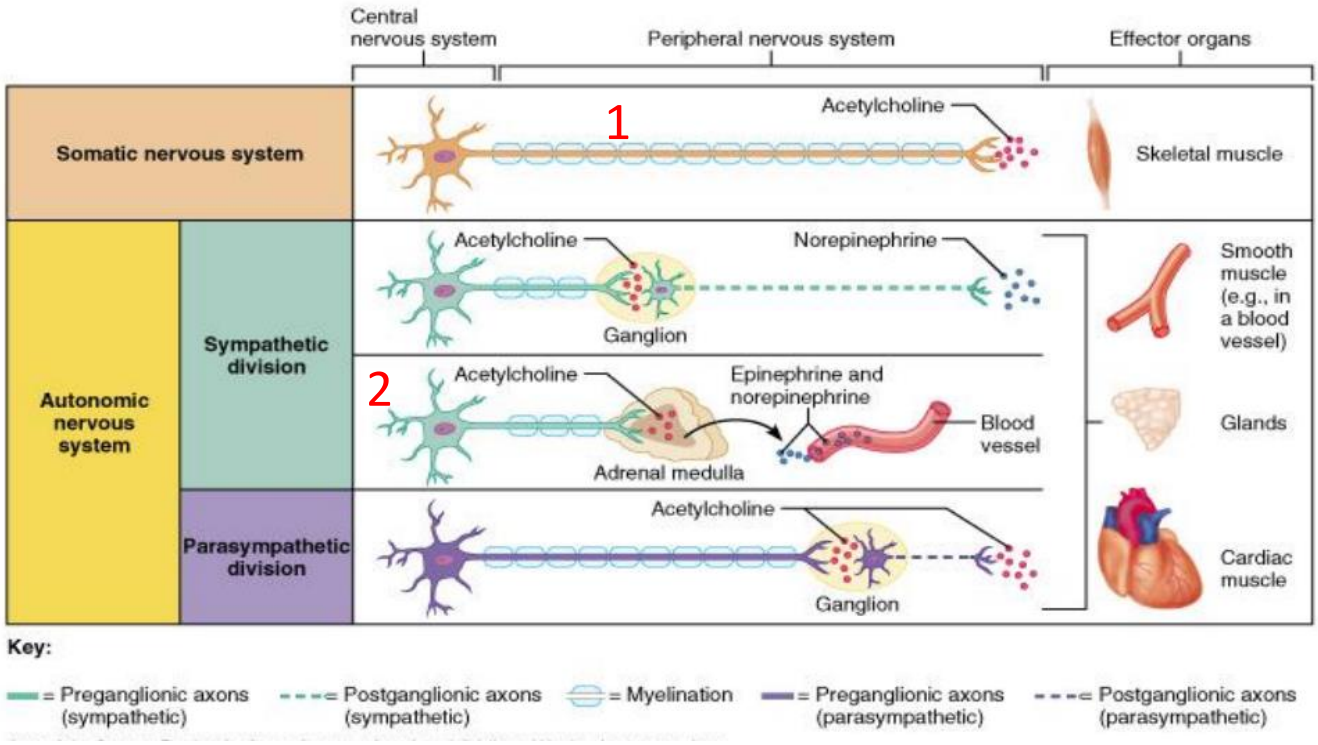
(b) Sympathetic division–innervation to most sweat glands

While the postganglionic neurons of sympathetic release norepinephrine to the effector cells except the postganglionic neurons that innervates sweat glands and piloerector muscles (small muscles attached to hair follicles), they release Ach Instead of norepinephrine.



(c) Parasympathetic division

The released Ach by parasympathetic system is inactivated by breakdown by acetylcholinesterase (an enzyme that breaks down Ach). Norepinephrine is inactivated by recapture by postganglionic nerve varicosities.



*you can see in this picture that the somatic fibers(1) also release Acetylcholine like the parasympathetic fibers, and(2) the sympathetic fibers that innervates the suprarenal gland (adrenal gland) release Acetylcholine(like sweat glands). The fibers that innervate adrenal gland don't pass throw any ganglia; so there are no postganglionic fibers. Keep in mind that adrenal gland is an endocrine gland that releases high concentration of epinephrine and low concentration of norepinephrine to the blood stream.

Receptors and signal transduction mechanisms: Receptors are found at postsynaptic or post junctional membranes and interact with transmitters released from the nerve terminals. These receptors function as coding system and they have high degree of specificity. The nature of response elicited in a particular tissue to a given transmitter is very precise and depends on the properties of receptor and the signaling mechanisms employed in that tissue. As we have taken before the receptors have high specificity for their ligands (the neurotransmitters), and the nature of response in a particular tissue depends on the type of receptor and the tissue. As we have taken before with Dr. eba'a

There is different type of receptors on each target, these receptors are:

1- Receptors on the postganglionic neurons (at ganglia) are called **nicotinic receptors.**

2- Receptors on the parasympathetic targets are called **muscarinic receptors.**

3-Receptors on the sympathetic targets are called **adrenergic receptors.**

Receptors at ganglion: On post synaptic membrane of sympathetic and parasympathetic there are **nicotinic receptors**. *a side note not mentioned in the hand out: nicotinic are also found on skeletal muscles and adrenal medulla*. These receptors are excited by acetylcholine. The drug nicotine can also stimulate these receptors.

This receptor is similar but not identical (they have different subunit structures) to nicotinic receptor of the neuromuscular junction. This receptor gates ligand gated Na+ channel. Activation of this receptor will cause depolarization on postsynaptic membrane.

So simply you can say that it is a ligand gated Na^+ channel so when the ligand (Ach) binds to it, it opens and causes depolarization of the post synaptic membrane and action potential is generated in it.

Receptors on effector cells:

- Muscarinic receptors:

These cholinergic receptors lie on effector cells of parasympathetic neuro-effector junctions. They differ from nicotinic receptors found on ganglia and neuromuscular junction.

- Many muscarinic receptors have been known (M1-M5) at these junctions. All these receptors are coupled to G protein.

-For example, the inhibitory receptor that is found in the heart (M2) is coupled to G_i protein, which inhibits adenylyl cyclase activity, which in turn decreases cyclic AMP and slows the heart rate (we will talk about the mechanism of that later on) this Gi protein is also linked to K+ channels, activation of this receptor will slow the rate of depolarization so it decreases heart rate.

So once Ach binds to M2 receptor it activates a K^+ channel and slows the depolarization of the conductive tissue of the heart so we are decreasing the number of beats per minute (decrease heart rate).

But we have other types of these receptors

smooth muscle (M3), and glands (M3).

are inhibitory in the heart (e.g., decreased heart rate, decreased conduction velocity in AV node).

are excitatory in smooth muscle and glands (e.g., increased GI motility, increased secretion).

1. heart: Gi protein, inhibition of adenylate cyclase, simultaneously it opens K+ channels, slowing of the rate depolarization, and decreased heart rate.

2. smooth muscle and glands: G_q protein, stimulation of phospholipase C, and increase in IP3 and intracellular [Ca 2+].

<u>Remember:</u> Gq proteins: a family of G-protein that activates phospholipase C.

Phospholipase C: membrane associated enzyme responsible for the cleavage of phospholipids and convert it to DAG and IP3.

This enzyme increases production of inositol-1,4,5- trisphosphate (IP3). IP3 causes release of Ca++ from internal stores in muscle or glands, causing contraction or secretion.

Nicotinic receptors are stimulated by Nicotine, and muscarinic receptors are activated by muscarine which is found in a type of toxic mushroom, so if someone has been ingested with it, all muscarinic receptors will be activated.

Agonist is a chemical that binds to a receptor and activates the receptor to produce a biological response. (muscarine and nicotine are agonists for Acetylcholine but for different receptors)

So when someone gets ingested with muscarine the receptors will be activated and he will develop obvious symptoms.

-stimulation of secretory activity: salivation, tearing, sweating, nasal and bronchial secretion.

-Increase gastrointestinal tract motility→ vomiting and diarrhea.

-Contraction of urinary bladder →urination.

-Slowing of the heart →Bradycardia.

So how do we reverse these effects when someone eats this mushroom by accident.

We use a drug (antagonist) to block the muscarinic receptors which is called Atropine [from a plant (*atropa Belladonna*)]. which induces reversal effects of muscarinic poisoning.

Effects of atropine include:

-Inhibition of glandular secretions→ dry mouth, dry eyes, and dry nasal passages. Tachycardia. (increase heart rate).

Loss of pupillary light reflex.

Loss of ability to focus the lens for near vision.

The doses are given to the patient until we notice a change reversing of the intoxication effects.

- Adrenergic receptors: These receptors respond to catecholamines (epinephrine (EP) and norepinephrine (NE)). Two types of receptors are known alpha (a) and beta (B) receptors.

Alpha receptors: The alpha receptors are subdivided into **a1 and a2 receptors**. **The alpha 1 (a1)** receptor is widely distributed on smooth muscles with the exception of bronchial muscle. NE and EPI are about equally effective on these receptors. Stimulation of this receptor produces excitation. This effect involves Gq protein, stimulation of phospholipase C, IP3 production, and release of Ca++ from intracellular stores. Some (a1 are coupled to Ca++ gated channels).

so this receptor causes excitation by increasing intra cellular Ca^{+2} level so it does by either being coupled to G_q protein or being coupled to Ca^{+2} channel.

Alpha II → are found on sympathetic postganglionic nerve terminals. These receptors are important for self-inhibition of NE release (negative feedback).

Alpha II heteroreceptor → found in non-adrenergic neurons, negatively coupled to Gi proteins, reduce the synthesis of c-AMP and inactivate these neurons.

Beta receptors:

these receptors are more sensitive to EP& NE than alpha, (lesser concentration needed for a response to occur)

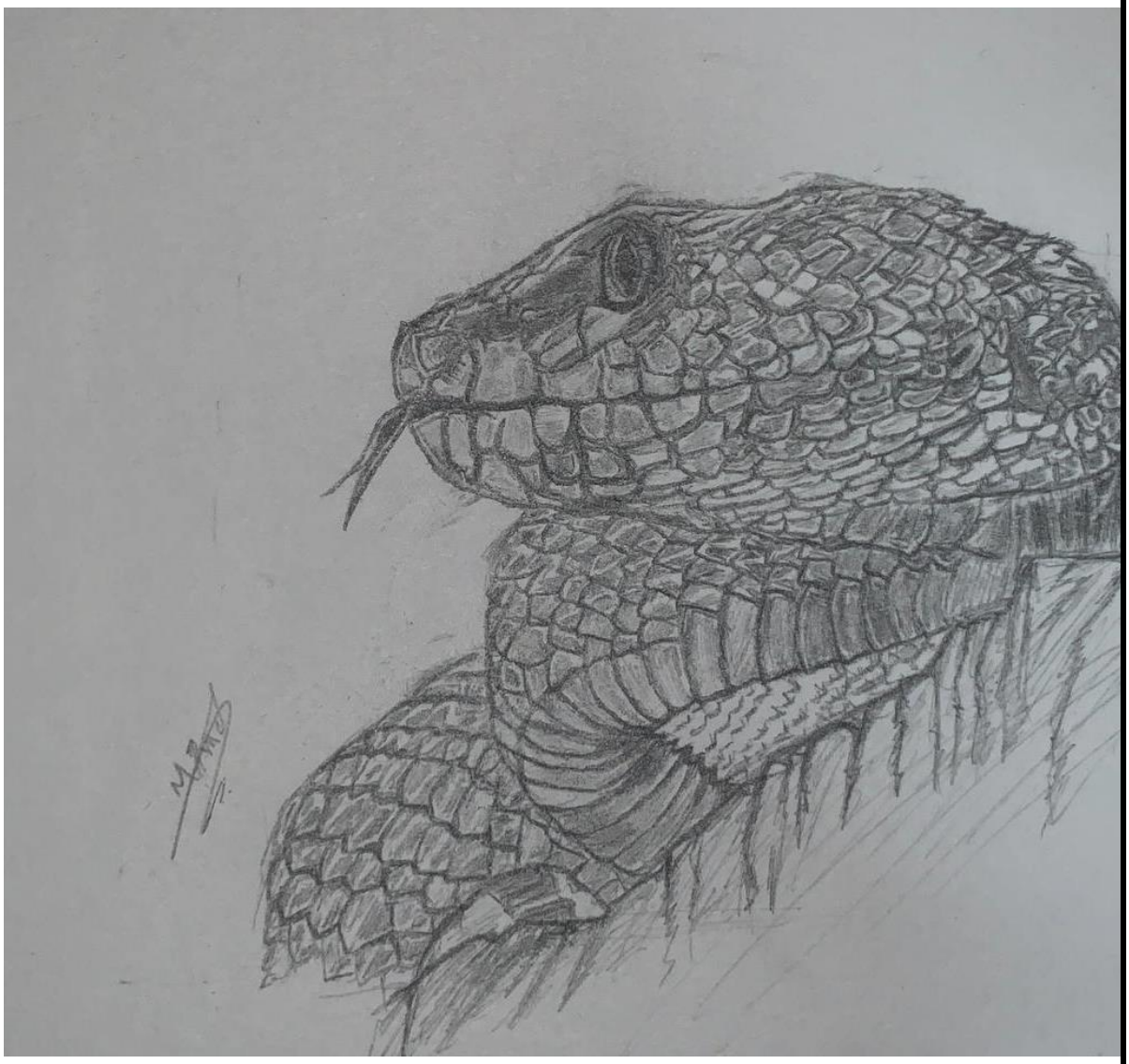
Beta 1 (B1) receptors: found on heart and produces excitation in the heart.

Beta II(B2) → an inhibitory receptors found in smooth muscle cells like in the bronchial muscle cells, the gastrointestinal tract, blood vessels supplying skeletal muscles. The B2 receptors are preferentially activated by EPI rather than NE. Both receptors are coupled to Gs protein, and increase c-AMP. This will result in subsequent activation of protein kinase and phosphorylation of one or more proteins. The response elicited depends on the role of phosphorylated proteins. All subclasses of adrenergic receptors can be blocked by specific blocking agents (antagonists).

B1 blockers are useful as *antiarrhythmic* drugs (**prevent and treat abnormal heartbeats (arrhythmias)**.

. B2 selective agonist (produce activation of B2 receptor) will dilate bronchi. This agonist is useful in *asthma. (***Asthma** is a condition in which your airways narrow and swell).

A quick reminder of your true nature. ☺



تراني اكتفيت بما ليس يكفي

أحقّ اعتباري بأنّي كغيري سأحيا وأغدو عظامًا ترابًا

وهل يعتريني الزّمان بظلمٍ فيخفي ضيائي

ويدفن شوقي لكلّ المعالي الّذي دون حدِّ

ويطفئ نارًا تلهّبُ فيَّ وتزعج نفسي لكلّ غمارٍ عليٍّ

تُعلّق أمريَ بعد ابتداءٍ ودونَ انتهاءِ.