بسم الله الرحمٰن الرحيم

(وَفَوْقَ كُلِّ ذِي عِلْمٍ عَلِيمٌ)

Pharmacology | FINAL 2

Introduction to ANS pt.2

Written by : Leen Alzoubi
Maram Darweesh

Reviewed by : Lana Alyaseen

وَلِلَّهِ الْأَسْمَاءُ الْحُسْنَى فَادْعُوهُ بِهَا

المعنى: المحمود في جميع أفعاله وأقواله، وصفاته وأسمائه، وشرعه وقدَرِه، يُحمد على كلّ حال، وهو المستحق للحمد والثناء لكمال صفاته ولكثير إحسانه إلى الخلق.

الورود: ورد في القرآن (١٧) مرة.

الشاهد: ﴿ إِنَّهُۥ حَمِيدٌ مَّجِيدٌ ﴾ [هود:٧٣].

٥١

اضغط هنا لشرحٍ أكثر تفصيلًا

Autonomic Receptors

1. **Cholinoceptors (Cholinergic): Receptors stimulated by acetylcholine.** Two types:
   - 1) **Muscarinic and** 2) **nicotinic receptors stimulated by the alkaloids muscarine and nicotine, respectively.**
2. **Adrenoceptors (Adrenergic): Receptors stimulated by catecholamines such as norepinephrine (noradrenaline).** Alpha and beta receptors
3. **Dopamine receptors (Dopaminergic): Receptors stimulated by dopamine.** Mainly D1 receptor

Autonomic Receptors

The professor will focus on the critical and clinically important receptors and mechanisms shown in the table. Other information is valid but will be ignored temporarily for this course.

TABLE 6–2 Major autonomic receptor types.

Receptor Name	Typical Locations	Result of Ligand Binding
Cholinoceptors		
Muscarinic M_1	CNS neurons, sympathetic postganglionic neurons, some presynaptic sites	Formation of IP_3 and DAG, increased intracellular calcium
Muscarinic M_2	Myocardium, smooth muscle, some presynaptic sites; CNS neurons	Opening of potassium channels, inhibition of adenylyl cyclase
Muscarinic M_3	Exocrine glands, vessels (smooth muscle and endothelium); CNS neurons	Like M_1 receptor-ligand binding
Muscarinic M_4	CNS neurons; possibly vagal nerve endings	Like M_2 receptor-ligand binding
Muscarinic M_5	Vascular endothelium, especially cerebral vessels; CNS neurons	Like M_1 receptor-ligand binding
Nicotinic N_N	Postganglionic neurons, some presynaptic cholinergic terminals; receptors typically contain two α3 and one β4 type subunits in addition to γ and δ subunits	Opening of Na^+, K^+ channels, depolarization
Nicotinic N_M	Skeletal muscle neuromuscular end plates; receptors typically contain two α1 and β1 type subunits in addition to γ and δ subunits	Opening of Na^+, K^+ channels, depolarization

⭐ M1 Receptor – Locations

1) CNS Neurons (Anything related to CNS is not our current focus in this pharmacology course)

2) Sympathetic Postganglionic Neurons (Important Clinical Focus)

When studying autonomic neurotransmission, we learned that:

- Presynaptic receptors can regulate neurotransmitter release.
- Autoreceptors regulate release from the same neuron.
- Heteroreceptors regulate release from a different type of neuron.

M1 receptors serve as heteroreceptors on sympathetic postganglionic nerve terminals.

Physiological Explanation

- Inside cardiac intramural autonomic ganglia, there are synaptic interconnections (anastomoses) between:
  - Parasympathetic (vagal) neurons
  - Sympathetic postganglionic neurons

  Anastomosis in this context means: local functional interconnections or synaptic cross-talk, not classical anatomical pre→post autonomic chains.

M1 as Heteroreceptors in the Heart

Inside cardiac intramural ganglia:

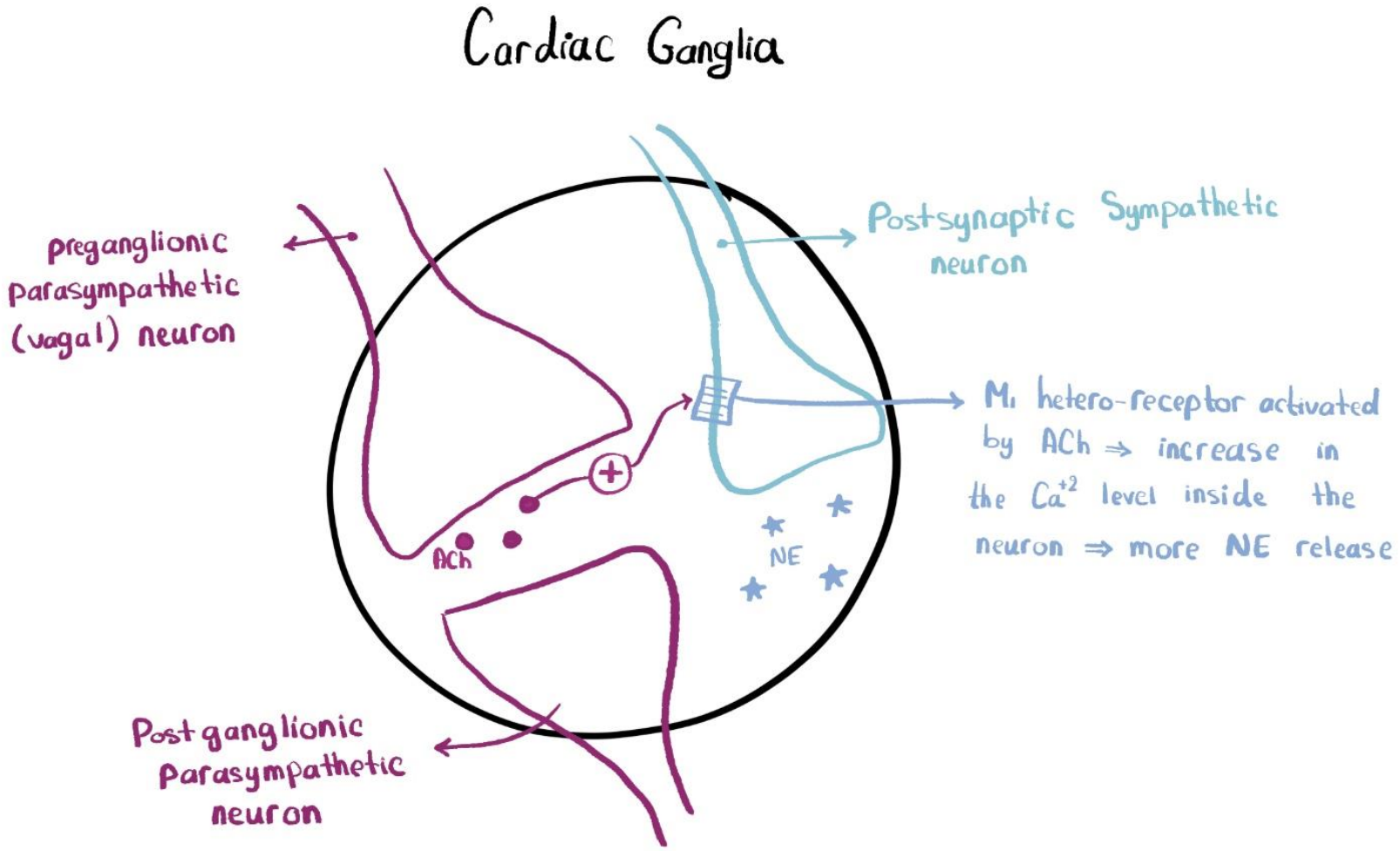
- Preganglionic parasympathetic neurons (vagal fibers) release ACh and synapse with parasympathetic postganglionic neurons.
- These parasympathetic postganglionic neurons also release ACh locally near sympathetic terminals.
- Sympathetic postganglionic terminals have presynaptic M1 heteroreceptors.
- When ACh (released from the preganglionic parasympathetic neuron) binds to these M1 receptors:
  - It increases NE release from sympathetic terminals.
  - This results in more sympathetic stimulation of the heart.

Mechanism of M1 Receptor Activation

- Phosphatidylinositol breakdown
- Release of three second messengers:
  - IP_3, DAG, intracellular Ca^{2+}

  Because three second messengers are generated, M1 receptors can elicit diverse cellular responses, depending on the tissue and signaling pathway.

This is to help you visualize M1 receptors found in the sympathetic post-ganglionic neurons



M2 Receptors – Location
- Myocardium
- Smooth muscle
- Some presynaptic sites (meaning they are auto receptors)

Mechanism:
- Opening of potassium channels
- Inhibition of adenylyl cyclase

M3 Receptors – Location
- Mainly in the exocrine glands (salivary, GI glands, pancreatic glands, and respiratory glands)
- Endothelium of blood vessels and smooth muscle of blood vessels

Mechanism:
- Like M1

M4 Receptors – Location
- Mainly CNS (not important)
- Possibly vagal nerve endings

Mechanism:
- Like M2

M5 Receptors – Location
- Vascular endothelium

Mechanism:
- Like M1

Nicotinic Receptors

1. Nicotinic Nm Receptors

- Located in skeletal muscle and involved in motor function.
- Not relevant to our current lecture.

2. Nicotinic Nn Receptors

- Located in autonomic ganglia, specifically on the post-ganglionic neuron, as previously discussed.
- A subset of these receptors may also be found presynaptically.

Mechanism of Action:

- Activation leads to opening of sodium and potassium channels, resulting in neuronal depolarization.

Autonomic Receptors

Adrenoceptors		
$Alpha_1$	Postsynaptic effector cells, especially smooth muscle	Formation of IP_3 and DAG, increased intracellular calcium
$Alpha_2$	Presynaptic adrenergic nerve terminals, platelets, lipocytes, smooth muscle	Inhibition of adenylyl cyclase, decreased cAMP
$Beta_1$	Postsynaptic effector cells, especially heart, lipocytes, brain; presynaptic adrenergic and cholinergic nerve terminals, juxtaglomerular apparatus of renal tubules, ciliary body epithelium	Stimulation of adenylyl cyclase, increased cAMP
$Beta_2$	Postsynaptic effector cells, especially smooth muscle and cardiac muscle	Stimulation of adenylyl cyclase and increased cAMP. Activates cardiac G_i under some conditions.
$Beta_3$	Postsynaptic effector cells, especially lipocytes; heart	Stimulation of adenylyl cyclase and increased cAMP[1]
Dopamine receptors		
D_1 (DA_1), D_5	Brain; effector tissues, especially smooth muscle of the renal vascular bed	Stimulation of adenylyl cyclase and increased cAMP
D_2 (DA_2)	Brain; effector tissues, especially smooth muscle; presynaptic nerve terminals	Inhibition of adenylyl cyclase; increased potassium conductance
D_3	Brain	Inhibition of adenylyl cyclase
D_4	Brain, cardiovascular system	Inhibition of adenylyl cyclase

[1] Cardiac β_3-receptor function is poorly understood, but activation does *not* appear to result in stimulation of rate or force.

Adrenergic Receptors

1) Alpha-1 (α_1) Receptors

Location

- Postsynaptic receptors in:
  - Smooth muscle of blood vessels
  - Respiratory smooth muscle
  - GI tract smooth muscle

Mechanism

- Formation of IP_3, DAG, Ca^{2+}
- Phosphatidyl-inositol breakdown mechanism

2) Alpha-2 (α_2) Receptors

Location

- Mainly presynaptic inhibitory autoreceptors
- Found in other non-presynaptic sites as well (less important)

Function

- Presynaptic inhibitory auto-receptors that mediate negative feedback inhibition of catecholamine release

Mechanism

- Inhibition of adenylyl cyclase
- Decreased cAMP production

3) Beta-1 (β1) Receptors

Location (Three important Sites)

- Heart
- Juxtaglomerular apparatus of renal tubules (Responsible for renin secretion, Renin is needed for activation of angiotensin Angiotensinogen → Angiotensin I → Angiotensin II)
- Presynaptic stimulatory auto receptors(They stimulate further release of catecholamines)

Mechanism

- Activation of adenylyl cyclase
- Increased cAMP

4) Beta-2 (β2) Receptors

Location

- Smooth muscle of blood vessels, especially in the skeletal muscular system (blood vessels in the muscles have β2 receptors)
- Also found in the respiratory tract (bronchial smooth muscle contains β2 receptors)

Metabolic Role

- Play a role in metabolic processes that result in the formation of glucose such as Gluconeogenesis and Glycogenolysis in the liver

Mechanism

- Activation of adenylyl cyclase
- Increased cAMP

5) Beta-3 (β3) Receptors

Location

- Found in lipocytes (fat cells)

Function

- Affect metabolic processes in the lipocyte

Dopamine Receptors

Dopamine Receptors

- There are five dopamine receptors in total.
- All of them are central (CNS) receptors.
- For our purposes, we will focus only on D1 and D5.

Location

- D1 and D5 receptors are also found in the smooth muscle of the renal vascular bed, especially:
  - Smooth muscle of blood vessels in the kidney

Mechanism

- Stimulation of adenylyl cyclase
- Increased cAMP

Just memorize what was mentioned in lecture no need for the extra details in the table

Presynaptic Regulation = Auto receptors

There is negative and positive feedback regulation

- **Negative feedback control is found at the presynaptic level of autonomic function, such as:**
- **Presynaptic α_2-adrenoceptors when activated by norepinephrine and similar substances lead to reduction of further norepinephrine release.**

→ Similar drugs (agonists) such as methyl dopa and clonidine and other hyper-tensive drugs

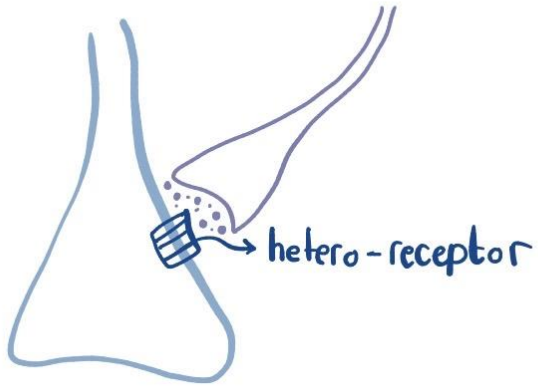
Presynaptic Regulation

- **Conversely, Presynaptic β-adrenoceptors when activated by norepinephrine and similar substances facilitate further norepinephrine release.**
- **These receptors are called autoreceptors.**
- **Heteroreceptors may also be involved in presynaptic regulation. They are activated by substances released from other nerve terminals.**

Auto means that the neurotransmitter regulates its own release by regulating its own receptors

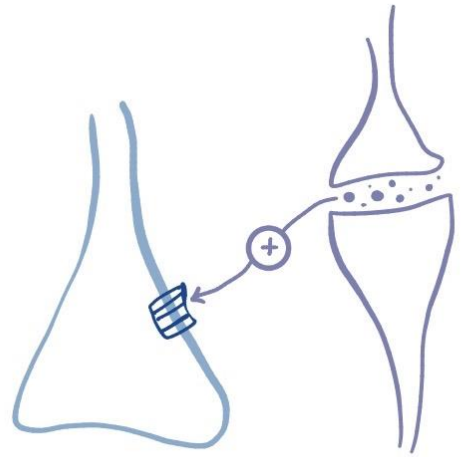
Heteroreceptors can be activated either by local diffusion of neurotransmitters or by neurotransmitter spillover from nearby synapses, allowing one neuron to modulate transmitter release from another neuron without requiring a direct synaptic connection. However, activation may also occur through real synapses when neurons are functionally connected, meaning both diffusion and true synaptic communication can regulate heteroreceptor activity.

Activation of Hetero-Receptors



1. Direct Synapse

2. local diffusion



3. Spill over from nearby Synapses

Presynaptic Regulation

Fibers= neurons

- Some vagal fibers (parasympathetic) in the myocardium synapse on sympathetic noradrenergic nerve terminals and inhibit norepinephrine release. Through cholinergic hetero-receptors
- Alternatively, some substances move to these receptors from the blood or nearby tissues. Some examples are mentioned in the next slide:

Presynaptic Regulation

5-Hydroxytryptamine

Hetero-receptors

1. Serotonin (5-HT) stimulation of its receptors at cholinergic preganglionic sites inhibits cholinergic transmission.
2. Adenosine and ATP stimulation of their receptors (P_1 and P_2 respectively) at adrenergic autonomic neurons inhibit adrenergic function. Also inhibiting norepinephrine release
3. Angiotensin II stimulates its receptor (AT_2-1) at adrenergic nerve terminals & stimulates adrenergic transmission. Causing further stimulation of the sympathetic nervous system

Angiotensin II and Adrenergic Function

- This topic is particularly important clinically because we have angiotensin II blockers and β-blockers, both of which influence blood pressure regulation.
- Angiotensin II increases adrenergic transmission, meaning it enhances the activity of sympathetic nerve terminals (as mentioned in the previous slide).
- The sympathetic nervous system increases the formation of angiotensin II through renin (this was mentioned while discussing beta1 receptor) , and both systems together contribute to increasing blood pressure
  (sympathetic transmission + angiotensin II).
- Angiotensin II is a more potent vasoconstrictor than norepinephrine, and it can produce severe vasoconstriction even at low concentrations.

Postsynaptic regulation

1. **Up-regulation of receptors: Increased number of receptors upon continued decreased receptor activation by antagonist.** Here there will be increasing in the action
2. **Down regulation of receptors: Decreased number of receptors upon continued increased receptor activation by agonist.** Here will be decreasing in the action.

When an **antagonist** blocks a receptor, it decreases the receptor's activation. The cell senses this decreased activity as if it is being "restricted" or "not allowed to work." In response, the cell compensates by **upregulating** the receptors.

The first step in upregulation is the increased movement of existing receptors to the cell surface through **exocytosis**. After that, the cell increases **gene transcription**, **translation**, and **synthesis** of new receptors. These newly formed receptors are then inserted into the cell membrane. This entire process is a reflex adaptation to the reduced stimulation caused by the antagonist.

As a result, the number of receptors on the cell surface increases. Because of this, the effect of the antagonist becomes weaker over time, since there are now more receptors available than the antagonist can block. However, if the antagonist is suddenly stopped, the natural agonist in the body will suddenly find a large number of upregulated receptors available. This leads to an exaggerated physiological response.

For example:
A hypertensive patient may experience **rebound hypertension**.
A patient with angina pectoris may be at risk of **myocardial infarction**.

Therefore, drugs that cause upregulation must be discontinued **gradually**. This process, known as **tapering**, gives the cell time to reverse the upregulation and restore normal receptor levels

Downregulation occurs when a receptor is continuously stimulated by an agonist. With constant stimulation, the cell becomes "overworked," so it initiates protective mechanisms to reduce its activity.

The first step is **invagination and internalization** of the receptors. By pulling receptors into the cell, they are no longer available for the agonist to bind. This reduces the stimulus on the cell and prevents overstimulation and cellular fatigue.

A second mechanism occurs at the **genetic level**. Chronic agonist stimulation leads to **inhibition of gene transcription**, which decreases the synthesis of new receptors. As a result, fewer receptors are produced and inserted into the membrane.

These processes together cause **tolerance**—a gradual reduction in the drug's effect during long-term use. Although downregulation does not occur with all drugs, it is an important mechanism that can develop with repeated or chronic agonist exposure

Effects of Autonomic Nerve Activation

TABLE 6–3 Direct effects of autonomic *nerve* activity on some organ systems. Autonomic *drug* effects are similar but not identical (see text).

	Effect of			
	Sympathetic Activity		Parasympathetic Activity	
Organ	***Action***[1]	***Receptor***[2]	***Action***	***Receptor***[2]
Eye				
Iris radial muscle	Contracts	α_1	…	…
Iris circular muscle	…	…	Contracts	M_3
Ciliary muscle	[Relaxes]	β	Contracts	M_3
Heart				
Sinoatrial node	Accelerates	β_1, β_2	Decelerates	M_2
Ectopic pacemakers	Accelerates	β_1, β_2	…	…
Contractility	Increases	β_1, β_2	Decreases (atria)	M_2
Blood vessels				
Skin, splanchnic vessels	Contracts	α	…	…
Skeletal muscle vessels	Relaxes	β_2	…	…
	[Contracts]	α	…	…
	Relaxes[3]	M_3	…	…
Endothelium of vessels in heart, brain, viscera	…	…	Synthesizes and releases EDRF[4]	M_3, M_5[5]
Bronchiolar smooth muscle	Relaxes	β_2	Contracts	M_3

The eye contains three important structures: the iris, the ciliary muscle, and the tissues surrounding them.
The iris consists of two smooth muscles that control the size of the pupil:

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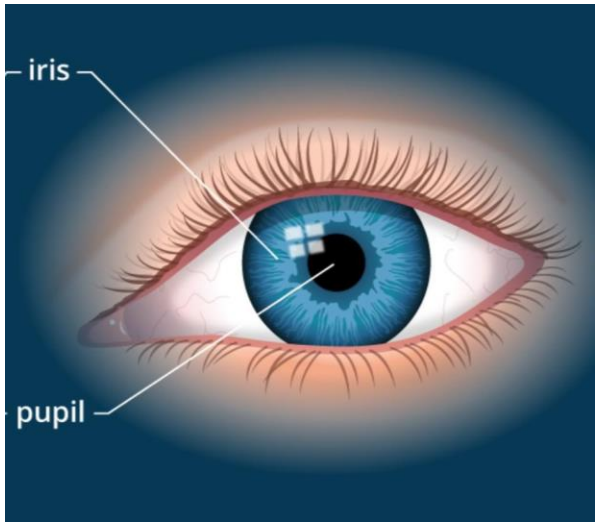
1. Iris Radial Muscle (Sympathetic – α1 receptlors)

The radial muscle looks like “sun rays” extending outward from the pupil.
It contains **only α1 adrenergic receptors** and **no parasympathetic receptors**.
When the radial muscle **contracts**, it pulls the iris outward.
This action **dilates the pupil** (mydriasis).



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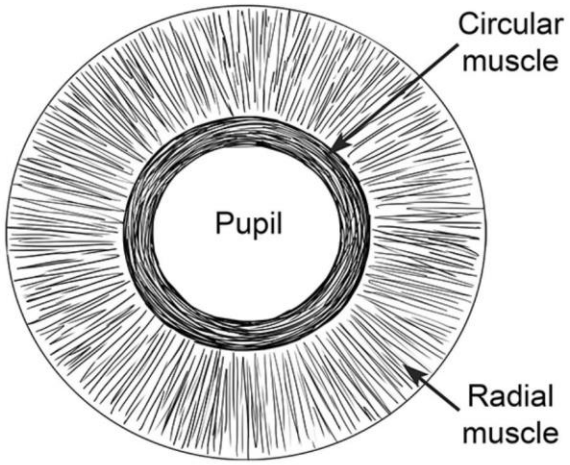
2. Iris Circular Muscle (Parasympathetic – M3 receptors)

This muscle forms a circular ring around the pupil.
It contains **M3 muscarinic receptors**.
When the circular muscle **contracts**, the pupil becomes **smaller** (miosis).
When it relaxes, the pupil widens.



Thus:
Radial muscle contraction → dilation (sympathetic, α1)
Circular muscle contraction → constriction (parasympathetic, M3)

3. Ciliary Muscle and Accommodation

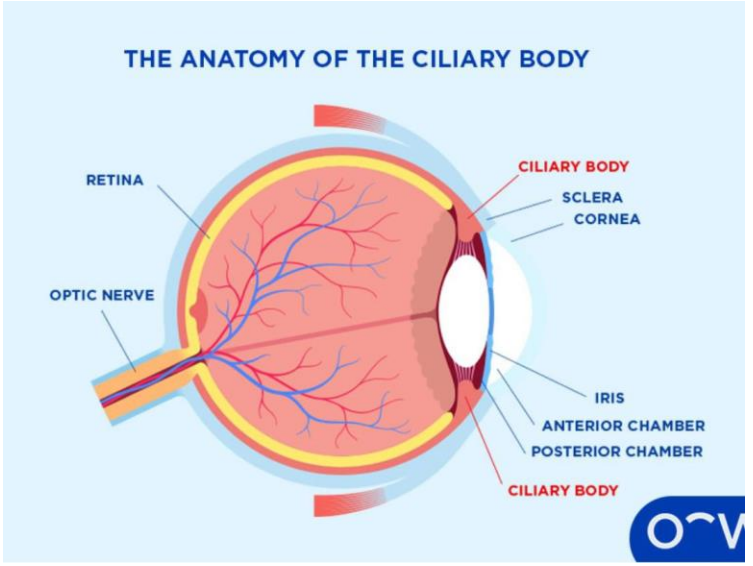
The ciliary muscle is responsible for accommodation, which means adjusting the lens to see objects at different distances.

Parasympathetic (M3 receptors):

Causes **contraction** of the ciliary muscle → accommodation for **near vision.**

Sympathetic (β receptors):

Causes **relaxation** of the ciliary muscle → accommodation for **far vision.**



Note the doctor said in the lecture **by mistake**

Contraction of the ciliary muscle → accommodation for far vision.

Relaxation of the ciliary muscle → accommodation for near vision.

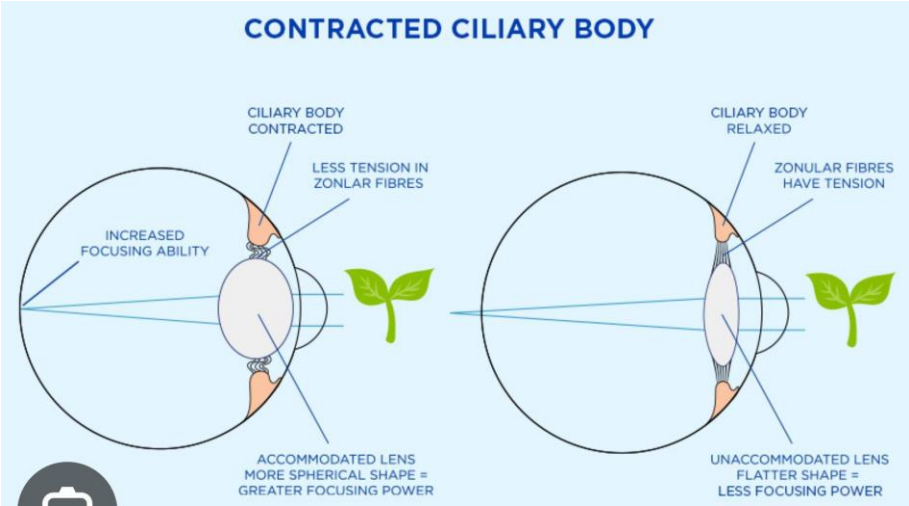
If you're still unsure, open this PDF on slide 12 click here

So:

Near vision → parasympathetic → contraction

Far vision → sympathetic → relaxation

People sometimes narrow their eyelids or use their fingers to limit the amount of light entering the eye, which reduces peripheral light rays and improves image sharpness for distant objects. This is a mechanical-optical adjustment and does not involve parasympathetic accommodation, which is responsible for near vision rather than distant vision.



Now, for the heart. The heart has three main functional components: the SA node, the myocardium (muscle), and the conduction system.
Beta-1 receptors increase the firing rate of the SA node, raise the heart rate, strengthen myocardial contraction, and enhance conduction through the AV node.

Ectopic pacemakers are non-SA-node foci capable of generating impulses when the SA node fails. Because there are multiple potential ectopic sites, each with its own rhythm, they often cause arrhythmias.

Parasympathetic M2 receptors have the opposite effect of beta-1 stimulation. They reduce SA node firing, decrease contractility, and slow conduction.
In summary: sympathetic activation (via beta-1 receptors) increases all cardiac activities, while parasympathetic activation (via M2 receptors) decreases them.

Let's talk about blood vessels and smooth muscle.
In the skin and splanchnic circulation, blood vessels contract through **alpha-1 receptors**. In contrast, blood vessels supplying skeletal muscle relax through **beta-2 receptors.**

Although blood vessels and smooth muscle do not have cholinergic innervation (they don't contain **parasympathetic** nerves)Note: here the doctor by mistake said that they don't contain sympathetic nerves, they do contain **cholinergic receptors**. These include **M2 receptors on smooth muscle** and **M3/M5 receptors on the vascular endothelium**.

Cholinergic receptors on **smooth muscle** cause **relaxation and vasodilation** when stimulated. Cholinergic receptors (which is stimulated by circulating Ach and like substances) located on the **endothelium** trigger the synthesis of **nitric oxide** (NO). NO is a potent vasodilator. Before its identity was known, NO was referred to as **endothelium-derived relaxing factor (EDRF).** Once produced, nitric oxide diffuses to the surrounding smooth muscle and causes relaxation.

In the airways, **bronchial smooth muscle** is relaxed by **beta-2 adrenergic receptors** and contracted by **muscarinic M3 receptors**.

By understanding these information , we can predict what receptor antagonists will do. And by understanding the action, we can know their therapeutic uses, and their potential adverse effects

Effects of Autonomic Nerve Activation

Organ	Effect of			
	Sympathetic Activity		Parasympathetic Activity	
	Action[1]	*Receptor*[2]	*Action*	*Receptor*[2]
Genitourinary smooth muscle				
Bladder wall	Relaxes	β_2	Contracts	M_3
Sphincter	Contracts	α_1	Relaxes	M_3
Uterus, pregnant	Relaxes	β_2	…	…
	Contracts	α	Contracts	M_3
Penis, seminal vesicles	Ejaculation	α	Erection	M
Skin				
Pilomotor smooth muscle	Contracts	α	…	…
Sweat glands			…	…
Eccrine	Increases	M	…	…
Apocrine (stress)	Increases	α	…	…
Metabolic functions				
Liver	Gluconeogenesis	β_2, α	…	…
Liver	Glycogenolysis	β_2, α	…	…
Fat cells	Lipolysis	β_3	…	…
Kidney	Renin release	β_1	…	…

[1]Less important actions are shown in brackets.

[2]Specific receptor type: α, alpha; β, beta; M, muscarinic.

[3]Vascular smooth muscle in skeletal muscle has sympathetic cholinergic dilator fibers.

[4]The endothelium of most blood vessels releases EDRF (endothelium-derived relaxing factor), which causes marked vasodilation, in response to muscarinic stimuli. Parasympathetic fibers innervate muscarinic receptors in vessels in the viscera and brain, and sympathetic cholinergic fibers innervate skeletal muscle blood vessels. The muscarinic receptors in the other vessels of the peripheral circulation are not innervated and respond only to circulating muscarinic agonists.

[5]Cerebral blood vessels dilate in response to M_5 receptor activation.

[6]Probably through presynaptic inhibition of parasympathetic activity.

Genitourinary system:
This includes the urinary bladder wall, the bladder sphincter, the uterus (specifically the pregnant uterus), and the male genitalia.

The **bladder wall** is relaxed by **beta-2 sympathetic (adrenergic) receptors.**
When the bladder wall relaxes, the bladder can hold more urine. Because it is relaxed, the **sphincter must be contracted** to prevent urine leakage.
The **bladder sphincter closes** due to **alpha-1 receptor stimulation**.

During **urination**, the opposite occurs:
The bladder **contracts** ,The sphincter **relaxes**
Both of these are **parasympathetic** actions.
So, urination—like defecation—is a **parasympathetic reflex**. The parasympathetic system is what becomes active when you go to urinate.

In summary:
Sympathetic → increases bladder capacity (by relaxing the bladder wall) and **closes** the sphincter.
Parasympathetic → contracts the bladder wall and **opens** (relaxes) the sphincter.

The **pregnant uterus** is not affected by parasympathetic activity.
However, as pregnancy progresses, **beta-2 receptor density increases** in the uterus.
This increase exists to relax the uterus, which helps prevent unwanted contractions that could lead to abortion (loss of the fetus). Doctors use this feature therapeutically: in cases of threatened premature labor or risk of early delivery, a **beta-2 agonist** can be given to relax the uterus, prevent contractions, and help prevent abortion.

The uterus also contains **alpha receptors**, but they are not functionally significant here.
The receptors that matter in the pregnant uterus are the **beta-2 receptors**.

For the **male genitalia**:
Sympathetic stimulation is responsible for **ejaculation**.
Parasympathetic stimulation is responsible for **erection**

The **pilo-motor smooth muscle** is what causes the hair to stand up. When this muscle contracts, the hair becomes erect. This contraction happens through **alpha receptors**, because the pilo-motor muscle contains alpha receptors.

Now, for the **sweat glands**, which come in different types. Sweat glands produce secretions. Normally, secretions are associated with **parasympathetic** activity. That is true for one type: the **eccrine glands**, which are stimulated **like** a parasympathetic response.(Although they are under sympathetic control) See the table in slide 27

The **apocrine glands**, on the other hand, are controlled by the **sympathetic adrenergic system**(**Note the doctor here make a mistake and he said that the apocrine glands are controlled by sympathetic cholinergic System, if you still unsure look at the table in slide 27 and you will find beside the apocrine glands there is alpha receptor which is adrenergic). Remember** from the first lecture: the sweat glands are supplied by **postganglionic sympathetic cholinergic neurons**. Even though it's sympathetic, the postganglionic fiber releases **acetylcholine**, and that acetylcholine produces the sweating.

So, to summarize:
Eccrine glands → stimulated in a **parasympathetic-like** manner, where acetylcholine acts on cholinergic receptors.
Apocrine glands → stimulated by the **sympathetic adrenergic system**

Gland type	Innervation type	Postganglionic NT	Receptor	Function
Eccrine	Sympathetic	Acetylcholine (ACh)	Muscarinic	Thermoregulation
Apocrine	Sympathetic	Norepinephrine	Adrenergic	Emotional/stress sweat

Eccrine glands produce the light, watery sweat—like the sweating on the palms, soles of the feet, and sometimes the face.
Apocrine glands are found in areas with **hair**, and they are the ones involved in producing a person's **body odor**. This is because their sweat is mixed with **sebum**, which is a fatty secretion. Sebum contributes to the unpleasant smell, and when bacteria act on this mixture, the odor becomes even stronger
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Metabolic functions:
For metabolism, we focus on **beta-2 receptors** (not alpha receptors here).
Beta-2 receptors stimulate **gluconeogenesis** and **glycogenolysis**.

Beta-3 receptors are responsible for **lipolysis**.
So, the receptors present in **fat cells** function to **induce lipolysis**.

In the **kidney**, **beta-1 receptors** stimulate **renin release**.
Renin acts on angiotensinogen, leading to the formation of **angiotensin II**.
Angiotensin II is a **vasoconstrictor** and also a **stimulator of aldosterone secretion**

Additional Resources:

رسالة من الفريق العلمي:

Reference Used:
(numbered in order as cited in the text)

1. Chat gpt
2. Photos from Google

In your 20s you will be hesitant about med school…….
It’s important you ignore that feeling!!

For any feedback, scan the code or click on it.

Corrections from previous versions:

Versions	Slide # and Place of Error	Before Correction	After Correction
V0 → V1	26	The doctor make a mistake: **Far vision → parasympathetic → contraction** **Nea2r vision → sympathetic → relaxation**	**Near vision →parasympathetic →contraction** **Far vision →sympathetic →relaxation**
V1 → V2	5	· It reduces NE release from sympathetic terminals. · This results in less sympathetic stimulation of the heart, helping prevent excess cardiac rate (tachycardia). Therefore, M1 receptors mediate feedback for the sympathetic NS as hetero-receptors located in the sympathetic NS.	• It increases NE release from sympathetic terminals. • This results in more sympathetic stimulation of the heart
	6	M1 hetero-receptor activated by Ach, less NE release, decreased heart rate	M1 hetero-receptor activated by Ach, increase in the ca+2 inside the neuron , more NE release
	26	Sympathetic	Parasympathetic
	24	People sometimes narrow their eyelids or use their fingers to reduce light and sharpen the image when looking far away—this indirectly helps the parasympathetic system accommodate for distant vision	People sometimes narrow their eyelids or use their fingers to limit the amount of light entering the eye, which reduces peripheral light rays and improves image sharpness for distant objects. This is a mechanical-optical adjustment and does not involve parasympathetic accommodation, which is responsible for near vision rather than distant vision
V2→V3	29		**Some changes happened**