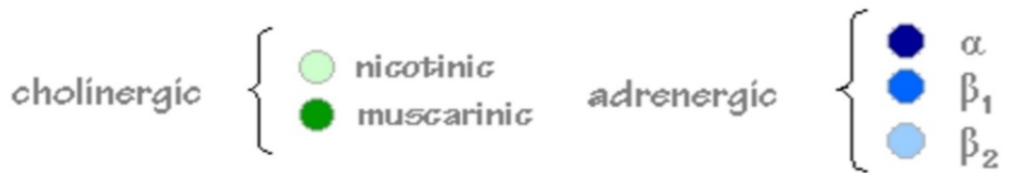
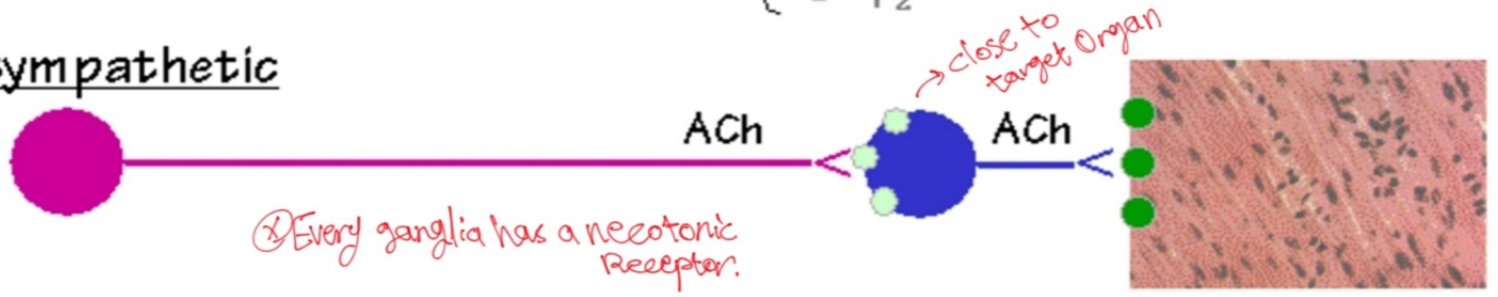




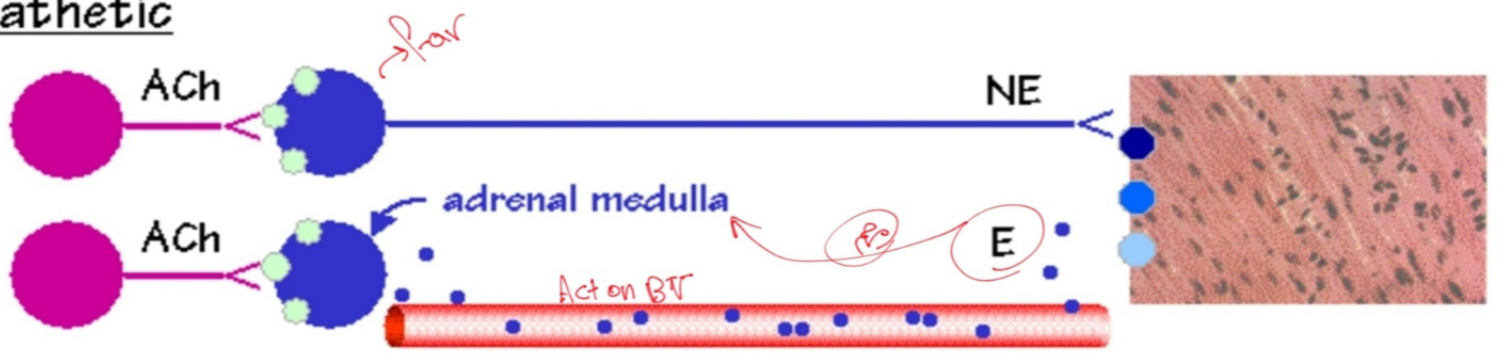
Autonomic Nervous System



Parasympathetic



Sympathetic



Somatic motor



Lecture-5 :Topics

- Functions of ANS
- Effect of Sympathetic & Parasympathetic stimulation
- Overall difference between 2 divisions of ANS

Learning Objectives

- Effects of sympathetic and parasympathetic neurotransmitters on target organs and tissues.

⇒ trigger / stimulates / signal

• Most of the organs are dual innervated (by P, S).

Sympathetic Effects

لبيجيو على الأكتين

- Fight, Fright or flight response
- Release of Neurotransmitters (NT)-
 - Norepinephrine (NT) from postganglionic fibers
 - Epinephrine (NT) from adrenal medulla

↳ Exception

⇒ Main Neurotransmitter in the Sympathetic NS is the Nor-Epinephrine

Sympathetic Effects

- Mass activation prepares for intense activity ⇒ High Maintenance Activity.

➤ Heart rate (HR) increases ⇒ Ex → during Running the HR ↑

➤ Bronchioles dilate ⇒ For Better Breathing during Running

➤ Blood [glucose] increases

↳ Brain is Peeking on it

Sympathetic Effects

↳ Fight + Flight

- GI motility decreases ⇒ During the Fight/Flight we don't need the GI that much! I'm paying attention somewhere else!
- Contraction of sphincters
- Relaxation of
 - Detrusor muscle ⇒ Outward muscle in the urinary bladder.
 - Ciliary muscle
- Mydriasis ⇒ اتسع بؤبؤ العين

↳ Better pumping for the Heart

Parasympathetic Effects

كل واحد مسئول بجاله بالوضع (الطبيعي)

- Normally not activated as a whole
 - Cumulation of separate parasympathetic nerves.
- Release as
- Relaxing effects-
 - Decreases HR. ⇒ Relaxing
 - Dilates visceral blood vessels. Mostly GI motility increases
 - Increases digestive activity. ← So

Parasympathetic Effects

Relaxation Mode be like

- Bronchoconstriction
- GI motility increases
- Relaxation of sphincters
- Contraction of
 - Detrusor muscle
 - Ciliary muscle
- Miosis ⇒ البؤبؤ حاد

Adrenergic and Cholinergic Synaptic Transmission

- **ACh** is **NT** for **parasympathetic fibers**
 - Sympathetic fibers
 - Parasympathetic fibers
- **Transmission at these synapses is termed cholinergic**
- **All sympathetic fibers terminate in ganglia**

ACh is associated w/ cholinergic
 NE, E ⇒ is associated w/ Adrenergic

ACh - مستقبلات

NE/E - مستقبلات

Ganglia ⇒ محطة! مترابطة
 بين Pre- و Post-
 و متواجدة بال P, S

⊗ We call (E, NE) also with Catecholamines

Adrenergic ← their receptor!

Adrenergic and Cholinergic Synaptic Transmission

- [redacted] is NT released by -
 - ([redacted]) [redacted] parasympathetic fibers
 - ([redacted]) [redacted] sympathetic fibers
- Postganglionic autonomic fibers innervate the target tissue / Target Organ

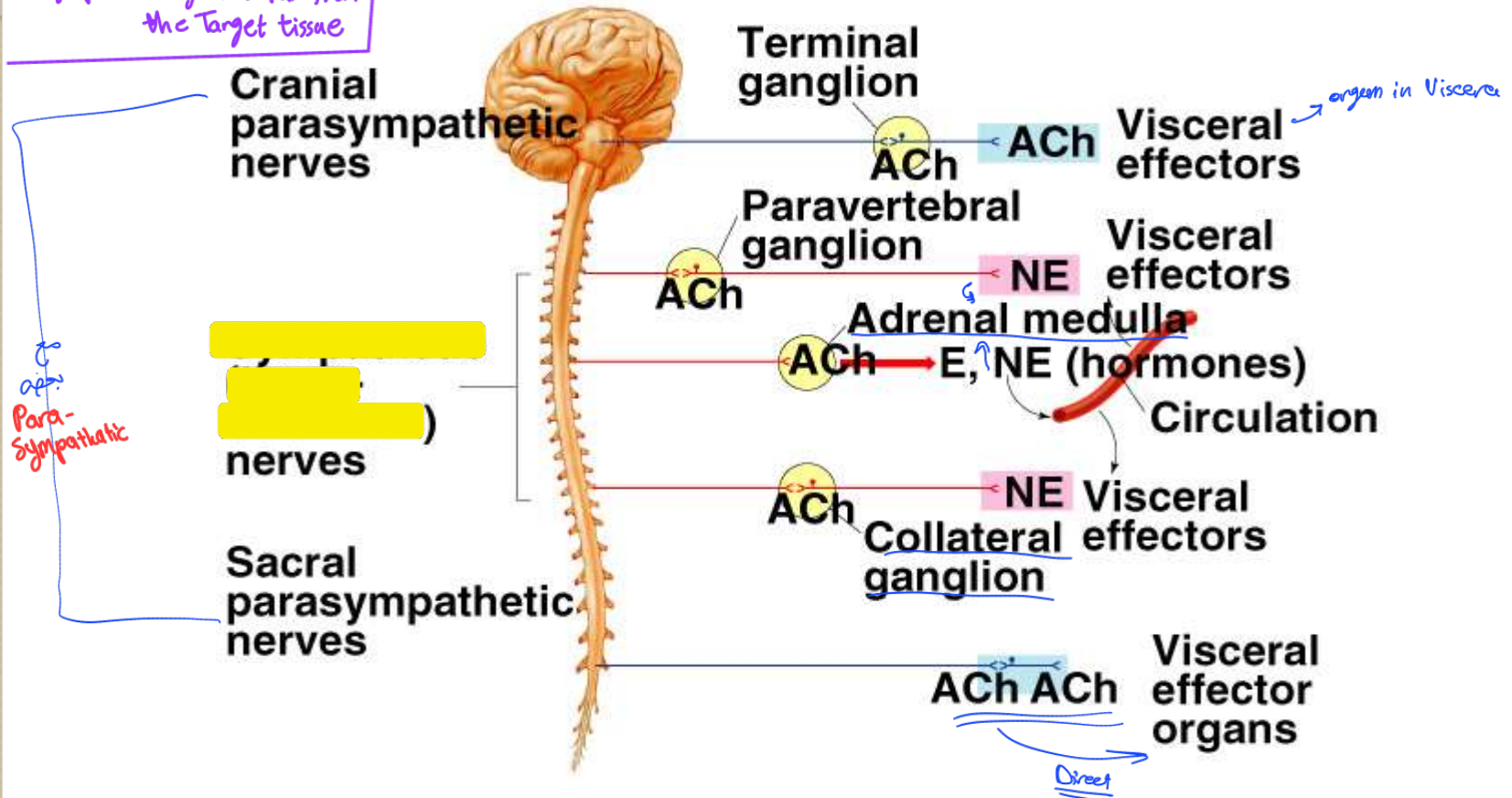
Adrenergic and Cholinergic Synaptic Transmission

*Some organs has dual innervation
From P + S*

Remember

- in Para ⇒ Ganglion is near the target tissue
in Symp ⇒ Ganglion is far from the target tissue

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Adrenergic Synaptic Transmission

(continued)

- Transmission at these synapses is called adrenergic:
 - Norepinephrine
 - ❖ released by most postganglionic sympathetic nerve fibers.
 - Epinephrine,
 - ❖ released by the adrenal medulla.
- Collectively called Catecholamines
 - ⊕ Ach, ⇒ cholinergic

Responses to Adrenergic Stimulation

- Beta adrenergic receptors:
 - Produces their effects by stimulating production of cAMP
 - NE binds to receptor
 - G protein dissociates: α and $\beta+\gamma$

Responses to Adrenergic Stimulation

- Depending upon tissue, either **alpha** or **beta** complex produces the effects
- **Alpha** *از اهاي بيشتره
ف بهر چينه پ*
 - ❖ **Activates** *Production for cAMP*
 - ❖ **Producing** **cAMP**
 - ❖ **cAMP** *Phosphorylation* **activates** **protein kinase**
 - ❖ **Opening** **ion channels** *leads*

Responses to Adrenergic Stimulation

(continued)

(α one)

- **Alpha₁ adrenergic receptors:**

- ▶ Produce their effects by the production of Ca^{2+}

- ▶ Epi binds to receptor

- ▶ Ca^{2+} binds to calmodulin

→ Protein that binds w/ Calcium

- ▶ Calmodulin activates protein kinase, which phosphorylates β receptors

⇒ Response
only by α_1

Responses to Adrenergic Stimulation

(continued)

- **Alpha adrenergic receptors:**

1. Located on Presynaptic terminal

➤ Decreases release of NE.

❖ Negative feedback control. ⇒

لما نزيد عن حده
بروح يظمن حاله

2. Located on postsynaptic membrane

➤ When activated, produces

vasoconstriction

Q

If I have a patient
w/ a hypertension, on what
type of Receptors should the
drug acts on? or what would
be target tissue of the drug?

Responses to Adrenergic Stimulation

(continued)

- Has both **excitatory** and **inhibitory** effects.
- Responses due to different membrane receptor proteins. *⊕ Major Mechanisms ⊖*
 - **α₁** : constricts (visceral) smooth muscles.
 - **α₂** : contraction of smooth muscle. *تقریباً Is بجہت!*
 - **β₁** : increases HR and force of contraction.
 - **β₂** : relaxes bronchial smooth muscles.
 - **β₃** : adipose tissue, function unknown




↳ In asthma ⇒ we give β₂ specific drugs.

Responses to Cholinergic Stimulation

Adrenergic → NE, EPIN

- Cholinergic fibers-
 - Release **[redacted]** as NT
 - ❖ All somatic motor neurons ^{→ Voluntary}
 - ❖ All preganglionic neurons
 - ❖ Most postganglionic parasympathetic neurons
 - ❖ Some postganglionic sympathetic neurons

Responses to Cholinergic Stimulation (continued)

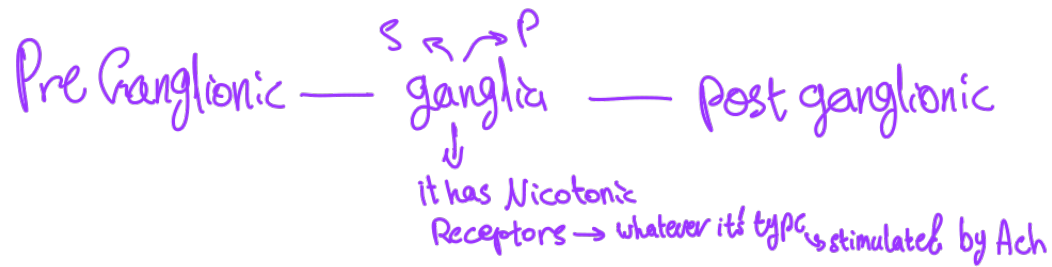
- Somatic motor neurons  • Excitatory
- All preganglionic autonomic neurons  • Excitatory
- Postganglionic axons  • Excitatory or
• Inhibitory

Responses to Cholinergic Stimulation (continued)

- **Muscarinic receptors**
 - ACh binds to receptor
 - Requires the mediation of G proteins
 - β_1 complex affects-
 - ❖ Opening a channel or
 - ❖ Closing a channel or
 - ❖ Activating enzymes

Responses to Cholinergic Stimulation (continued)

- Nicotinic receptors (ligand-gated)
 - ACh binds to 2 nicotinic receptor binding sites.
 - Causes ion channel to open within the receptor protein.
 - ❖ Opens a Na⁺ channel.
- Always excitatory ⇒ For muscle contraction

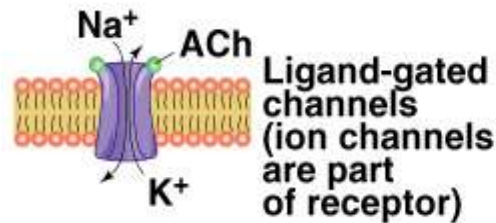


Responses to Cholinergic Stimulation (continued)

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Nicotinic ACh receptors

- Postsynaptic membrane of
- All autonomic ganglia
 - All neuromuscular junctions
 - Some CNS pathways



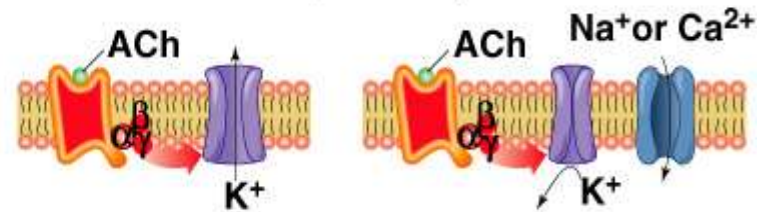
Depolarization

Excitation

*Action Potential
caused by
movement
of charged
particles
inside.*

Muscarinic ACh receptors

- Produces parasympathetic nerve effects in the heart, smooth muscles, and glands
- G-protein-coupled receptors (receptors influence ion channels by means of G-proteins)



Hyperpolarization

(K⁺ channels opened)

Inhibition

Produces slower heart rate

Depolarization

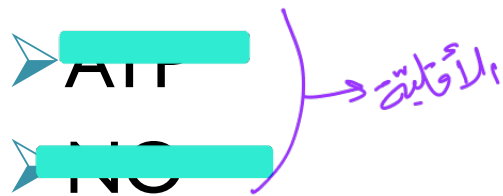
(K⁺ channels closed)

Excitation

Causes smooth muscles of the digestive tract to contract

Other Autonomic NTs

- Certain nonadrenergic, noncholinergic postganglionic autonomic axons produce their effects through other NTs



Organs With Dual Innervations

- **Dual innervations**
 - Innervations by both
 - ❖ **Sympathetic**
 - ❖ **Parasympathetic**
- **Most visceral organs receive dual innervations**
- **Effects of dual innervations**
 - **Antagonistic** ⇒ عاكس / يعكس
 - **Complementary** ⇒ متكاملين / ليعملوا
 - **Cooperative** ⇒ متعاونين

Organs With Dual Innervations

- Antagonistic :
 - Sympathetic and parasympathetic fibers innervate the same cells.
 - Actions counteract each other.
 - Heart rate.
- Complementary:
 - Sympathetic and parasympathetic stimulation produces similar effects.
 - Salivary gland secretion. *No Inhibition*
- Cooperative:
 - Sympathetic and parasympathetic stimulation produce different effects that work together to produce desired effect.
 - Micturition.

التبول

*التعاون بينه
الجهازين في تنظيم
عضلات sphincter والآن
Muscles*

Organs Without Dual Innervations

- Regulation achieved by **increasing** or **decreasing** firing rate.

Q

What are the organs that receive **only sympathetic** innervations-?

➤ **Adrenal medulla** / *Suprarenal*

➤ **Arrector pili muscle** *صواعيق*

➤ **Sweat glands**

➤ **Most blood vessels.**

All the Following Except?

Applied

Horner's syndrome

- Characterized by- *Symptoms?*
 - Constriction of the pupil
 - Enophthalmos ⇒ العين داخلة لجوا
Exo ⇒ لبرا
 - Drooping of eye lid ⇒ سايح
 - Anhidrosis on affected side of face ⇒ No sweat
- Occurs due to- *Huge Nerves Present in the Neck*
 - Damage of stellate ganglia
 - Paralysis of Cervical Sympathetic nerve trunk

Horner's syndrome



Figure 1: Left pupillary miosis, marked hypochromia of the left iris, ipsilateral mild ptosis and left hemifacial anhidrosis

Drugs acting on autonomic ganglia

Increases activity

- Direct effect
 - Acetylcholine
 - Nicotine (Low doses)
- Indirect effect
(ACE inhibitors) *→ chE*
 - Physostigmine
 - Neostigmine
 - Parathion
 - DFP

Decreases activity

- Ganglion blockers-
 - Hexamethonium
 - Mecamylamine
 - Pentolinum
 - Trimethaphan

Drugs acting on Postganglionic sympathetic nerve endings

Increases activity

↑ Release NE (TEA) ^{سای}

- Tyramine
- Ephedrine
- Amphetamine

in vivo
CNS stimulant

Decreases activity

- ❖ Block NE Synthesis
 - Metyrosine
- ❖ Block Storage
 - Reserpine
 - Guanethidine
- ❖ Prevent Release
 - Bretylium
- ❖ False transmitters
- ❖ Methyldopa

Drugs acting on Muscarinic receptors

Increases activity

- Acetylcholine

Decreases activity

- Atropine ⇒ *Parasympathetic*
- scopolamine

Drugs acting on Beta adrenergic receptor

Increases activity

- **β stimulators**
 - Isoproterenol
- **β_2 stimulators**
 - Salbutamol
 - Terbutaline

Decreases activity

- **β blockers**
 - Propranolol *⇒ Regulates HR*
 - Metoprolol
- **β_1 blockers**
 - Atenolol
- **β_2 blockers**
 - Butoxamine

Drugs acting on Alpha adrenergic receptors

Increases activity

(α_1 stimulators)

- Methoxamine
- Phenylephrine

Decreases activity

(α blockers)

- Phenoxybenzamine
- Phentolamine
- Prazocin (α_1 blockers)
- Yohimbine (α_2 blockers)



**Thank
You!!!**

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