النادي الطبي

Pharmacology

Done by :Johainah Taha....

lecture no.!2...

Drugs affecting adrenergic and cholinergic system

Drugs affecting adrenergic and cholinergic system:

1. **Cholinergic agonists.**
2. **Cholinergic antagonists.**
3. **Adrenergic agonists.**
4. **Adrenergic antagonists.**

* Cholinergic drugs: drugs act upon the neurotransmitter acetylcholine, the primary neurotransmitter within the parasympathetic nervous system.

Cholinergic agonists

depending on the mechanism of action

Directly acting

Indirectly acting

1. **Directly acting cholinergic agonists** also known as parasympathomimetics.

Mimics the effects of Ach by binding directly to cholinoceptors (muscarinic or nicotinic).

2. **Indirectly acting cholinergic agonists:**

Cholinesterase inhibitors.

إنزيم موجود بال Cleft حكينا عنه المحافزة الماضية.

ولما أعمل الـ inhibition أنا بخلي الـ Ach تبع الـ action يدوم لفترة أطول.

Directly acting cholinergic agonists

الأفضل حفظ

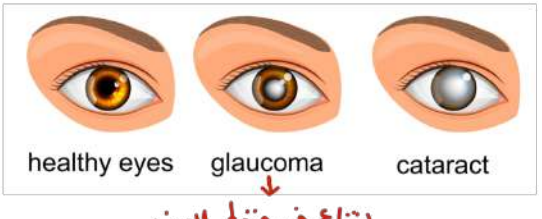
- It mimics the effects of acetylcholine by **binding directly to cholinoceptors**. [muscarinic]
- These agents may be broadly classified into **two groups:**
- **1-choline esters,** which include acetylcholine and synthetic esters of choline, such as carbachol and bethanechol
  ↳ longer duration
- **2-Naturally occurring alkaloids,** such as pilocarpine
- **All direct-acting cholinergic drugs have longer durations of action than acetylcholine.**
- Direct-acting agonists **show little specificity in their actions**, which limits their clinical usefulness.
- **Muscarinic agents** preferentially bind to muscarinic receptors. e.g. pilocarpine, bethanechol. They are more therapeutically useful.

* many of these drugs effect on both muscarinic and nicotinic receptors, ex: acetylcholine.

* Whereas a few of them are highly selective for muscarinic or nicotinic receptors.

Directly acting cholinergic agonists

1. **Acetylcholine** ← specificity ماعندهم
2. **Bethanechol (synthetic ester):** we use it as a treatment of bladder and GI hypotonia. **increase intestinal motility and tone.** It stimulates the detrusor muscles of the bladder while the trigone and sphincter are relaxed, causing the **expulsion of urine**
3. **Carbachol (synthetic ester):** in the eye as a **miotic agent** to treat glaucoma ↳ local effect.
4. **Pilocarpine (Natural alkaloid): miosis and contraction of the ciliary muscle, used to treat glaucoma, potent stimulator of secretions** such as sweat, tears, and saliva. ↓ Side effect because it is more specific.
5. Atropine.



ارتفاع في ضغط العين

* Bethanechol and pilocarpine acts on muscirinic receptors.

Acetylcholine

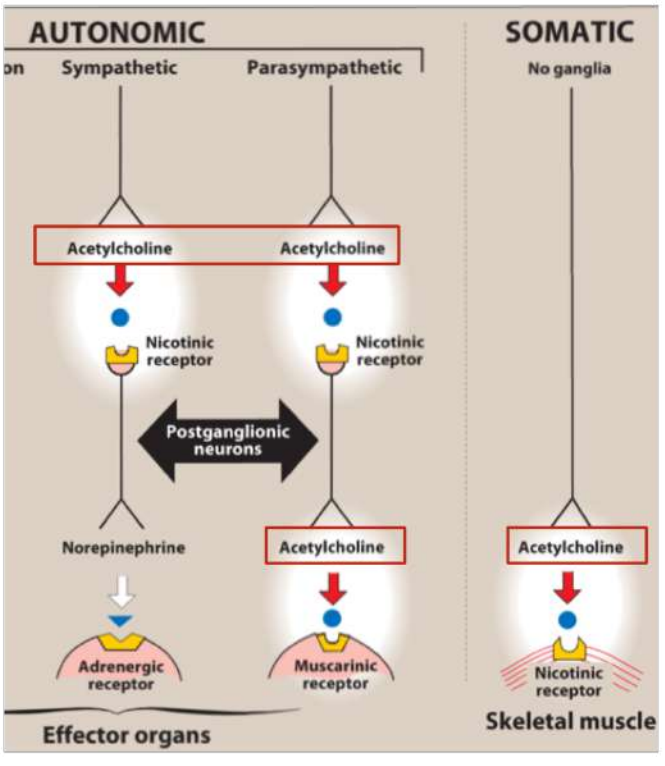
- Acetylcholine is a quaternary ammonium compound that cannot penetrate membranes. → Hydrophilic
- It is the neurotransmitter of parasympathetic[1] and somatic[2] nerves as well as autonomic[3] ganglia.
- Therapeutically, of no importance because of its multiplicity of actions and its rapid inactivation by cholinesterase.
- Acetylcholine has both* muscarinic and nicotinic activity → no speceficity
- Its actions include:

1. Decrease in heart rate and cardiac output: mimics vagal stimulation (Contractility — HR بتقل عمل Vegus nerve بموضع Ach)
2. Decrease in blood pressure → Soit could cause Ulcer
3. GI: increase salivary and gastric acid secretions and stimulate intestinal secretions and motility. +↓ Sphicter tone
4. Bronchoconstriction ← Ach لـ inhibition بنحتاج asthma لمريض بحالة الـ
5. Induce urination
6. Eyes: ciliary muscle contraction and constriction of the pupils (miosis) → Could cause glaucoma.

؟ accommodation و miosis شو الفرق بين

contraction of smooth muscles of the iris sphincter ← Miosis

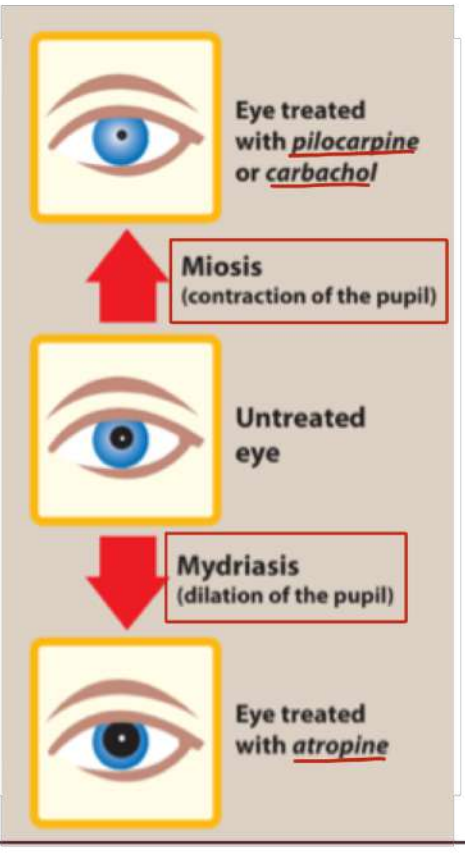
contraction in ciliary muscles ← Accommodation



- All ANF leaving CNS release Ach which acts on nicotinic receptors.

- All postgangilionic parasympathetic fibers release Ach which acts on muscarinic

Actions of Pilocarpine and carbachol and atropine on the iris and ciliary muscle



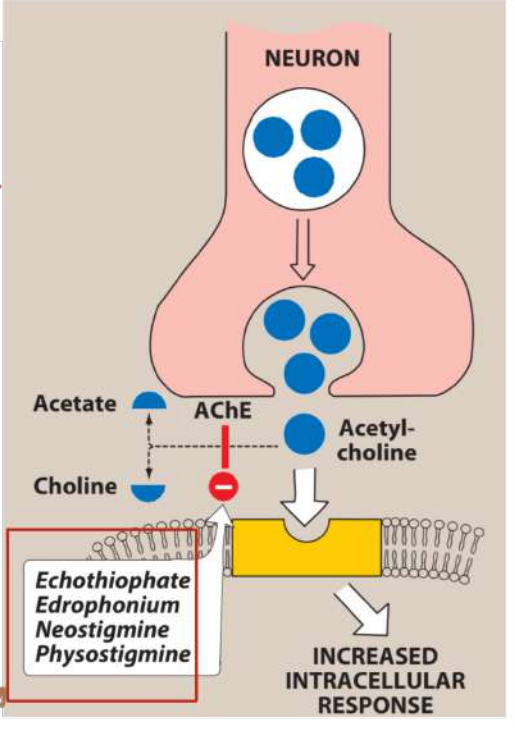
Indirect-acting cholinergic agonists: anticholinesterases(AChE inhibitors)

- Indirect-acting cholinergic agonists: anticholinesterases (reversible)
  بالعلاج بنلجأ لـ reversible
- Indirect-acting cholinergic agonists: anticholinesterases (irreversible).
- Toxicology of anticholinesterase.

هاي الرسمة بتوضح دور Ach E inhibitors.

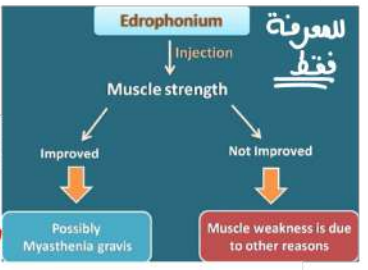
Acetylcholine —AchE→ Acetate + Choline

- الأدوية بتوقف عمله فنزيد تراكم Ach
- تأثير هذه الأدوية temporary.
- مثال على الأدوية



Indirect-acting cholinergic agonists: anticholinesterases

- **Acetylcholinesterase (AChE)** is an enzyme that specifically cleaves acetylcholine to acetate and choline and, thus, terminates its actions.
- It is located both pre- and postsynaptically in the nerve terminal, where it is membrane bound.
- Inhibitors of acetylcholinesterase **indirectly provide a cholinergic action by prolonging the lifetime of acetylcholine** produced endogenously at the cholinergic nerve endings. This results in the accumulation of acetylcholine in the synaptic space.
- These drugs can thus provoke (stimulate) a response at all cholinoceptors in the body, **including both muscarinic and nicotinic** receptors of the **autonomic nervous system,** as well as at neuromuscular junctions **(NMJ)**and in the brain.



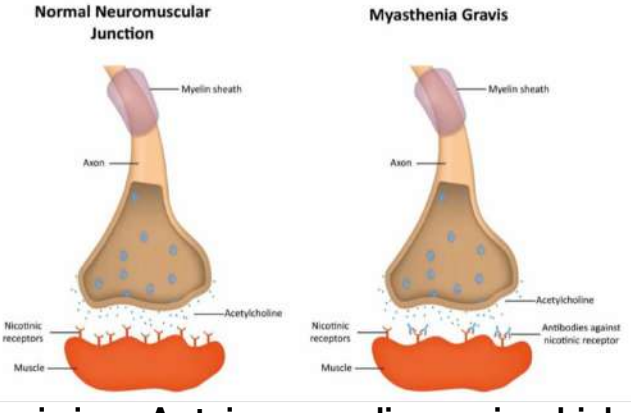
Indirect-acting cholinergic agonists: anticholinesterases (reversible)

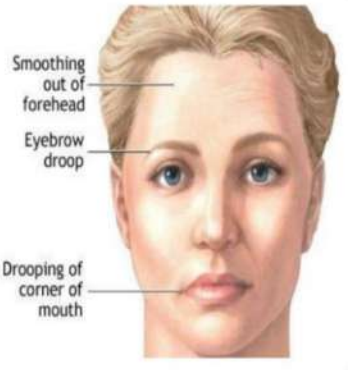
A. Edrophonium: *Used for* ***diagnosis*** **of Myasthenia gravis**. 2-Reversing the effect of nonpolarizing neuromuscular blockers(NMBs) after surgery. ***Atropine*** is the antidote. (distinguish) (بشتغل هنه) [Short acting → use it for diagnosis]

B. Physostigmine: treatment of **overdoses of drugs with anticholinergic actions**, such as *atropine, ant to reverse the effect of NMBs.*

1. Neostigmine: Used to stimulate the bladder and GI tract, antidote neuromuscular blocking agents, and symptomatic treatment of myasthenia gravis (لا يعطى للمريض بشكل يومي إلا حسب symptoms .)

A. Pyridostigmine: Chronic management of Myasthenia gravis





SYMPTOMS:

- The first noticeable symptom is weakness of the **eye muscles,** difficulty in swallowing and slurred speech may also be the first signs.
- Muscles that control eye and eyelid movement, facial expressions, **chewing, talking and swallowing** becomes weaker.
- The muscles that control **breathing** and neck and limb movements can also be affected.

Myasthenia is anAutoimmune disease in which antibodies complex with nicotinic receptors at the neuromuscular junction to cause skeletal muscle weakness and fatigue.

Indirect-acting cholinergic agonists: anticholinesterases (irreversible)

مواد دوية أكيد إغاشغلات سامة بتعرض الها

↳ organic derivatives of phosphoric acid.

- It is a **synthetic organophosphate** compounds have the capacity to bind covalently to AChE.
- The result is a long-lasting increase in acetylcholine at all sites where it is released.
- Many of these drugs are extremely toxic and were developed by the military **as nerve agents.**

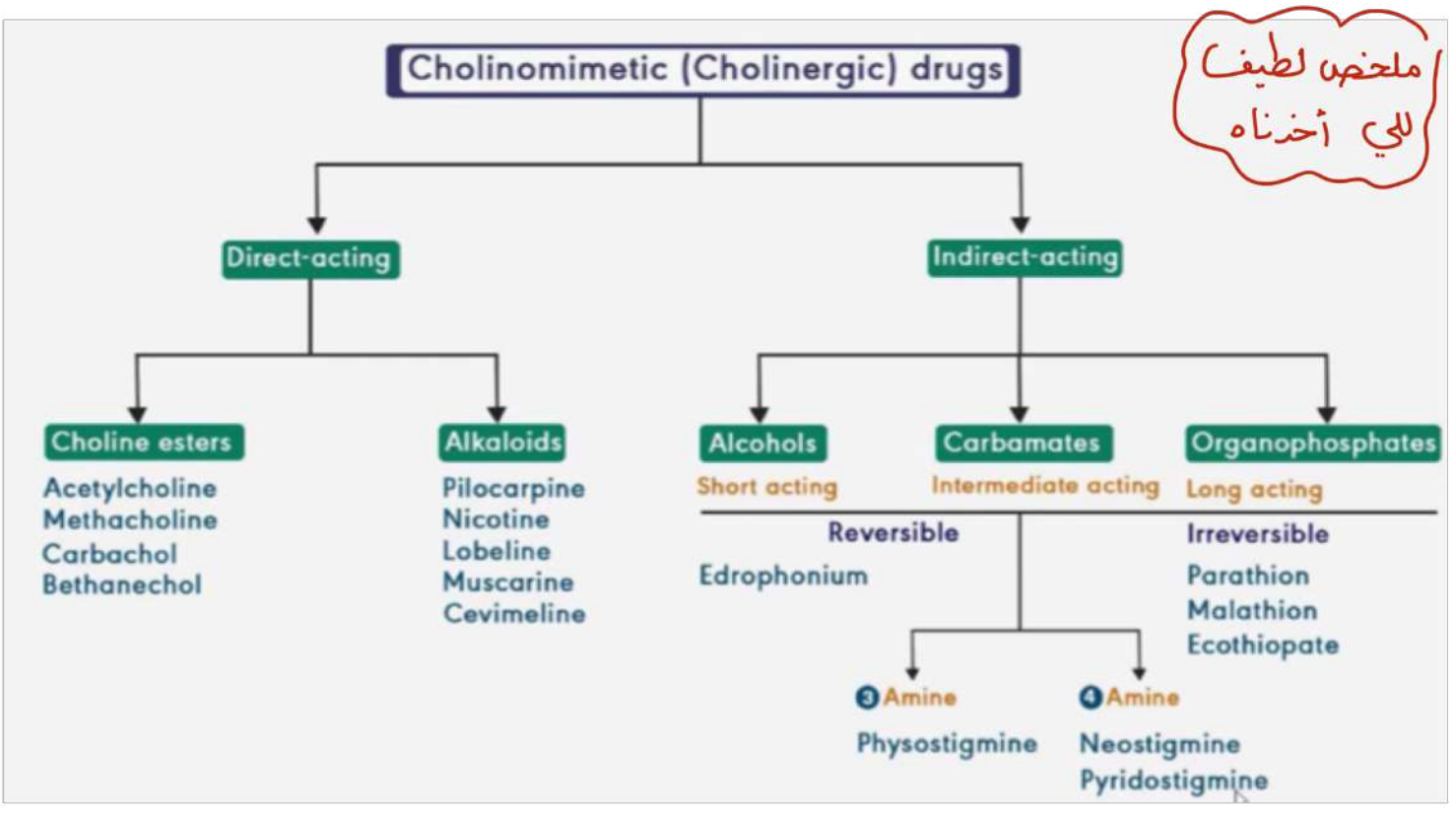
للمعرفة

They are quite lipid-soluble and well absorbed from the skin, lung, gut, and conjunctiva thereby making them dangerous to humans and highly effective as insecticides.
+ they penerate the blood-brain barrier freely

Toxicology of anticholinesterase

- Irreversible AChE inhibitors are commonly used **agricultural** زراعي **insecticide,** مبيد حشري which has led to numerous cases of accidental poisoning of these agents.
- Frequently used for **suicidal** انتحار **and homicidal** قتل **purposes**.
- Toxicity with irreversible AChE inhibitors leads to cholinergic crises (nicotinic and muscarinic signs and symptoms). ← all the body

ملخص لطيف للي أخذناه



Cholinergic Antagonist

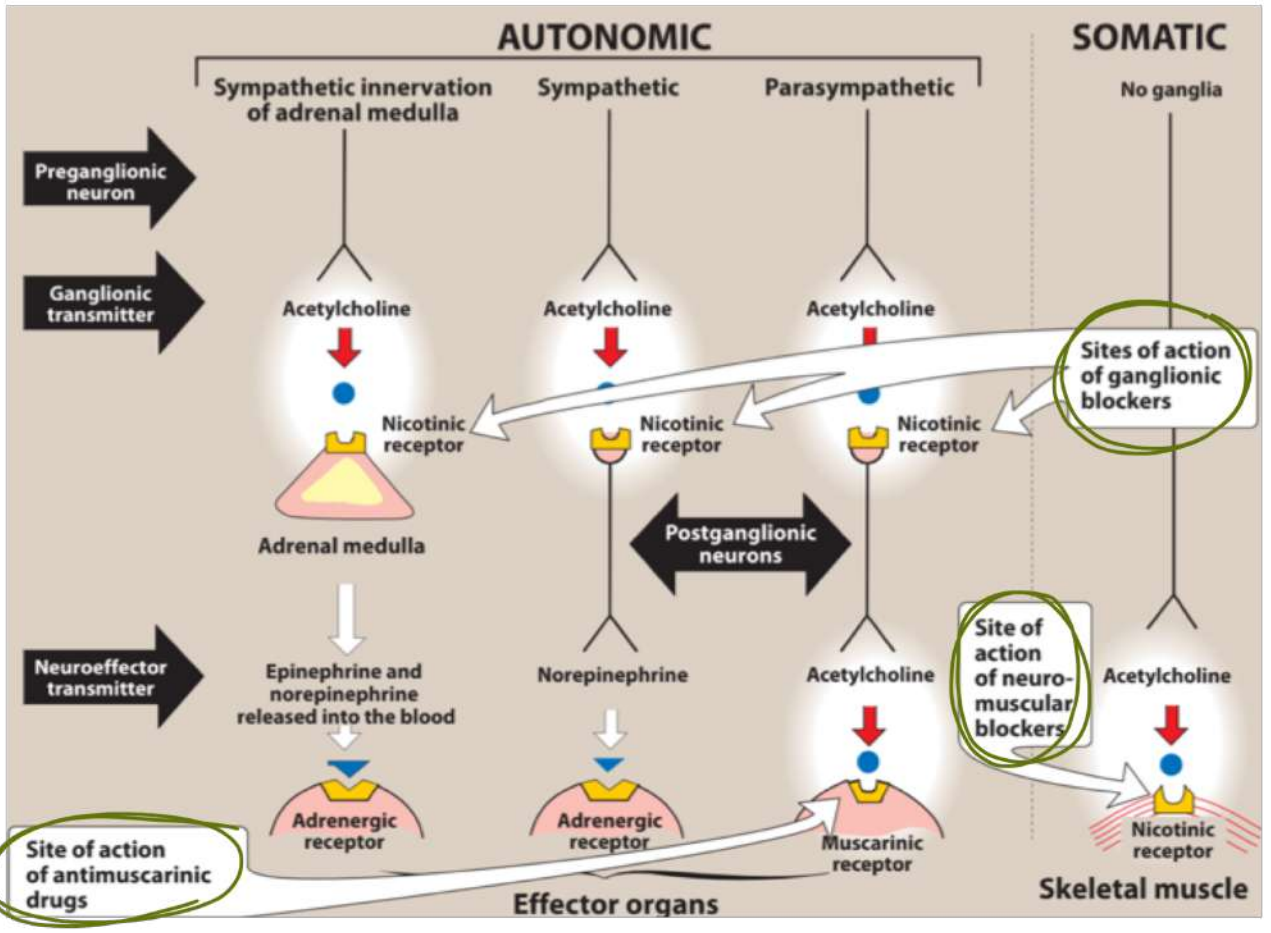
Based on their action

- Antimuscarinic agents
- Gangelionic blockers
- Neuromuscular blockers

Cholinergic antagonists

- They are agents **that bind to cholinoceptors** (muscarinic and nicotinic) and **prevent the effect of acetylcholine** and other cholinergic agonists.
- Cholinergic antagonists are classified into three basic classes based on their sites of action:

1. Antimuscarinic agents → action on muscarinic receptors.
2. Ganglionic blockers → action on nitric receptors near gangalia
3. Neuromuscular blockers → action on neuromuscular receptors.



Cholinergic antagonists

1. **Antimuscarinic agents:** selective muscarinic receptor blockers. The effects of parasympathetic innervation are thus disrupted, leaving sympathetic stimulation unopposed. → There is no parasympathetic effect.
2. **Ganglionic blockers:** specifically act on the nicotinic receptors of both parasympathetic and sympathetic autonomic ganglia (serve as tools **in experimental pharmacology)**.They are the least important anticholinergic drugs in terms of clinical efficacy. → There is no sympathetic + parasympathetic effect
3. **Neuromuscular blocking agents**, prevent efferent impulses from reaching the skeletal muscles. These agents are used as **anesthetic adjuvants during surgery**. ← لتصير عملية الفتح أسرع وأسهل.

↳ to relax for muscle

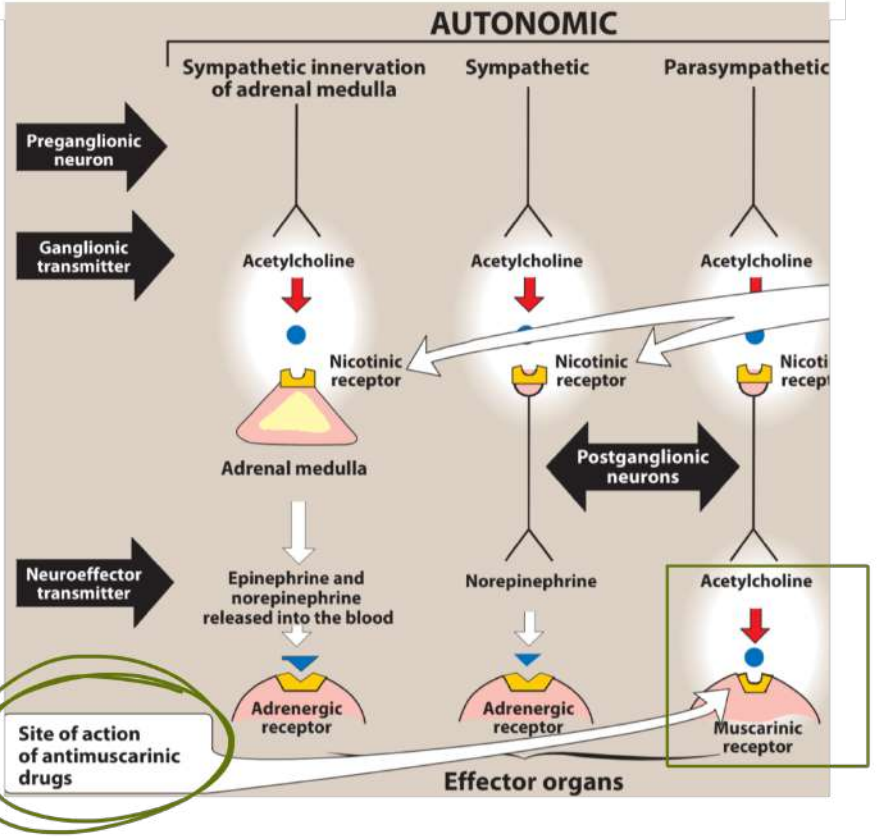
Antimuscarinic Agents

- They are commonly known as **anticholinergic drugs (a misnomer as they only block muscarinic receptors).**
- ***** These drugs also block the few exceptional sympathetic neurons that are cholinergic, such as those innervating salivary and sweat glands.

← ماعدا استثناء → sympathetic innervation of Sweet glands → Ach → muscarinic receptors. بدل nicotinic

- The cholinergic blockers are beneficial in a variety of clinical situations.

- Antimuscarinic drugs have little or no effect on skeletal neuromuscular junctions or autonomic ganglia because they do not block nicotinic receptors.



Antimuscarinic Agents:

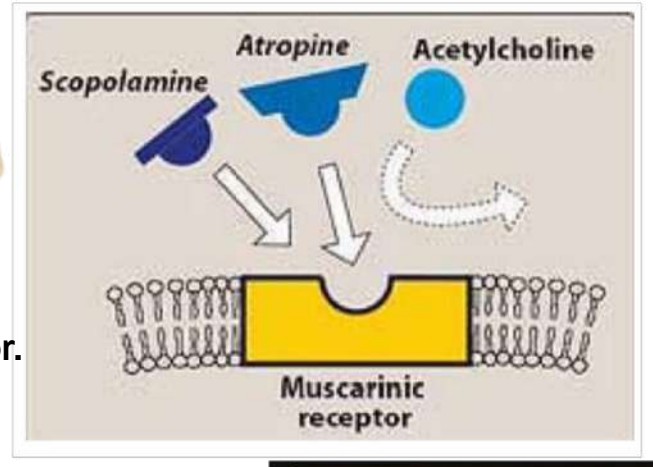
A. Atropine.

competitive inhibitors

B. Scopolamine.

***Competition of atropine and scopolamine with acetylcholine for the muscarinic receptor.**

مبدأ عملها



https://youtu.be/kOT2sT3_RTg

Atropine → it blocks the action of parasympathetic

Actions:

I. **Eye:** *Atropine* blocks all cholinergic activity on the eye, resulting in persistent ① **mydriasis** (dilation of the pupil), ② **unresponsiveness to light,** and ③ **cycloplegia** (inability to focus for near vision).

II. **Gastrointestinal (GI):** Atropine can be used as an antispasmodic to reduce activity of the GI tract.

لمضاد للتشنجات اي بتهدئ الأمعاء

III. **Cardiovascular:** Atropine produces divergent effects on the cardiovascular system, **depending on the dose:**

- **At low doses**, the predominant effect is a decreased cardiac rate (**bradycardia**), the effect results from blockade of the **M1** receptors on the inhibitory prejunctional (or presynaptic) neurons, thus permitting increased acetylcholine release.
- **Higher doses** of atropine cause a **progressive increase in heart rate** by blocking the **M2** receptors on the sinoatrial node.

IV. **Secretions:** Atropine **blocks the salivary glands**, producing a drying effect on the oral mucous membranes (xerostomia).

- **Sweat and lacrimal glands** are also affected. [Note: **Inhibition of secretions by sweat glands can cause elevated body temperature**, which can be dangerous in children and elderly.

تعرق ← دموع ← رفع

Atropine

- **Therapeutic uses:**

1. **Ophthalmic:** Topical atropine **exerts both mydriatic and cycloplegic effects.** ⇐ dilation in pupil ⇐ Paralysis of ciliary muscle of the eye.
2. **Antispasmodic:** Atropine is used as an antispasmodic agent to relax the GI tract
3. **Cardiovascular:** to treat bradycardia. = induce tachycardia.
4. **Antisecretory:** block secretions in the upper and lower respiratory tracts prior to surgery.
5. **Antidote for cholinergic agonists:** *Atropine* is used for the treatment of organophosphate (insecticides or nerve gas poisoning) overdoses of acetylcholinesterase inhibitors, such as *physostigmine* and some types of mushroom poisoning (certain mushrooms contain cholinergic substances that block cholinesterase).

← الي حكينا toxic غير

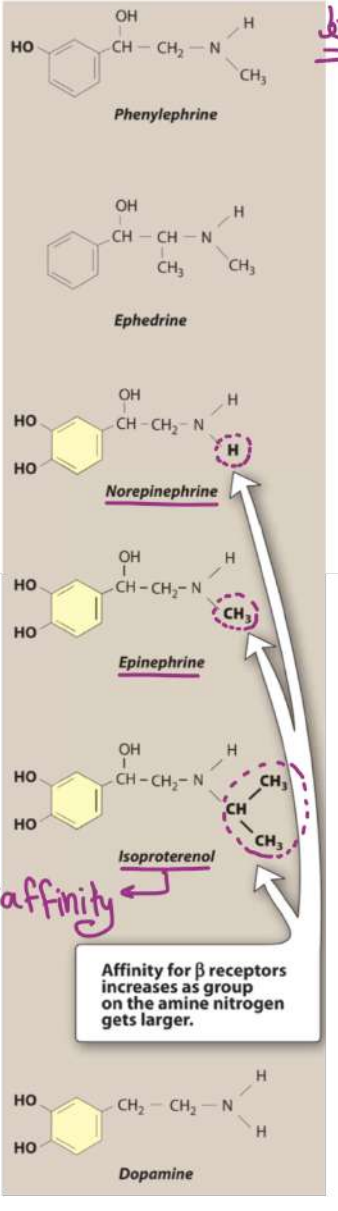
Adrenergic Agonists

Characteristics of Adrenergic Agonists

Most of the adrenergic drugs **are derivatives of β-phenylethylamine**. Substitutions on the benzene ring or on the ethylamine side chains produce a great variety of compounds with **varying abilities to differentiate between α and β receptors and to penetrate the CNS.**

Two important structural features of these drugs are

1. The number and location **of OH substitutions on the benzene ring.**
2. The nature of the substituent on the amino nitrogen.



Mechanism of action of the adrenergic agonists

1. **Direct-acting agonists:** These drugs act directly on α or β receptors, producing effects similar to those that occur following stimulation of sympathetic nerves or release of the hormone epinephrine from the adrenal medulla. not parasympathetic.

2. **Indirect-acting agonists:**

A)Block the uptake of norepinephrine (uptake blockers) e.g., **Cocaine.**

B) Are taken up into the presynaptic neuron and cause the release of norepinephrine from the cytoplasmic pools or vesicles of the adrenergic neuron. As with neuronal stimulation, the norepinephrine then traverses the synapse and binds to the α ± or β receptors. e.g., **amphetamine**.

3. **Mixed-action agonists:** Some agonists, such as **ephedrine, pseudoephedrine and metaraminol,** have the capacity to stimulate adrenoceptors directly and to release norepinephrine from the adrenergic neuron.

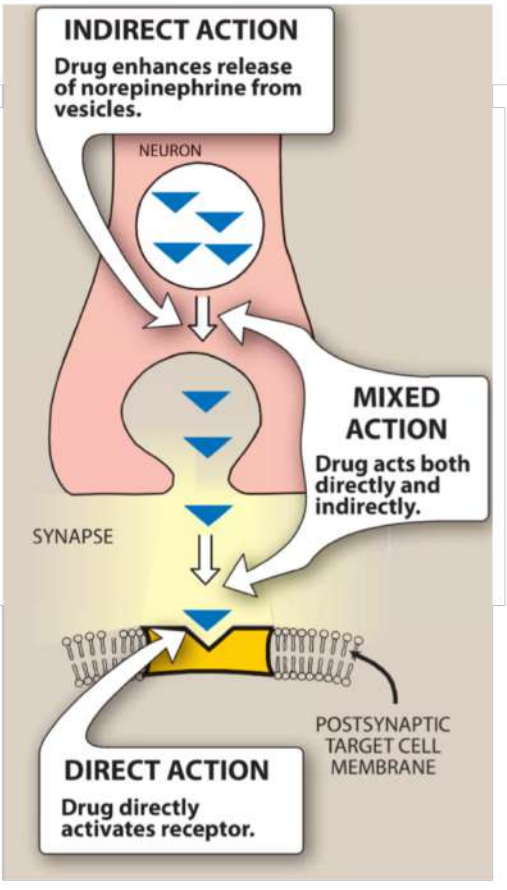
Direct-Acting Adrenergic Agonists

- Direct-acting agonists **bind to adrenergic receptors without interacting with the presynaptic neuron.**
  يعني مو α2 .
- The activated receptor initiates synthesis of **second messengers and subsequent intracellular signals.**

* All α, β receptors are G-protein.

- They are **widely used clinically**.

- **Epinephrine**
- Norepinephrine
- Isoproterenol
- Dopamine
- Dobutamine
- Oxymetazoline
- Phenylephrine
- Albuterol, metaproterenol, and terbutaline
- Salmeterol and formoterol.



توقعوا سؤالين ثلاث عراي الصفحة
المحسر: هب مني
بس مهمـــة جدًا وانته

Epinephrine

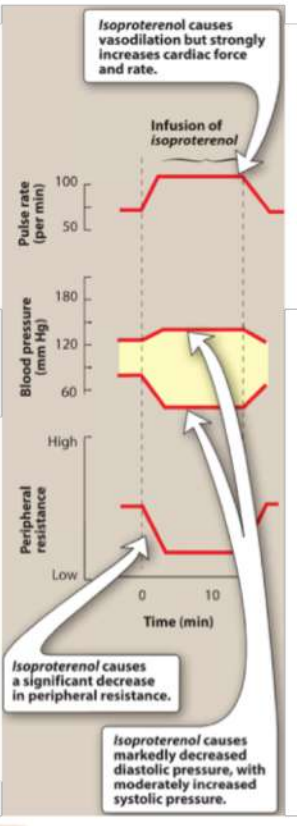
catecholamine
non-catecholamine
} أحنا بنقسم الـ direct الى قسمين

- Epinephrine is one of four **catecholamines**: epinephrine, norepinephrine, dopamine, and dobutamine commonly used in therapy.
- Epinephrine is synthesized from **tyrosine in the adrenal medulla** and released, along with small quantities of norepinephrine, into the bloodstream. Adrenal medulla → Blood Stream.
- Epinephrine interacts with both α and β receptors. **At low doses, β effects (vasodilation)** on the vascular system predominate, whereas at **high doses, α effects (vasoconstriction) are strongest.**

1. **Cardiovascular:**

Epinephrine strengthens the contractility of the myocardium (positive inotropic: β1 action) and increases its rate of contraction (positive chronotropic: β1 action). Cardiac output therefore increases.

→ increase heart rate
→ it makes stronger contraction



With these effects **comes increased oxygen demands on the myocardium**. Epinephrine **constricts arterioles in the skin, mucous membranes, and viscera (α effects)**, and it **dilates vessels going to the liver and skeletal muscle (β2 effects)**. Therefore, the cumulative effect is **an increase in systolic blood pressure, coupled with a slight decrease in diastolic pressure**

120 → systolic Blood Pressure.
80 → diastolic Blood Pressure.

2. **Respiratory:** Epinephrine causes powerful bronchodilation by acting directly on bronchial smooth muscle (β2 action). This action relieves all known allergic- or histamine-induced bronchoconstriction.

↓BP, airway narrow, block breathing.

- In the case of **anaphylactic shock**, this can be lifesaving. In individuals suffering from an acute asthmatic attack, epinephrine rapidly relieves the dyspnea and increases the tidal volume (volume of gases inspired and expired). **Epinephrine also inhibits the release of allergy mediators such as histamines from mast cells.**

Shortness in breathing

3. **Blood sugar :** Epinephrine has a significant **hyperglycemic** effect because of increased glycogenolysis in the liver (β2 effect), increased release of glucagon (β2 effect), and a decreased release of insulin (α2 effect).

4. **Lipolysis:** Epinephrine initiates lipolysis through **its agonist activity on the β receptors of adipose tissue**.

نصيحة : ريتوا هاد الموضوع بجدول أو mind map.
بايديكم طبعًا موجاهن

Epinephrine therapeutic uses

1. **Bronchospasm:** Epinephrine is the primary drug used in the emergency treatment of any condition of the respiratory tract when bronchoconstriction has resulted in diminished respiratory exchange. Thus, in treatment of **acute asthma and anaphylactic shock**, epinephrine is the drug of choice; within a few minutes after subcutaneous administration, greatly improved respiratory exchange is observed.
2. **Anaphylactic shock:** *Epinephrine* is the drug of choice for the treatment of **Type I hypersensitivity reactions** in response to allergens.
3. **Cardiac arrest:** *Epinephrine* may be used to restore cardiac rhythm in patients with cardiac arrest regardless of the cause.
4. **Anesthetics:** Local anesthetic solutions usually contain 1:100,000 parts epinephrine. The effect of the drug is to **greatly increase the duration of the local anesthesia.** It does this by producing vasoconstriction at the site of injection, thereby allowing the local anesthetic to persist at the injection site before being absorbed into the circulation and metabolized.

   ↱ loss of sensation

   التخدير
5. **Intraocular surgery**: for induction and maintenance of Mydriases.

   ↳ dilated + large pupil

Adrenergic antagonists

- The adrenergic **antagonists (also called blockers or sympatholytic agents)** bind to adrenoceptors but do not trigger the usual receptor-mediated intracellular effects.
- These drugs act **by either** reversibly or irreversibly attaching to the receptor, thus preventing its activation by endogenous catecholamines.
- Like the agonists, the adrenergic antagonists are classified according to their **relative affinities for α and β receptors in the peripheral nervous system.**

α-Adrenergic Blocking Agents

- Drugs that block α-adrenoceptors profoundly affect blood pressure.

- Because normal sympathetic control of the vasculature occurs in large part through agonist actions on **α1-adrenergic receptors**, blockade of these receptors reduces the sympathetic tone of the blood vessels, resulting in **decreased peripheral vascular resistance**.

- This induces a reflex tachycardia **resulting from the lowered blood pressure.**

Examples:

- **Phenoxybenzamine.** فينو؟
- **Phentolamine.** فين؟
- **Prazosin, terazosin, doxazosin**  سن

B-Adrenergic Blocking Agents

- All the clinically available β-blockers are **competitive antagonists.** Nonselective β-blockers act at both β1 and β2 receptors, whereas cardioselective β antagonists primarily block β1 receptors.
- There are no clinically useful β2 antagonists
- These drugs also differ in intrinsic sympathomimetic activity, in CNS effects, and in pharmacokinetics.
- Although all β-blockers lower blood pressure **in hypertension, they do not induce postural hypotension, because the α -adrenoceptors remain functional.** Therefore, normal sympathetic control of the vasculature is maintained.

- β-Blockers are also effective **in treating angina, cardiac arrhythmias, myocardial infarction, congestive heart failure, hyperthyroidism, and glaucoma.**
- Can be used as prophylaxis of **migraine headaches.**
- The names of all β-blockers end in **olol except for labetalol and carvedilol.**

- Nonselective β- antagonists: propranolol
- Selective β1- antagonists: Atenolol
- Partial β- agonists: Pindolol
- Antagonists of both α and β-adrenoreceptors: Carvedilol

+ Labetalol

Propranolol

- Propranolol is the prototype β -adrenergic antagonist and blocks both β1 and β2 receptors.

1. **Propranolol diminishes cardiac output, having both negative inotropic and chronotropic** ; these effects are useful in the treatment of angina.

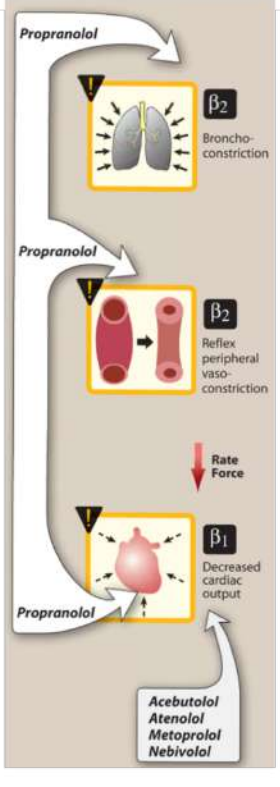
The β -blockers are effective in **attenuating supraventricular cardiac arrhythmias.**

2. **Peripheral vasoconstriction:** Blockade of β receptors prevents β 2-mediated vasodilation. **No postural hypotension occurs, because the α1-adrenergic receptors that control vascular resistance are unaffected.**

2. **Bronchoconstriction:** Blocking β 2 receptors in the lungs of susceptible patients causes contraction of the bronchiolar smooth muscle (This can precipitate a respiratory crisis in patients with chronic obstructive pulmonary disease (COPD) or asthma.

- **β -Blockers, and in particular nonselective ones, are thus contraindicated in patients with COPD or asthma.**

- **Increased Na+ retention:** Reduced blood pressure causes a decrease in renal perfusion, resulting in an increase in Na+ retention and plasma volume.In some cases, this compensatory response tends to elevate the blood pressure.
- **For these patients, β -blockers are often combined with a diuretic to prevent Na+ retention.**
- **Disturbances in glucose metabolism:** β -blockade leads to decreased glycogenolysis and decreased glucagon secretion. Therefore, if a Type I (formerly insulin-dependent) diabetic is to be given propranolol, very careful monitoring of blood glucose is essential, because pronounced hypoglycemia may occur after insulin injection.
- **Blockers also attenuate the normal physiologic response to hypoglycemia.**



- **Therapeutic effects:**
- **Hypertension**
- **Glaucoma**
- **Angina pectoris**
- **Myocardial infarction:** *Propranolol* and other β -blockers have a protective

Adverse effects:

1. **Bronchoconstriction**
2. **Arrhythmias**
3. **Sexual impairment**
4. **Disturbances in metabolism:** β -Blockade leads to decreased glycogenolysis and decreased glucagon secretion. Fasting hypoglycemia may occur.

Cardioselective β -blockers are preferred in treating asthmatic patients and patients who use insulin.

Thank you 🙂👀

المحاضرة فيها كمية حفظ رهيبة يارب ما تكونوا عم تدرسوها ليلة الامتحان

الى هنا انتهت مادة الميد ❤️🙏 بالتوفيق يارب 🙏 ما تنسونا من دعواتكم لأنه والله بحاجة لدعوة منكم ❤️❤️

Quiz Time ⏳

الأسئلة بتيجي Cases و بتعتمد على الحفظ و الربط و الاستنتاج بشكل رهيب ، حتلاحظوا هاد الشي و انتوا بتحلوا الأسئلة

1)Which of the following drugs could theoretically improve asthma symptoms?

A. Bethanechol

B. Pilocarpine

C. Pyridostigmine

D. Atropine

2)Sarin is a nerve gas that is an organophosphate cholinesterase inhibitor. Which agent could be used as an antidote to sarin poisoning?

A. Pilocarpine

B. Carbachol

C. Atropine

D. Physostigmine

3)Which drug is useful in treating sinus bradycardia?

A. Atropine

B. Cisatracurium

C. Neostigmine

D. Succinylcholine

4)Which of the following is correct regarding responses mediated by adrenergic receptors?

A. Stimulation of a1 receptors increases blood pressure.

B. Stimulation of sympathetic presynaptic B receptors increases norepinephrine release.

C. Stimulation of B receptors increases heart rate (tachycardia).

D. Stimulationof B receptors causes bronchoconstriction.

5)A 22-year-old male is brought to the emergency room with suspected cocaine overdose. Which of the following symptoms is most likely in this patient?

A. Hypertension

B. Bronchoconstriction

C. Bradycardia

D. Miosis (constriction of pupil)

6)A 12-year-old boy with a peanut allergy is brought to the emergency room after accidental consumption of peanuts. He is in anaphylactic shock. Which of the following drugs is most appropriate to treat this patient?

A. Norepinephrine

B. Phenylephrine

C. Dobutamine

D. Epinephrine

7)A 50-year-old male was in anaphylactic shock after being stung by a hornet. The medical team tried to reverse the bronchoconstriction and hypotension using epinephrine; however, the patient did not fully respond to the treatment. The patient's wife mentioned that he is taking a prescription medication for blood pressure. Which medication is he most likely taking that contributed to a reduced response to epinephrine?

A. Doxazosin

B. Propranolol

C. Metoprolol

D. Acebutolol

8)cause of death in organophosphate toxicity is:

A. Bradycardia

B. Increased bronchial secretions

C. Paralysis of the respiratory muscles

D. Depressionoftherespiratorycenter

E. All of the above

9)Alpha-1 agonists cause reflex bradycardia, which can be blocked by:

A. atenolol

B. atropine

C. mirtazapine

D. phenylephrine

E. propranolol

10)Following pretreatment with a muscarinic receptor blocking agent, the IV administration of norepinephrine is likely to result in:

A. ↑ HRand ↑ BP

B. ↑ HRand ↓ BP

C. ↓ HRand ↓ BP

D. ↓ HRand ↑ BP

E. no effect on HR, but ↑ BP

11) Commonalities of the sympathetic, parasympathetic, and somatic nervous systems involve which of the following neuroeffector transmitters?

(A) Acetylcholine. (B) Dopamine

(C) Epinephrine. (D) Norepinephrine

(E) Serotonin

12)A 47-year-old man is given atropine to decrease dental secretions during a root canal procedure. This agent is most likely to have an effect on which of the following target organs/glands?

(A) Adrenal medulla

(B) Kidney

(C) Pilomotor muscles

(D) Salivary glands

(E) Sweat glands

13)A 38-year-old woman presents to the ophthalmologist for a routine eye examination. She is given intraocular pilocarpine. She was supposed to be administered two drops in each to dilate the eyes for the examination. Unfortunately, the eyedrops were administered by a new technician who inadvertently administered 10 drops of pilocarpine in each eye. Which of the following agents should be immediately given to the patient?

(A) Atropine

(B) Carbachol

(C) Donepezil

(D) Galantamine

(E) Rivastigmine

14)A medical student is performing a summer research project evaluating the pharmacologic effects of atro- pine at varying doses. Doses are extrapolated from normal human doses of this agent. Slow infusion of this agent to a steady state dose of 0.5 mg would be expected to produce which of the following effects?

(A) Bradykinesia

(B) Coma

(C) Dilation of the pupils

(D) Dry mouth

(E) Tachycardia

لا تفوح رائحة الكعك الزكية إلا حين تمسها حرارة الفرن. كذلك أحلامنا لن تنضج مالم تمسها قسوة التجارب!

كيف بس التشبيه 😂👀