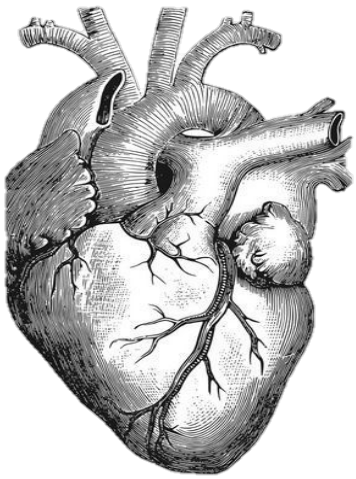
Pharmacology
Team 437

MED437
KING SAUD UNIVERSITY



Adrenergic Antagonists

Editing File

Objectives:

- Describe the different classifications for drugs that can block sympathetic nervous system.
- Describe the kinetics, dynamics, uses and side effects of alpha adrenergic drugs.
- Identify Difference between selective and non selective alpha blockers.
- Know the difference between tamsulosin and other selective alpha receptor blockers.
- Identify the different classifications for beta receptors blockers.
- Describe the kinetics, dynamics, uses and side effects of beta adrenergic drugs.
- Know the preferable drug for diseases as hypertension, glaucoma, arrythmia, myocardial infarction, anxiety, migraine and ect….

Important Notes Extra

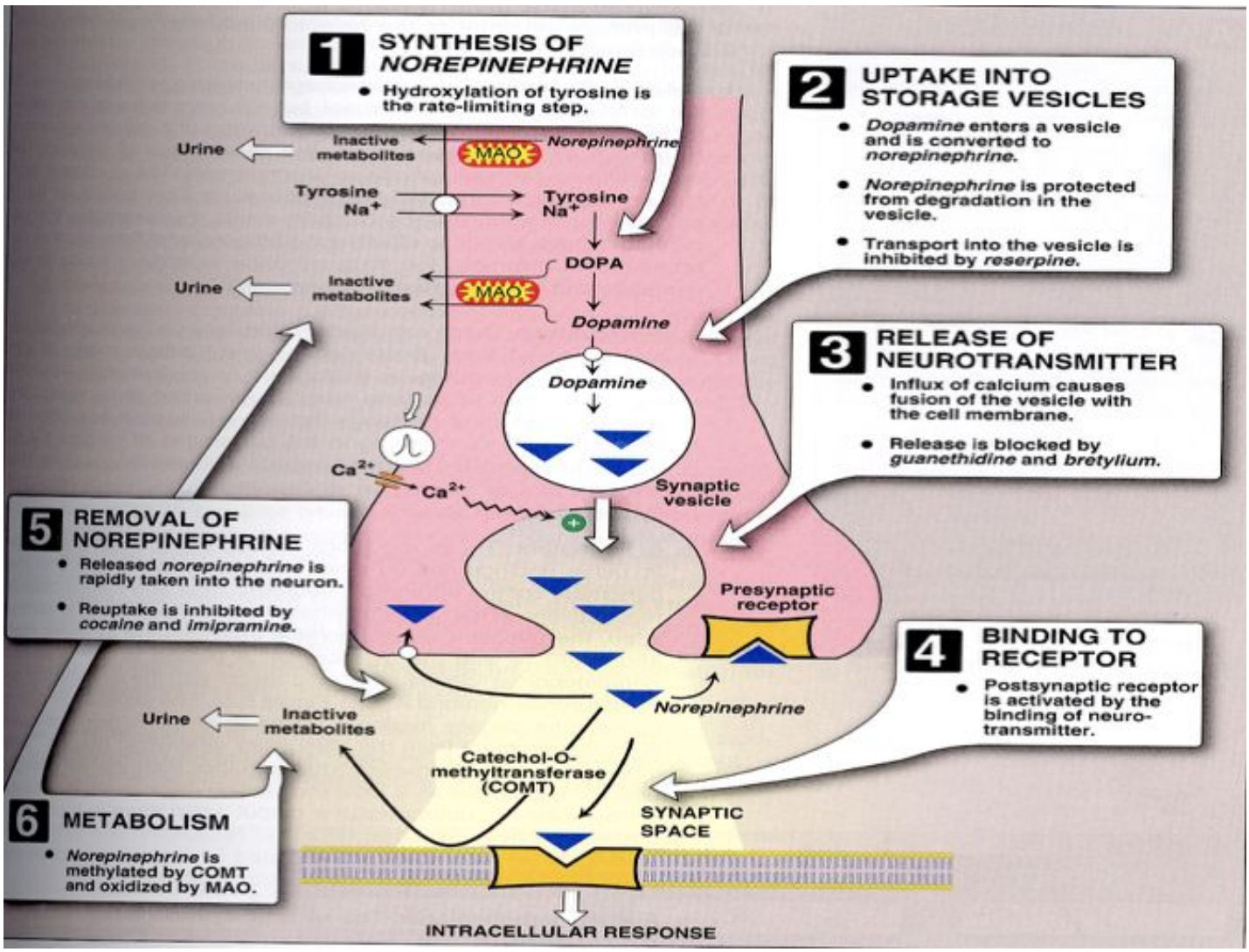
Review :)

Actions of Adrenergic Receptors

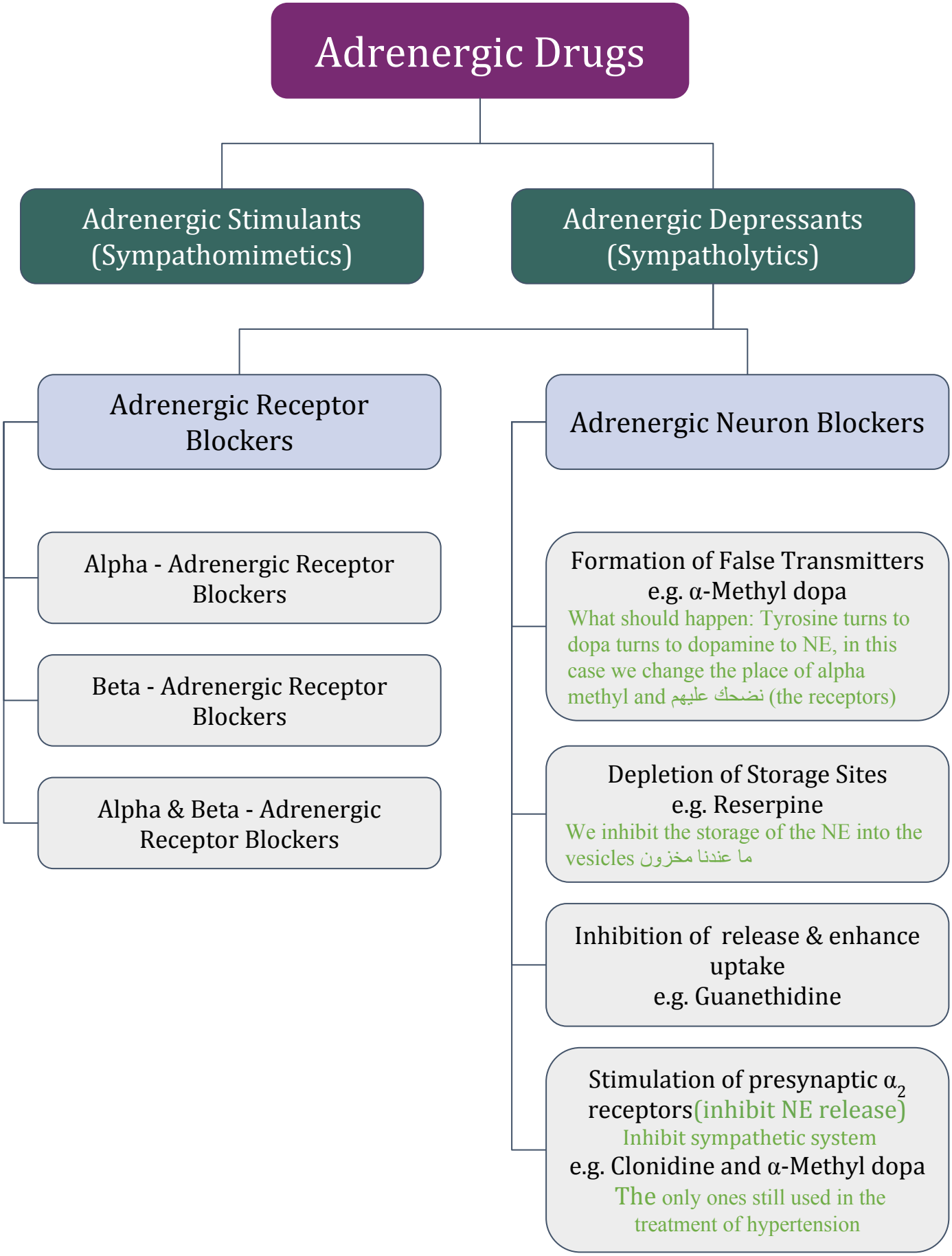
α_1	α_2	β_1	β_2		β_3
Post-synaptic	Pre-synaptic	Post-synaptic	Post-synaptic	Pre-synaptic	Post-synaptic
found in smooth muscle	-	found in heart & JG cells in kidney	found in smooth muscle	-	found in adipose tissue
excitatory (except GIT)	inhibition of NE (-ve feedback)	excitatory	inhibitory	↑release of NE (+ve feedback)	-
Contraction of: -radial eye muscle -pregnant uterus. -vasoconstriction of skin & peripheral blood vessels(hyper-tension) -sphincters in GIT & urinary bladder. -Relaxation of GIT muscles. -↑Glycogenolysis		-↑ heart rate: + chronotropic effect, Tachycardia. -↑ force of contraction : + inotropic effect, arrhythmia. -↑ conduction velocity: + dromotropic effect. -↑ blood pressure. -↑ renin release.	Relaxation of: -skeletal & coronary blood vessels (vasodilatation) - bronchial smooth muscles. -GIT muscles (constipation). -urinary bladder. -uterus (Delay premature labor) -Increase blood glucose level (hyperglycemia). -Tremor of skeletal muscles.		↑lipolysis ↑ free fatty acids.

If you know it skip it...

Review :)



If you know it skip it...


Adrenergic Drugs
Adrenergic Stimulants (Sympathomimetics)
Adrenergic Depressants (Sympatholytics)
Adrenergic Receptor Blockers
Alpha - Adrenergic Receptor Blockers
Beta - Adrenergic Receptor Blockers
Alpha & Beta - Adrenergic Receptor Blockers
Adrenergic Neuron Blockers
Formation of False Transmitters
e.g. α-Methyl dopa
What should happen: Tyrosine turns to dopa turns to dopamine to NE, in this case we change the place of alpha methyl and نضحك عليهم (the receptors)
Depletion of Storage Sites
e.g. Reserpine
We inhibit the storage of the NE into the vesicles ما عندنا مخزون
Inhibition of release & enhance uptake
e.g. Guanethidine
Stimulation of presynaptic α_2 receptors(inhibit NE release)
Inhibit sympathetic system
e.g. Clonidine and α-Methyl dopa
The only ones still used in the treatment of hypertension

Adrenergic Neuron Blockers

Drug	α-Methyl Dopa	Clonidine	Apraclonidine
Action	-Forms false transmitter that is released instead of NE -Centrally acting a2 adrenergic agonist that inhibits NE release - Centrally acting, crosses BBB and placenta.Safe to use by pregnant women	-Central a2 receptor agonist to inhibit NE release -Suppresses sympathetic outflow activity from the brain	Acts by decreasing aqueous humor formation. decreases I.O.P
Uses	Drug of choice in treatment of hypertension in pregnancy pre-eclampsia (condition of hypertension with impaired kidney function in pregnant women) gestational hypertension (hypertension only during period of pregnancy)	-Management of withdrawal symptoms of opiate treatment, alcohol withdrawal, benzodiazepines and nicotine dependence - Little use as antihypertensive agent due rebound hypertension upon abrupt withdrawal	Open angle glaucoma as eye drops.

Adrenergic receptor blockers

Classification of α-receptor Antagonists		
Non-selective antagonists	**α_1-selective antagonists**	**Selective α_2- adrenoceptor antagonists**
e.g. phenoxybenzamine & phentolamine.	e.g. prazosin, doxazosin, terazosin,tamsulosin.	e.g. yohimbine

Non-Selective α-Receptor Blockers

Drug	Phentolamine	Phenoxybenzamine
MOA	**Non-selective** antagonists of both α1 & α2 receptors.	
P.K	• Reversible block of both α1 & α2 receptors. (competitive inhibition) given IV • Short acting (4 hrs)	• Irreversible blocking of α1 & α2 receptors. (drug will make strong covalent bonds with receptor, non-competitive inhibition) Given Orally • Long-acting (24 hrs)
Pharmacological actions	They mostly affect blood pressure because they act on smooth muscle (endothelial cells of blood vessels) • Decrease peripheral vascular resistance.(because of vasodilation) • Postural hypotension.(Hypotension upon standing or movement يدوخ اذا قام) • Increase cardiac output (α_2 block). • Reflex tachycardia (due to fall in B.P, mediated by baroreceptor reflex and due to block α_2 in heart). • Vasodilation of blood vessels (α_1 block). -occurs by **two mechanisms:** 1) **Stimulation of baroreceptor reflex** that increase NE release. 2) **α2 blockade in heart** that abolishes presynaptic negative feedback for NE release.	
Indication	• Before removal of Pheochromocytoma **(tumor in the adrenal medulla that cause increase secretion of norepinephrine and epinephrine)** to prevent Hypertensive Crisis. Patient will complain from symptoms as if taking adrenaline. *(To memorize: Phentolamine, Phenoxybenzamine indicated for Pheochromocytoma)*	
ADRs	1)Headache vasodilation always causes headache 2)Nasal stuffiness or congestion (remember no excitation for A1) 3)Vertigo & drowsiness 4)Male sexual dysfunction (Inhibits ejaculation) 5) Tachycardia. 6) Postural hypotension.	
Contradiction	Both drugs can precipitate (cause) arrhythmias and angina. -Patients with decreased coronary perfusion. why? because these drugs will cause tachycardia = diastolic duration of the heart will ↓ = ↓ coronary perfusion.	

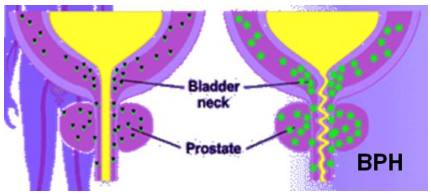
Selective α1 - adrenoceptor Antagonists

Drug	**α1-adrenoceptor Antagonists:** • Prazosin • Doxazosin • Terazosin
P.K	• Prazosin has short half-life. • Doxazosin, terazosin have long half lives.
MOA	Selective α1 -adrenoceptor Antagonists
Effect	• Vasodilation due to relaxation of arterial and venous smooth muscles. • Fall in arterial pressure with less reflex tachycardia than with non-selective α-blockers.
Indication	• Treatment of **essential hypertension**. (unknown cause) • Urinary obstruction associated with **benign prostatic hyperplasia** (BPH) (it can be either hyperplasia or hypertrophy) The idea is to relax the smooth muscle which will relieve urinary retention • **Raynaud's disease** (Vasospasm) causes some areas of your body such as your fingers and toes to feel numb and cold in response to cold temperatures or stress. Peripheral vascular disease

- Fall in arterial pressure with less reflex tachycardia than with non-selective α- blockers. why less tachycardia ??

non-selective a-blockers has two mechanism in tachycardia
1- it will block a1 receptor, so hypotension will occur and cause reflex tachycardia.
2- it will block a2 receptor, so that will increase NE release and affect the heart by binding to b1 receptor and cause more tachycardia.

Selective α1A & Selective α2 Antagonists

	Selective α1A	Selective α2
Drug	Tamsulosin(Uroselective)	Yohimbine(Yohimbe bark)
MOA	▪Relaxation of smooth muscles of bladder neck & prostate →improve urine flow. The hypertrophy will compress the bladder neck so relaxation will allow urine to flow.  ▪Has minimal effect on blood pressure. ▪α_{1A} receptors present in prostate and bladder neck.	Increase nitric oxide "NO"released in the corpus cavernosum thus producing vasodilator action and contributing to the erectile process.
Indication	▪Treatment of benign prostatic hypertrophy (BPH).(it can be either hyperplasia or hypertrophy) ▪Help with the passage of kidney stones.	Used as aphrodisiac* in the treatment of erectile dysfunction. *Aphrodisiac: a food, drink, or other thing that stimulates sexual desire.
ADRs	▪As before with non selective but to a lesser degree. only on α1A receptor so will have less ADR.	_____

Classification of β Adrenoceptors Blockers

1-According to Selectivity

Non-Selective (block β1 and β2)	Selective (block β1)	Mixed (block α & β receptors)
Propranolol, Pindolol, Sotalol, Timolol (PST) (eye)	Atenolol, Acebutolol, Bisoprolol, Esmolol, Metoprolol	Carvedilol Labetalol

2-According to presence of agonistic/antagonistic action i.e Intrinsic Sympathomimetic Activity (ISA)

No ISA

Atenolol, Bisoprolol, Metoprolol Propranolol Sotalol, Timolol, Carvedilol.

With ISA

Labetalol.

When a drug has ISA it is able to mimic the action of norepinephrine and epinephrine

3-According to presence of membrane stabilizing effects

Propranolol (Non selective β blocker)

Labetalol (α and β blocker)

i.e. Block Na Channels Quinidine-like action Antiarrhythmic action. These drugs will block the depolarizing process i.e will give local anesthetic drugs action

β ADRENOCEPTOR BLOCKERS
Pharmacokinetic Classification
According to their lipid solubility

- Lipophilic
- Hydrophilic

	Lipophilic	Hydrophilic
Oral Absorption	Complete	Irregular
Liver Metabolism	Yes	No
$t_{1/2}$	Short	Long
CNS Side Effects	High	Low
Drugs	Metoprolol Propranolol, Timolol Labetalol, Carvedilol	Atenolol, Bisoprolol, Esmolol , Sotalol

★ **CNS depressant effects i.e. Sedative effect→↓ Anxiety**

Pharmacokinetics of β–blockers:

Most of them are lipid soluble

- Lipid soluble β–blockers
- well absorbed orally.
- are rapidly distributed, cross readily BBB
- Have CNS depressant actions
- Metoprolol, propranolol, timolol, labetalol, carvedilol

Most of them metabolized in liver & excreted in urine.

Most of them have half-life from 3-10 hrs

- Except Esmolol (hydrophilic drug) (10 min. given intravenously)

Pharmacological actions of β–Adrenergic Blockers:

Cardiovascular System	Negative (inotropic,chronotropic,dromotropic) → ↓Cardiac Output (lower blood pressure as a result.)
Antianginal effects (ischemic heart disease)	• ↓ Heart rate (bradycardia). • ↓ force of contraction→ ↓cardiac work • ↓ Oxygen consumption due to bradycardia. Ischemic heart disease means no blood flow therefore no O2
Antiarrhythmic effects	↓excitability, ↓ automaticity & ↓conductivity (due to its sympathetic blocking).
Blood vessels β_2	↑peripheral resistance (PR) by blocking vasodilatory effect β2 ↓ blood flow to organs → cold extremities and fatigue contraindicated in peripheral diseases like Raynaud's disease why? because patients will already have vasoconstriction, by blocking β2 we will only make it worse. To treat → vasodilation
Blood pressure	Antihypertensive → ↓ BP in hypertensive patients due to effects on: • Inhibiting heart properties →↓ cardiac output (β_1) • β Blockade ↓renin secretion ↓ Ang II & aldosterone secretion(β_1) The inhibition of renin secretion is important because it is converted to angiotensin I then angiotensin II which is the most effective vasoconstrictor. It has direct constriction on smooth muscle (causing peripheral resistance), it goes to adrenal gland which makes Aldosterone causing water retention and Na reabsorption. This increases the blood pressure. This mechanism turns on during hypovolemic shock and bleeding. • Presynaptic inhibition of NE release from adrenergic nerves
Respiratory tract β_2	• Bronchoconstriction so it is Contraindicated in asthmatic patients & COPD patients, in this case it is better to give them a selective β_1 antagonist
Eye	↓ Aqueous humor production from ciliary body ↓ Reduce intraocular pressure (IOP) e.g. timolol as eye drops for glaucoma why eye drops? Because Timolol is nonselective so causes many ADRs therefore it is better to give it locally.
Intestine	↑ Intestinal motility
Metabolic effects	- Hypoglycemia by ↓ glycogenolysis in liver, ↓ glucagon secretion in pancreas - ↓ lipolysis in adipocytes - Na+ retention secondary to ↓BP → ↓renal perfusion This is a response reflex to the lowered blood pressure

Clinical Uses of β–receptor Blockers

Cardiovascular disorders	**Hypertension:** Propranolol, atenolol, bisoprolol Labetalol: α, β blockers in hypertensive pregnant & hypertensive crisis.
	Cardiac Arrhythmias: In supraventricular & ventricular arrhythmias. Bisoprolol(a selective B1) and carvedilol(Beta & alpha blocker) are preferred
	Angina pectoris: اقلل الشغل على القلب (reduce the load) ↓ heart rate, ↓ cardiac work & oxygen demand. ↓ the frequency of angina episodes. Reduces amount of blood flow to the chest
	Congestive heart failure: e.g. carvedilol: antioxidant and non selective α,B blocker – ↓ myocardial remodeling & ↓ risk of sudden death. During heart failure remodeling occurs which changes normal function of heart, so this drug will prevent it from happening.
	Myocardial infarction: Reduces the consequences Have cardioprotective effect ↓ infarct size ↓ morbidity & mortality which lead to ↓ myocardial O2 demand. Antiarrhythmic action. ↓ incidence of sudden death.
Pheochromocytoma	used with α-blockers (never alone) why? If I close beta I will protect the heart, but alpha will still be open and NE can overstimulate it and cause severe vasoconstriction leading to hypertensive crisis which is severe shooting of BP. - α-blockers lower the elevated blood pressure. - β-blockers protect the heart from NE.
Chronic glaucoma	Timolol as eye drops
Hyperthyroidism (thyrotoxicosis)	Protect the heart against sympathetic overstimulation by thyroxine Controls symptoms; tachycardia, tremors, sweating. (+C.O)
Anxiety (Social and performance type)	Controls symptoms due to sympathetic system stimulation as tachycardia, tremors, sweating. e.g. Propranolol
Migraine	Prophylactic by ↓ episodes of chronic migraine ↓ catecholamine-induced vasodilatation in the brain vasculature Headaches caused by vasodilation, Beta blockers cause vasoconstriction. e.g. Propranolol

Adverse Effects of β- Adrenoceptors Blockers

β1- receptor	Bradycardia. Hypotension. Heart failure.
β2- receptor only occur with non-selective β-blockers	Hypoglycemia. Bronchoconstriction(asthma,emphysema). Cold extremities and intermittent claudication by vasoconstriction. Erectile dysfunction and impotence. Coronary spasm in variant angina patients.
All β- Adrenergic blockers	Mask hypoglycemic manifestations i.e tachycardia,sweating ⟶ COMA. Diabetics won't know they are hypoglycemic because the drug will hide usual symptoms such as tachycardia which is why it is important that diabetics on beta blockers are monitored to avoid complications that could lead to coma. Depression & hallucinations. Gastrointestinal disturbances. Sodium retention.
Precautions	Sudden stoppage will give rise to a withdrawal syndrome: -Rebound angina, arrhythmia, myocardial infarction & hypertension WHY? Up-regulation of β-receptors. -To prevent withdrawal manifestations ⟶ drug withdrawn gradually. withdrawal of beta blockers has similar effect of corticosteroids withdrawal

Intermittent Claudication

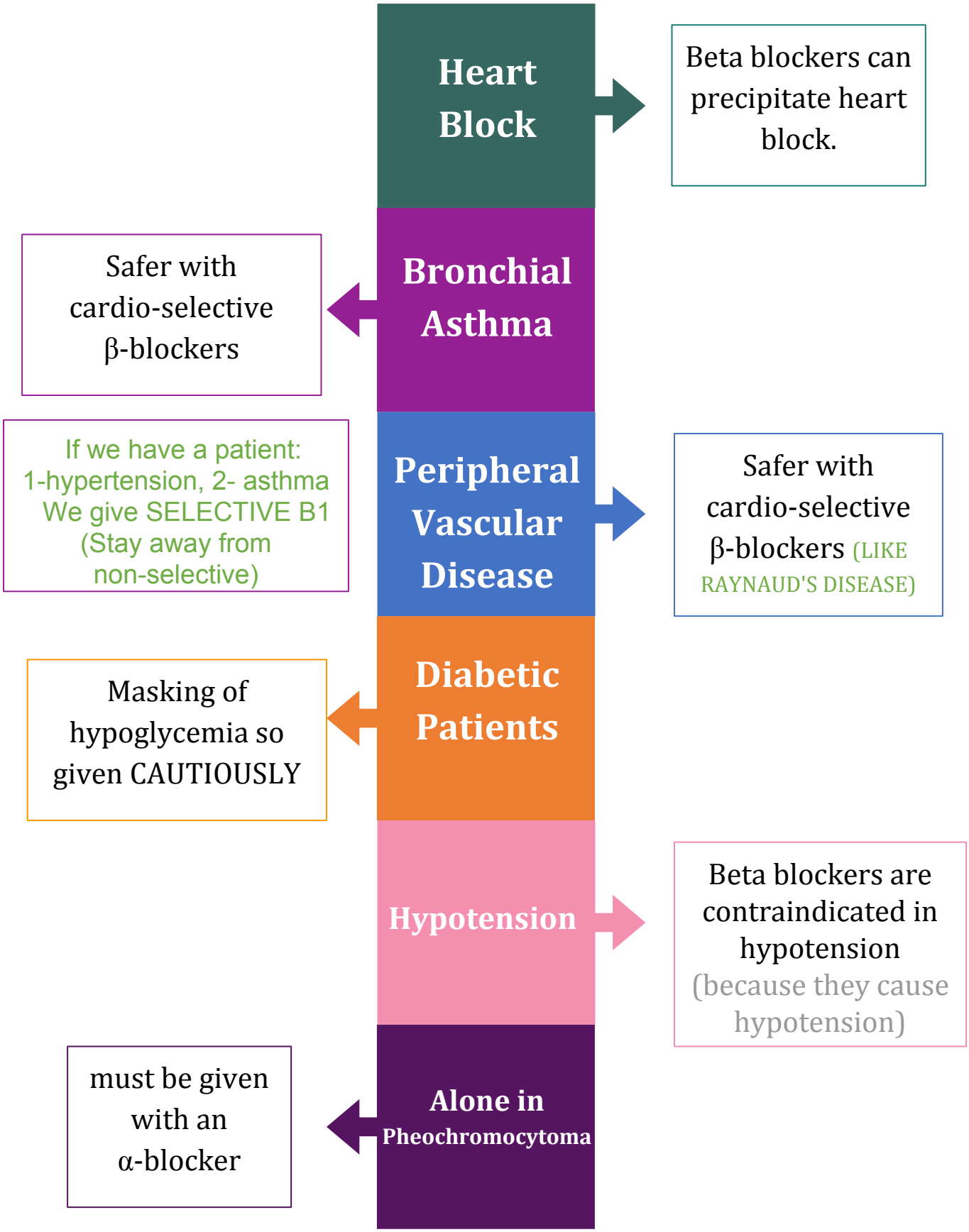
Peripheral artery disease that most commonly affects the legs, but other arteries may also be involved.

Symptoms: Leg pain with walking , resolves with rest.

Risk factors:

- Diabetes.
- Hypercholesterolemia.
- High blood pressure.

Contraindications of β- Adrenoceptors Blockers



β–Adrenergic blockers

Drug	**Propranolol** (is the chosen as prototype)
MOA	• Non-Selective Competitive Blocker of β1 & β2 • Membrane stabilizing action/ quinidine-like /local anesthetic effect • sedative actions /No ISA
Kinects	• Lipophilic, completely absorbed acts on CNS, sedative effect • 70% destroyed during 1st pass hepatic metabolism, • 90-95% protein bound • cross BBB and • excreted in urine • Can be given p.o or parenteral
Dynamics	β-blocking Effect: 1. Membrane Stabilization: Block Na channels → direct depressant to myocardium → has local anesthetic effect (anti-arrhythmic effects). 2. CNS Effect: Has sedative action ↓ tremors & anxiety → used to protect against social anxiety performance anxiety
Indications	• Hypertension • Arrhythmias • Angina • Myocardial infarction • Migraine [Prophylaxis] • Pheochromocytoma; used with α-blockers (never alone) • Chronic glaucoma • Tremors • Anxiety: (specially social & performance type) sedates them • Hyperthyroidism

β–Adrenergic Blockers

Drug	**Propranolol** cont.
Actions	Heart; by block β1 • Inhibit heart properties → ↓ cardiac output • Has anti -ischemic action → ↓ cardiac work + ↓ O2 consumption • Has anti-arrhythmic effects → ↓excitability, automaticity & conductivity + by membrane stabilizing activity BP; by block β1 & β2 • Has antihypertensive action by→ Inhibiting heart properties → ↓ cardiac output • B blockade : ↓ renin & RAAS system • Presynaptic inhibition of NE release from adrenergic nerves • Inhibiting sympathetic outflow in CNS Mainly by β2 blockade: • Blood Vessels: Vasoconstriction → ↓blood flow specially to muscles, other organs except brain cold extremities • Bronchi: Bronchospasm specially in susceptible patients • Intestine: ↑ Intestinal motility • Metabolism: ○ In liver: ↓ Glycogenolysis → Hypoglycaemia ○ In pancreas:↓ Glucagon secretion ○ In adipocytes:↓ Lipolysis ○ In skeletal muscles: ↓glycolysis • On peripheral & central nervous systems: ○ Has local anesthetic effect ○ ↓ tremors & ↓ anxiety

Selective β*1*- receptor Blockers

- Selectivity present in low doses but is lost at high doses will have no change on glucose metabolism or beta 3(lipid profile).

- no change in lipid or glucose ⟶ Preferable in diabetics\Dyslipidemias.

- no bronchoconstriction ⟶ Preferable in patients with asthma and COPD

- no effect on peripheral resistance.

also preferable in :

- Raynaud's phenomenon & peripheral vascular disease (PVD).
- Variant Angina (coronary spasm).

α and β- Adrenoceptors Blockers

✓Non selective β blockers with concurrent α_1 blocking action.
✓Produce peripheral vasodilation
✓Decrease blood pressure
✓Used in the treatment of hypertensive emergencies as they can rapidly lower BP.
Very fast acting thats why used in emergencies eg: Labetalol and Carvedilol

Drug	Labetalol	Carvedilol
P.K	• Do not alter serum lipids or blood glucose. • given p.o and i.v	• Favorable metabolic profile.
MOA	• Rapid acting non-selective α_1 & β blocker . • has ISA and local anesthetic effect.	• Non-selective α_1 & β Blocker • no ISA & no local anesthetic effect. • has antioxidant action.
Indication	• Severe hypertension in pheochromocytoma. • Hypertensive crisis (e.g. during abrupt withdrawal of clonidine). clonidine causes rebound hypertension in abrupt withdrawal • pregnancy-induced hypertension .(alpha-methyl dopa for pre-eclampsia)	• Used effectively in CONGESTIVE HEART FAILURE reverses its patho-physiological changes.
ADRs	• Orthostatic hypotension • sedation and dizziness.	• Edema • Orthostatic hypotension .

Summary of β–Adrenergic blockers:

Uses	Drugs
Hypertension	Atenolol, Bisoprolol,Metoprolol, Propranolol
Cardiac arrhythmia	Esmolol (ultra-short acting), Atenolol, Propranolol
Congestive heart failure	Carvedilol, Bisoprolol, Metoprolol MBC
Myocardial infarction	Atenolol, Metoprolol, Propranolol
Glaucoma	Timolol
Migraine prophylaxis	Propranolol
Relief of anxiety (social & performance)	Propranolol
Thyrotoxicosis	Propranolol

Drug	MOA	Uses
Propranolol	Non selective β1, β2 blocker	Migraine prophylaxis Hyperthyroidism (thyrotoxicosis) Relieve anxiety (social performance)
Timolol	β1, β2 blocker	Glaucoma
Atenolol Bisoprolol Metoprolol	β1 blocker	Myocardial infarction Hypertension
Esmolol	β1 blocker Ultra short acting	Cardiac arrhythmia
Carvedilol	Non selective α, β blocker	Congestive heart failure
Labetalol	α, β blocker	Hypertension in pregnancy Hypertensive emergency

Summary of α- Adrenergic blockers

Adrenergic neuron blockers drugs work in the presynaptic neurons		
Drug	MOA	Uses
α-Methyl Dopa	1-Forms false transmitter that is released instead of NE. 2-Acts as central α2 receptor agonist to inhibit NE release	- gestational hypertension - pre-eclampsia Treatment of hypertension in pregnancy
Clonidine	1-Acts as central α2 receptor agonist to inhibit NE release. 2-suppresses sympathetic outflow activity from the brain.	Little used as antihypertensive agent due to rebound hypertension upon abrupt withdrawal.
Apraclonidine	acts by decreasing aqueous humor formation.	in open angle glaucoma as eye drops.
Reserpine	Depletion of storage sites	-
Guanethidine	Inhibition of release & enhance uptake	-

Adrenergic Receptor Blockers

Drugs work postsynapticon the receptor

	Drug		Pharmacological actions	Therapeutic Uses	contra-indicated	Side effect
Non-selective antagonists	Phenoxy-benzamine	Irreversible block of α_1 and α_2 receptors Long-acting (24 hrs)	- Decrease peripheral vascular resistance - Postural hypotension. - Reflex tachycardia, due to the fall in B.P, mediated by baroreceptor reflex and due to block a_2 in heart.	Pheochromocytoma: given before surgical removal to protect against hypertensive crisis.	Both can precipitate arrhythmias and angina in patients with decreased coronary perfusion.	-Postural hypotension -Headache -Tachycardia -Vertigo & drowsiness -Nasal stuffiness or congestion -Male sexual dysfunction (inhibits ejaculation).
	Phentolamine	Reversible blocking of α_1 &$\alpha_2$receptors Short acting (4 hrs)				
a_1-selective antagonists	Prazosin	Short half-life	-Vasodilatation due to relaxation of arterial and venous smooth muscles - Fall in arterial pressure less reflex tachycardia than with non-selective a blockers	-Treatment of essential hypertension - Urinary obstruction of benign prostatic hypertrophy (BPH). Raynaud's disease.	-	-
	Doxazosin	Long half life			-	-
	Terazosin				-	-
Selective a_{1A}–antagonists	Tamsulosin	-	- Relaxation of smooth muscles of bladder neck & prostate →improve urine flow. Has minimal effect on blood pressure.	is used in the treatment of benign prostatic hypertrophy (BPH).	-	as non-selective but to a lesser degree
a_2-selective antagonists	Yohimbine	-	Increase nitric oxide released in the corpus cavernosum thus producing vasodilator action and contributing to the erectile process.	Used as aphrodisiac in the treatment of erectile dysfunction.	-	-

Questions

MCQs:

1- A 67 year old man with history of Angina and hypertension presented to the ER with tachycardia. Which of the following drugs should be prescribed to him:

A) Phenoxybenzamine
B) Doxazosin
C) Clonidine
D) Yohimbine

2- A 27 year old smoker with Anxiety disorder presented to the ER with symptoms of withdrawal. He was previously prescribed benzodiazepines as treatment for Anxiety. Which of the following is the drug of choice:

A) Clonidine
B) Apraclonidine
C) α-Methyl dopa
D) Reserpine

3- A 32 year old man presented to the ER with hypertension and was prescribed treatment. He later returned to the ER complaining of sexual dysfunction. Which of the following was prescribed:

A) Yohimbine
B) Clonidine
C) α-Methyl dopa
D) Phenoxybenzamine

4- A 56 year old man with hypertension complained of urinary hesitancy. He was diagnosed with Benign Prostatic Hypertrophy. Which of the following is the drug of choice:

A) Tamsulosin
B) Yohimbine
C) Doxazosin
D) Phentolamine

1- B
2- A
3- D
4- A

Questions

MCQs:

5- Which of the following is used to treat erectile dysfunction:

A) Tamsulosin
B) Yohimbine
C) Doxazosin
D) Phentolamine

6- Which can be used to treat Raynaud's disease:

A) Non-Selective α-Adrenoceptor Antagonists
B) Selective α1- adrenoceptor Antagonists
C) Selective α2- adrenoceptor Antagonists
D) Selective α1A-adrenoceptor Antagonist

7- Patient with glaucoma, which of the following drugs is used in the treatment of glaucoma?

A) Atropine.
B) Timolol.
C) Tropicamide.
D) Propranolol

8-Pregnant woman with hypertension, what is the best choice to treat her high blood pressure?

A) Labetalol.
B) Propranolol.
C) Timolol.
D) Atenolol

9-Which one of the following beta blockers can be used for migraine prophylaxis ?

A) Propranolol.
B) Timolol.
C) Carvedilol.
D) Esmolol

10-Which of the following is not a clinical use of β–receptor Blockers?

A) Hypotension
B) Arrhythmia
C) Angina pectoris
D) Myocardial infarction

11- A 49 years old diabetic patient was brought to the emergency because he is in hypoglycemia coma, after taking history the patient is diabetic and has hypertension and the doctor prescribe to him a drug for his hypertension, what is the most likely drug the doctor prescribed ?

A) Atenolol.
B) Metoprolol.
C) Propranolol.
D) Bisoprolol.

5- B
6- B
7-B
8-A
9-A
10-A
11-C

Questions

SAQ:

- **A pregnant woman was diagnosed with Gestational Hypertension. What is the drug of choice and its mechanism of action?**

1) α-Methyl dopa
2) It is a centrally acting α2 adrenergic agonist that inhibits the release of NE by forming false transmitters

---

- **A former alcoholic presented with symptoms of withdrawal. What is the drug of choice and its mechanism of action?**

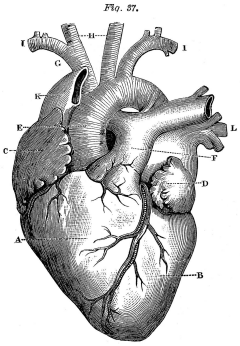
1) Clonidine
2) Acts as α-2 receptor agonist to inhibit NE release and suppresses sympathetic outflow activity from the brain.

- **A 55 years old diabetic patient was brought to the emergency because he is in hypoglycemia coma, after taking history the patient is diabetic and has hypertension and the doctor prescribed to him a drug for his hypertension**
  What is the most likely drug the doctor prescribed in this case and
  What is the mechanism of action of this drug ?

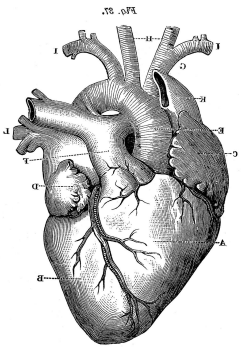
1) Propranolol or any Non-selective B
2) Block all type of beta receptor (B1 & B2 & B3) .

- **What are the 3 mechanism in which beta blockers act as Antihypertensive drugs?**

1) Decreasing cardiac output by blocking beta 1 in heart
2) inhibiting renins release and stopin RAAS therefore decreasing volume and blood pressure
3) Presynaptic inhibition of NE release from adrenergic nerves



“It is not hard, you just made it to the end!”



Team Leaders:

Hadeel Awartani Yazeed Al-Harbi

Thanks for those who worked on this lecture:

Dana Al-Rasheed
Ghadah Al-Muhanna
Aljohara Al-Shunaifi
Ghadah Al-Haidari
Rotana Khateeb
Rinad Al-Ghoraiby
Sarah Al-Sultan
Lujain Al-Zaid
Alanoud Al-Mufarrej
Rawan Al-Tamimi
Majd Al-Barrak
Ghaida Al-Sanad
Sarah Al-Kathiri
Ghadah Al-Qarni
Alanoud Al-Mansour
Alanoud Al-Eissa
Dana Al-Kady
Khloud Al-Waheibi
Maha Al-Amri
Munira Al-Hadleg
Nouf Al-Otaibi
Noura Al-Othaim
Rahaf Al-Thunayan
Shahad Al-Tayash
Dimah Al-Araifi
Fatimah Al-Bassam
Hind Al-Orier
Laila Al-Sabbagh

References:

✓ Doctors’ notes and slides

@Pharma4370 pharmacology437@gmail.com