Adrenergic Antagonists

• Red: important
• Black: in male / female slides
• Pink: in female's slides only
• Blue: in male's slides only
• Green: Dr's notes
• Grey: Extra information, explanation

OBJECTIVES:

By the end of this lecture, students should be able to:

**Chapter 1**
- ✓ Outline the mechanisms of action of adrenergic neuron blockers.
- ✓ Classify α-receptor blockers into selective & non-selective.
- ✓ Know the pharmacokinetic aspects & pharmacodynamic effects of adrenergic blockers.
- ✓ Identify the specific uses of non selective and selective α-adrenergic blockers.

**Chapter 2**
- ✓ Outline the mechanisms of action of B-blockers.
- ✓ Classify B-receptor blockers into selective & non-selective.
- ✓ Know the pharmacokinetic aspects & pharmacodynamic effects of B-adrenergic blockers.
- ✓ Identify the specific uses of non selective and selective B-adrenergic blockers.

Editing File
### Review

"just for better understanding ":

**Post-synaptic located in tissue**

(meaning it is mediated by a neuron which received a signal from a preganglionic neuron by synopsis)

<table>
<thead>
<tr>
<th>α1</th>
<th>β2</th>
<th>β1</th>
<th>β3</th>
</tr>
</thead>
<tbody>
<tr>
<td>excitatory in function (cause contraction) except in GIT</td>
<td>inhibitory in function (cause relaxation)</td>
<td>excitatory in function, present mainly in heart, juxtaglomerular cells of the kidney</td>
<td>In adipose tissue</td>
</tr>
</tbody>
</table>

### Present mainly in smooth muscles

- Contraction of pregnant uterus
- Relaxation of the uterus (Delay premature labor) also called tocolytic effect
- Vasoconstriction of skin & peripheral blood vessels → increased peripheral resistance (resistance to blood flow due to constriction of blood vessels) → hypertension. Agonists used as nasal decongestants.
- Relaxation of skeletal & coronary blood vessels (vasodilatation)
- Relaxation of GIT muscles & urinary bladder’s muscles. Contraction of GIT sphincter (constipation) & urinary bladder’s sphincter urinary retention
- Contraction of radial muscle of eye causes active mydriasis, (dilation of pupil, cholinergic agents have no effect on this muscle)
- Relaxation of bronchial smooth muscles (bronchodilation)
- Tremor of skeletal muscles
- Increase blood glucose level (hyperglycemia), by:
  - ↑ glycolysis
  - ↑ glucagon release from pancreas
  - ↑ liver & muscle glycogenolysis

### Pre-synaptic

**Inhibition of norepinephrine release (negative feedback mechanism)**

How? this mainly happen by an autoreceptor ‘presynaptic receptor’ which is present on the neuron releasing the neurotransmitter itself, the neurotransmitter bind to the receptor of the same neuron it was released by and inhibiting further release of the neurotransmitter, producing a negative feedback mechanism

**Increase release of norepinephrine (Positive feedback mechanism)**

- ↑ heart rate: chronotropic effect (Tachycardia)
- ↑ force of contraction:
  - ↑ inotropic effect
  - Increase cardiac output
  - ↑ conduction velocity:
    - ↑ dromotropic effect (via A.V. node)dromotropic effect means an effect in the speed of conduction of electrical impulses
- ↑ blood pressure
- ↑ renin release
  - (this is an enzyme produced by the kidney in response to stretch receptors found on blood vessels, its function is to increase blood pressure)
- ↑ lipolysis
- ↑ free fatty acids
Adrenergic Drugs

Adrenergic Stimulants (Sympathomimetics)
- Selective
  - Act on: $\alpha_1$
    - e.g. Phenylephrine
  - Act on: $\beta_1$
    - e.g. Dobutamine
- Non-Selective
  - Act on: $\alpha_1, \alpha_2, \beta_1, \beta_2, \beta_3$
    - e.g. Adrenaline
  - Act on: $\beta_1, \beta_2, \beta_3$
    - e.g. Isoprenaline

Adrenergic Receptor Blockers
- Selective
  - $\alpha$-Adrenergic Receptor Blockers
    - e.g. Phentolamine
  - $\beta$-Adrenergic Receptor Blockers
    - e.g. Propranolol
  - $\alpha$ & $\beta$-Adrenergic Receptor Blockers
    - e.g. Labetalol
- Non-Selective
  - $\alpha$1+$\alpha$2
    - e.g. Prazosin
  - $\beta$1+$\beta$2
    - e.g. Esmolol

Adrenergic Neuron Blockers
- Formation of False Transmitters
  - e.g. $\alpha$-Methyl dopa
- Depletion of storage sites
  - e.g. Reserpine
- Inhibition of release & enhance uptake
  - e.g. Guanethidine
- Stimulation of presynaptic $\alpha_2$ receptors
  - e.g. Clonidine AND $\alpha$-Methyl dopa

Adrenergic Depressants (Sympatholytics)
### Adrenergic Neuron Blockers Drugs

<table>
<thead>
<tr>
<th>Action</th>
<th>α-Methyl Dopa</th>
<th>Clonidine</th>
<th>Apraclonidine</th>
</tr>
</thead>
</table>
| - Forms false transmitter that is released instead of NE “α-methylnoradrenaline replaces NE in vesicles”  
- Centrally acting α2 adrenergic agonist that inhibits NE release “Can cross BBB” | - Central α2 receptor agonist to inhibit NE release  
- Suppresses sympathetic outflow activity from the brain | Acts by decreasing aqueous humor formation. |

### Uses

<table>
<thead>
<tr>
<th>Uses</th>
<th>α-Methyl Dopa</th>
<th>Clonidine</th>
<th>Apraclonidine</th>
</tr>
</thead>
</table>
| - Drug of choice in treatment of hypertension in pregnancy (gestational hypertension, pre- eclampsia “disorder of pregnancy characterized by proteinuria and rise in BP” )  
“NO teratogenic effect “ | - Little use as antihypertensive agent due to rebound hypertension upon abrupt withdrawal. “side effect”  
“Extra: Clonidine causes downregulation of α2 receptors, but its efficacy as an α2 agonist compensates for the decreased receptors. Therefore in withdrawal physiological neurotransmitters fail to produce the same effect, and due to poor stimulation of α2 receptors rebound hypertension occurs” | Open angle glaucoma as eye drops (topical) |

### Adrenergic Receptors Blockers

**Classification of α-receptor Antagonists**

<table>
<thead>
<tr>
<th>selective α1- antagonists</th>
<th>Non-selective α antagonists</th>
<th>Selective α2-adrenoceptor</th>
</tr>
</thead>
<tbody>
<tr>
<td>e.g. prazosin, doxazosin, terazosin</td>
<td>e.g. phenoxybenzamine, phentolamine</td>
<td>e.g. yohimbine</td>
</tr>
</tbody>
</table>
Non-Selective α-Receptor Blockers

<table>
<thead>
<tr>
<th>MOA</th>
<th>Phentolamine</th>
<th>Phenoxybenzamine</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Non-selective antagonists of both α1 &amp; α2 receptors.</td>
<td></td>
</tr>
</tbody>
</table>

**P.K**
- Reversible block of both α1 & α2 receptors. *“non-covalent bond so less duration of action”*
- Short acting (4 hrs)
- Irreversible blocking of α1 & α2 receptors. *“by covalent bond”*
- Long-acting (24 hrs)

**Pharmacological actions**
- Increase cardiac output (α2 block)
- Decrease peripheral vascular resistance.
- Postural (orthostatic) hypotension. *“due to baroreceptor reflex, pull of gravity and reduced BP contribute to low venous return which causes hypotension when standing”*
- Reflex tachycardia due to fall in B.P, mediated by baroreceptor reflex and due to block α2 in heart.

**Indication**
In Pheochromocytoma: Should be given before surgical removal to protect against hypertensive crisis. (Pheochromocytoma is a tumor of the adrenal medulla that causes an excessive release of NA "synthesized in the medulla", resulting in an overstimulation of α1 receptors, resulting in hypertension)

**ADRs**
- Headache
- Nasal stuffiness or congestion
- Vertigo & drowsiness *“caused by the hypotension”*
- Male sexual dysfunction (Inhibits ejaculation)
- Tachycardia
- Postural hypotension.

**Contradiction**
Patients with decreased coronary perfusion, because both drugs can precipitate arrhythmias and angina.

---

Selective α1 - adrenoceptor Antagonists

- **Prazosin**
- **Doxazosin**
- **Terazosin**

<table>
<thead>
<tr>
<th>MOA</th>
<th>Selective α1 -adrenoceptor Antagonists</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Prazosin has short half-life.</td>
</tr>
<tr>
<td></td>
<td>Doxazosin, terazosin have long half lives.</td>
</tr>
</tbody>
</table>

**P.K**
- Vasodilation due to relaxation of arterial and venous smooth muscles.
- Fall in arterial pressure with less reflex tachycardia than with non-selective α- blockers.
- First dose may produce an orthostatic hypotensive response that can result in syncope and fainting.

**Pharmacological actions**
- Treatment of essential hypertension WITH prostate enlargement.(Hypertrophy)
- Urinary obstruction associated with benign prostatic hyperplasia (BPH).
- Raynaud's disease causes some areas of your body such as your fingers and toes to feel numb and cold in response to cold temperatures or stress.
# Selective α1A & Selective α2 Antagonists

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Tamsulosin</th>
<th>Yohimbine</th>
</tr>
</thead>
</table>
| MOA            | - Relaxation of smooth muscles of bladder neck & prostate – improve urine flow.  
                | - Has minimal effect on blood pressure.                                        | 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).                            | Used as aphrodisiac in the treatment of erectile dysfunction.                                                        |
| ADRs           | As before with non-selective but to a lesser degree.                        | ————————————————————                                                                                                       |
### β- Adrenoceptors Blockers

#### Pharmacodynamic Classifications:

<table>
<thead>
<tr>
<th>According to selectivity</th>
<th>Non-selective “β1 &amp; β2”</th>
<th>Selective “β1”</th>
<th>Mixed “β &amp; α” act on α1 and classes of β :β1,β2,β3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Propranolol</td>
<td>Atenolol</td>
<td></td>
<td>Labetalol</td>
</tr>
<tr>
<td>Pindolol</td>
<td>Bisoprolol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sotalol</td>
<td>Esmolol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Timolol</td>
<td>Acebutolol</td>
<td></td>
<td>Carvedilol</td>
</tr>
<tr>
<td>Oxprenolol</td>
<td>Celiprolol</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

#### According to presence of agonistic/antagonistic action

<table>
<thead>
<tr>
<th>Intrinsic Sympathomimetic Activity (ISA)</th>
<th>Without ISA</th>
<th>With ISA “adrenergic partial agonists”</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Without ISA</td>
<td>With ISA “adrenergic partial agonists”</td>
</tr>
<tr>
<td>Atenolol</td>
<td></td>
<td>Labetalol</td>
</tr>
<tr>
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<td></td>
<td></td>
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<td>Pindolol</td>
</tr>
<tr>
<td>Timolol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carvedilol</td>
<td></td>
<td>Oxprenolol</td>
</tr>
</tbody>
</table>

#### According to presence of membrane stabilizing effects

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Propranolol</td>
<td>-Block Na Channels</td>
</tr>
<tr>
<td></td>
<td>-Quinidine-like action</td>
</tr>
<tr>
<td>labetalol</td>
<td>-Antiarrhythmic action</td>
</tr>
<tr>
<td></td>
<td>“Local anesthetic”</td>
</tr>
</tbody>
</table>
Pharmacokinetic Classification:

### According to lipid solubility

<table>
<thead>
<tr>
<th></th>
<th>Lipophilic</th>
<th>Hydrophilic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral absorption</td>
<td>Complete</td>
<td>Irregular</td>
</tr>
<tr>
<td>Liver metabolism</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>T 1/2</td>
<td>Short</td>
<td>Long</td>
</tr>
<tr>
<td>CNS side effects</td>
<td>Cross BBB, High depressant actions</td>
<td>Low</td>
</tr>
<tr>
<td>Drugs</td>
<td>Metoprolol / Propranolol, Timolol / Labetalol, Carvedilol</td>
<td>Atenolol / Bisoprolol, Esmolol / Sotalol</td>
</tr>
</tbody>
</table>

Pharmacological actions:

**CVS**
- Negative (inotropic, chronotropic, dromotropic) → ↓ Cardiac Output

**Antianginal effects (ischemic heart disease):**
- ↓ Heart rate (Bradycardia) → ↓ Oxygen consumption
- ↓ Force of contraction → ↓ Cardiac work

**Blood vessels β2:**
- Increase Peripheral resistance (PR) by blocking vasodilatory effect β2
- ↓ Blood flow to organs → cold extremities contraindicated in Raynaud's disease.

**Blood pressure:**
- Antihypertensive → ↓ BP in hypertensive patients due to effects on:
  - Inhibiting heart properties → ↓ cardiac output (β1)
  - β Blockade ↓ renin secretion ↓ Angiotensin II & aldosterone secretion (β1)
  - Presynaptic inhibition of norepinephrine release from adrenergic nerves

**Antiarrhythmic effects:** (class II)
- ↓ Excitability, ↓ automaticity & ↓ conductivity
  Due to its sympathetic blocking.

**Respiratory tract β2**
- Bronchoconstriction contraindicated in asthmatic patients.

**Eye**
- ↓ Aqueous humor production from ciliary body
- ↓ Intraocular pressure (IOP)
  E.g. timolol as eye drops for glaucoma.

**Metabolic effects & Intestine**
- Hypoglycemia by: ↓ glycogenolysis in liver & ↓ glucagon secretion in pancreas.
- Lipolysis in adipocytes
- Na+ retention secondary to ↓ blood pressure → ↓ renal perfusion
- Increase Intestinal motility.
## Clinical uses:

### Cardiovascular disorders

<table>
<thead>
<tr>
<th>Propranolol</th>
<th>Bisoprolol carvedilol</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hypertension</strong></td>
<td><strong>Angina pectoris</strong></td>
</tr>
<tr>
<td>Atenolol “Preferred in regular treatment of hypertension”</td>
<td>↓ Heart rate, ↓ cardiac work &amp; oxygen demand.</td>
</tr>
<tr>
<td>Bisoprolol Labetalol: α &amp; β blockers in hypertensive pregnant &amp; hypertensive crisis.</td>
<td>↓ The frequency of angina episodes.</td>
</tr>
</tbody>
</table>

### Carvedilol

- **Antioxidant** “Decrease formation of free radicals” and non selective α & β blocker.
- ↓ Myocardial remodeling & risk of sudden death.

### Congestive heart failure

- “Myocardial remodeling = hypertrophy, hyperplasia and increase apoptosis of cardiac muscle cells after injury”

### Myocardial infarction

- Cardio-protective effect
  - ↓ infarct size “infarct = dead tissue due to an ischemic process”
  - ↓ morbidity & mortality → ↓ myocardial O2 demand.
  - Anti-arrhythmic action.
  - ↓ Incidence of sudden death.

### Other disorders

- **Pheochromocytoma**
  - Used with α-blockers never alone
    - α-blockers lower the elevated blood pressure.
    - β-blockers protect the heart from norepinephrine.
  - **Timolol** as eye drops
    - ↓ secretion of aqueous humor by ciliary body.
    - ↓ Intraocular pressure (IOP)

- **Chronic glaucoma**

- **Thyrotoxicosis**
  - Protect the heart against sympathetic overstimulation.
  - Controls symptoms; Tachycardia, tremors and sweating.

- **Anxiety**
  - Social and performance type.
  - Controls symptoms due to sympathetic system stimulation as tachycardia, tremors, sweating.

- **Migraine**
  - Prophylactic
    - ↓ episodes of chronic migraine.
    - ↓ catecholamine-induced vasodilatation in the brain vasculature.

- **Propranolol**

- **Pheochromocytoma**
  - Used with α-blockers never alone
    - α-blockers lower the elevated blood pressure.
    - β-blockers protect the heart from norepinephrine.

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- **Migraine**
  - Prophylactic
    - ↓ episodes of chronic migraine.
    - ↓ catecholamine-induced vasodilatation in the brain vasculature.
Adverse effects:

Due to blockade of $\beta_1$-receptor:
- Bradycardia
- Hypotension
- Heart failure

Due to blockade of $\beta_2$-receptor:
- Hypoglycemia.
- ↑ Triglyceride $\rightarrow$ hypertriglyceridemia.
- Bronchoconstriction.
- cold extremities & intermittent claudication (due to vasoconstriction).
- Erectile dysfunction & impotence except Nebivolol will increase NO.
- Coronary spasm in variant angina patients.

All $\beta$ adrenergic blockers
- Mask hypoglycemic manifestations in diabetic patients "Delay the recovery of hypoglycemia" $\rightarrow$ COMA.
- Depression and hallucinations.
- Gastrointestinal disturbances.
- Sodium retention, due to reduced renal perfusion which is secondary to hypotension.
- Fatigue.

Precautions
- Sudden stoppage will give rise to a withdrawal syndrome: Rebound angina, arrhythmia, myocardial infarction & Hypertension.
- This is due to Up-regulation of $\beta$-receptors. "Increase numbers of $\beta$ receptor" To prevent withdrawal manifestations $\rightarrow$ drug withdrawn gradually.

Contraindications:
- Peripheral vascular disease e.g: Raynaud's
- Hypotension
- Diabetes GIVEN CAUTIOUSLY
- Bronchial Asthma
- Heart Block
- Alone in Pheochromocytoma "must be given with an $\alpha$-blockers"
### Propranolol

#### MOA
- Non-Selective $\beta_1$ & $\beta_2$ blockers.
- Membrane stabilizing action.
- Quinidine-like.
- No ISA, but have sedative action.

#### P.K
- Lipophilic:
  - Completely absorbed.
  - 70% destroyed during first pass hepatic metabolism.
  - 90-95% protein bound.
  - Cross BBB and excreted in urine.
  - Can be given Orally (P.O) or parenteral.

#### Pharmacological actions

**$\beta$-blocking Effect:**
1. **Antiarrhythmic effects:** Membrane Stabilization: Block Na channels → direct depressant to myocardium → has local anesthetic effect.
2. **CNS Effect:** Sedative action
3. **antihypertensive:** same as previously mentioned + inhibiting sympathetic outflow in CNS

**$\beta_1$-blocking Effect:**
1. Inhibit heart properties → ↓ cardiac output.
2. Anti-ischemic action
3. Antiarrhythmic effect

**$\beta_2$-blocking Effect:**
1. **Metabolism:**
   - In skeletal muscles: ↓ glycolysis, in liver: ↓ glycogenolysis & in pancreas: ↓ glucagon secretion.
   - Cause vasoconstriction + Bronchospasm + Increase Intestinal motility.

#### Indication
- Hypertension
- Arrhythmias
- Angina
- Myocardial infarction
- Migraine (Prophylaxis).
- Pheochromocytoma used with $\alpha$-blockers (never alone).
- Chronic glaucoma.
- Tremors.
- Anxiety (social & performance).
- Hyperthyroidism.

### Carvedilol
- Non-Selective $\alpha_1$ & $\beta$ blockers
- Without ISA
- Antioxidant action

#### Indication
Congestive heart failure reverses its pathophysiological changes.

#### ADR
- Orthostatic hypotension
- Edema

### Labetalol
- Non-Selective $\alpha_1$ & $\beta$ blockers
- Rapid acting
- With ISA = characterizes a group of beta blockers that are able to stimulate beta-adrenergic receptors (agonist effect) and to oppose the stimulating effects of catecholamines (antagonist effect) in a competitive way.
- Produce peripheral vasodilation
- Decrease blood pressure

#### Indication
- Severe hypertension in pheochromocytoma
- Hypertensive crisis (e.g. during abrupt withdrawal of clonidine).
- Used in pregnancy-induced hypertension

#### ADR
- Orthostatic hypotension (postural hypotension)
- Sedation & dizziness
<table>
<thead>
<tr>
<th>Drug</th>
<th>Act on ....</th>
<th>Uses</th>
</tr>
</thead>
</table>
| α-Methyl Dopa               | Neuron      | - hypertension in pregnancy  
                                           - pre-eclampsia  
                                           - gestational hypertension |
| Clonidine                   | α1 and α2 (non-selective) | -Management of withdrawal symptoms  
                                           - Little use as antihypertensive agent due rebound hypertension upon abrupt withdrawal |
| Apraclonidine               |             | Open angle glaucoma as eye drops                                      |
| Phenoxybenzamine and Phentolamine |             | Before removal of Pheochromocytoma to prevent Hypertensive Crisis.   |
| Prazosin, doxazosin and terazosin | α1 (Selective) | - Treatment of essential hypertension.  
                                           - Urinary obstruction associated with benign prostatic hyperplasia (BPH)  
                                           - Raynaud's disease. |
| Tamsulosin                  | α1A (more selective) | benign prostatic hypertrophy (BPH)                                    |
| Yohimbine                   | α2 (selective) | Used as aphrodisiac in the treatment of erectile dysfunction          |
| Propranolol                 | β1 and β2 (non-selective) | -Migraine prophylaxis  
                                           -Hyperthyroidism  
                                           - Social anxiety |
| Timolol                     |             | Glaucoma                                                             |
| Atenolol, Bisoprolol and Metoprolol | β1 | -Myocardial infarction  
                                           -Hypertension |
| Esmolol Ultra short acting  |             | Cardiac arrhythmia                                                   |
| Carvedilol                  | α and β     | Congestive heart failure                                             |
| Labetalol                   |             | -Hypertension in pregnancy  
                                           -Hypertensive emergency |
## MCQ

1. A new antihypertensive drug was tested in an animal model of hypertension. The drug when given alone reduces blood pressure in the animal. Norepinephrine when given in the presence of this drug did not cause any significant change in blood pressure or heart rate in the animal. The mechanism of action of the new drug is similar to which of the following agents?

   - A-Doxazosin  
   - B-Atenolol  
   - C-Carvedilol

2. A beta blocker was prescribed for hypertension in a patient with asthma. After a week of treatment, the asthma attacks got worse, and the patient was asked to stop taking the beta blocker. Which beta blocker would you suggest as an alternative that is less likely to worsen the asthma?

   - A-Metoprolol  
   - B-Propranolol  
   - C-Labetalol

3. A 70-year-old male is treated with doxazosin for overflow incontinence due to his enlarged prostate. He complains of dizzy spells while getting up from bed at night. Which drug would you suggest as an alternative that may not cause dizziness?

   - A-Propranolol  
   - B-Phentolamine  
   - C-Tamsulosin

4. Which of the following drugs is commonly used topically in the treatment of glaucoma?

   - A-Esmolol  
   - B-Timolol  
   - C-Yohimbine

5. Which of the following is correct regarding alpha adrenergic blockers?

   - A-Alpha adrenergic blockers are used in the treatment of hypotension in anaphylactic shock.  
   - B-Alpha adrenergic blockers may cause bradycardia.  
   - C-Alpha adrenergic blockers are used in the treatment of benign prostatic hyperplasia (BPH)

   1-C 2-A 3-C 4-B 5-C

## SAQ

1. A 50-year-old male was brought to the emergency room after being stung by a hornet. The patient was found to be in anaphylactic shock, and the medical team tried to reverse the bronchoconstriction and hypotension using epinephrine. However, the patient did not fully respond to the epinephrine treatment. The patient’s wife mentioned that he is taking a prescription medication of his blood pressure, the name of which she does not remember.

   Q1. Which of the adrenergic antagonist medications is he most likely taking that could have prevented the effects of epinephrine?

   Q2. What is the mechanism of action of that drug?

   Q3. A 70-year-old male needs to be treated with an α-blocker for overflow incontinence due to his enlarged prostate. Which drug would you suggest in this patent that will not affect his blood pressure significantly?

   Q4. A 32-year-old pregnant female was brought to the ER, after investigations she was diagnosed with Gestational Hypertension. What is the drug of choice of this case?

   Q5. A 82 year old man with history of Angina and hypertension presented to the ER with tachycardia. Which adrenergic antagonist drug should be prescribed to this patient?

   1-Propranolol  
   2-Non selective β1, β2 blocker  
   3-Tamsulosin  
   4- α-Methyl dopa  
   5-Doxazosin
GOOD LUCK

Team Leaders:
May Babaeer         Zyad Aldosari

Team Members:
Lama Alzamil
Reema Almutawa
Noura Almazrou
Njoud Almutairi
Shahad Alsahil
Shahad Bin Selayem
Jude Alkhalifah

Share with us your ideas!