L2

PHARMACOLOGY
TEAM 439

MED439
KING SAUD UNIVERSITY

Adrenergic Agonists

Objectives:

✓ classify adrenergic agonists according to chemical
✓ structure, receptor selectivity and mode of action.
✓ Discuss pharmacodynamic actions, ADRs, indications
and contraindication of adrenergic agonists.

Important
In male and female slides
Only in male slides
Only in female slides
Extra information

helpful video (epinephrine)

Adrenergic receptors

Editing file

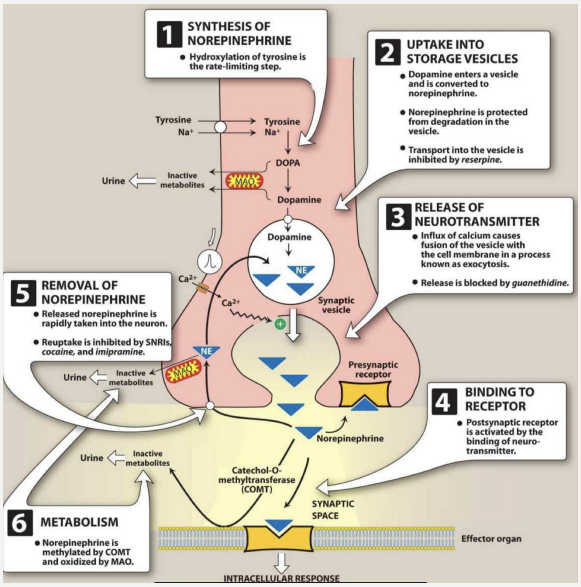
Adrenergic drugs are the drugs that work upon **sympathetic** nervous system, also called sympathomimetics.

The first 3 pages are very important for understanding the lecture,
they were only in girls' slides. However, boys must study them too.

Neurotransmission at adrenergic neurons

Adrenergic Transmission:

1- Synthesis of norepinephrine (by:hydroxylation)
2- Storage of norepinephrine
3- Release of norepinephrine
4- Binding to post synaptic receptors
5- Ending of action by:
   - Neuronal reuptake into neuron
   - Monoamine oxidase (MAO) in neuronal mitochondria
   - Catechol -O-methyl transferase (COMT) in synaptic space



الـTyrosine يتحول إلى DOPA. ← الـDOPA يتحول إلى Dopamine ← الـDopamine يتخزن في vesicles. ← جوا الـVesicles بيتحول الـDopamine الى Norepinephrine .
← يصير Action potential ويفرز الـNorepinipherine في الـSynaptic space.

طيب حلو الحين الـNorepinphrine بيحصل له واحد من ثلاث حاجات:
١- ممكن يطلع ويروح لـ systemic circulation
٢- ممكن يحصل له metabolism ويتكسر من قبل انزايم **COMT**.
٣-ممكن يحصل له reuptake ويرجع يدخل لـneuron مرة ثانية ويتأكسد من انزايم **MAO**.

الواضح أن COMT بيكون موجود في الـsynaptic space. / و MAO بيكون موجود جوا الـ neuron في الميتوكندريا.

Adrenergic Receptors

- **α** - adrenoreceptors
  - **α1**
  - **α2**
- **β** - adrenoreceptors
  - **β1**
  - **β2**
  - **β3**
- Dopaminergic Receptors
  - **E.g. D1**

- **α1 β1 β2 β3** are located postsynaptically. (At the organ)
- **α2 β2** are located presynaptically. (Before the organ)

If you memorize this very well, you will be able to guess the pharmacological actions just by knowing the receptor of the drug.

Post-synaptic (located in the tissue)

α1	β2	β1	β3
Excitatory function (they cause **contraction**) except in GIT (**inhibition**)	**Inhibitory in function** (cause relaxation) نفس مكان α1 لكن عكسها بالوظيفة	**Excitatory in function**	In adipose tissue
Present mainly in **smooth muscles**		Present mainly in heart, **juxtaglomerular cells** of the kidney (discussed later with renin)	↑ lipolysis ↑ free fatty acids
Contraction of uterus (helps with delivery) * راح أجهض طفلي الأول α1	Relaxation of the uterus (Delay premature labor) (The 2nd baby is coming.)*	(قلبي هو بيتك الأول) ↑ Heart rate: Chronotropic effect (Tachycardia) ↑ Force of contraction : inotropic effect (Contraction of ventricles & Increase cardiac output) ↑ Conduction velocity: dromotropic effect Pulse is conducted faster causing tachycardia ↑ Blood pressure ↑ Renin release Enzyme released by the juxtaglomerular cells of the kidneys in response to low blood pressure, causing the transformation of angiotensinogen to angiotensin I which in turn stimulates release of aldosterone **(Which Causes vasoconstriction & increases BP).** This is useful during hemorrhaging to conserve blood	
Vasoconstriction of skin & peripheral blood vessels →increased peripheral resistance (resistance to blood flow due to constriction of blood vessels)→ hypertension.	هذا بيتك الثاني ارتاح = relaxations Relaxation of skeletal & coronary blood vessels (vasodilatation)		
Relaxation Of GIT **muscles** (constipation) & Urinary bladder **muscles**. **Contraction** Of GIT **sphincter** & urinary bladder **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		
BOTH Increase blood glucose level (hyperglycemia) either by:			
↑ glycogenolysis So hyperglycemia	↑ glucagon release from pancreas ↑ liver & muscle glycogenolysis		

Pre-synaptic (Regulation of Noradrenaline release)

α2	β2
Inhibition of norepinephrine release (Neg**at**ive feedback mechanism) This feedback decreases NE when it's elevated **a2 =(at)wo**	Increase of norepinephrine release (**P**osi**t**ive Feedback mechanism) This feedback increases NE when its levels are low **b2=(PT))wo**

ADRENOCEPTORS

α1

Uterus Contraction

Vasoconstriction

Increased peripheral resistance

Increased BP (hypertension)

Relaxation of GIT muscles

Contraction of sphincters

Mydriasis

Hyperglycemia

β1

Tachycardia

inotropic effect

dromotropic effect

Increased BP (hypertension)

Increase renin release.

β2

Uterus Relaxation

Vasodilatation

Relaxation of GIT muscles

Contraction of sphincters

Bronchodilation

Hyperglycemia

Adrenergic Agonists "Sympathomimetics" actions:

- Mydriasis (dilatation of eye pupil) **α1**
- Increase heart rate. **β1**
- Bronchodilation **β2**
- Inhibit peristalsis of GIT and secretion. **α1 & β2**
- Relaxation of GIT muscles (constipation). **β2 α1**
- Relaxation of urinary bladder. **β2**
- Relaxation of the uterus (Delay premature labor) **β2**
- Increase conversion of glycogen to glucose (hyperglycemia) **α1 & β2**

Classification of Adrenergic Agonists

Chemistry | Mode of action | Spectrum of action

1- According to Chemistry

Catecholamines	Non-Catecholamines
Rapidly acting Have short half-life, Due to rapid degradation by MAO & COMTin GIT	Delayed action Have Long half-life, because they resist degradation by MOA & COMT in GIT
Have catechol ring, Water soluble (polar), thus not effective orally and have Poor penetration to CNS (Poor central action)	**Lack catechol ring** Lipid soluble , thus Effective orally and Cross BBB well , have Prominent CNS effects
Parenterally administered	**Orally** administered
Natural: **Adrenaline, Noradrenaline, Dopamine** Synthetic: Isoprenaline. *Adre Nora is Dopa =ادري نورا از دوبا	e.g. Ephedrine, amphetamine, phenylephrine.

2- According to Mode of Action

Direct-Acting	Indirect-Acting	Dual-Acting (Mixed)
Stimulate adrenergic receptors directly. **e.g. adrenaline,noradrenaline, dopamine, isoprenaline, phenylephrine, clonidine, dobutamine, salbutamol, methoxamine**	Stimulate adrenergic receptors by: ↑ noradrenaline release from presynaptic adrenergic nerve endings. The drug enters the vesicles that has noradrenaline & squeezes it out (release it) e.g. **amphetamine**, Tyramine Or Inhibit uptake of noradrenaline e.g. **Cocaine** & antidepressants	Direct and indirect stimulation of adrenergic receptors (mixed) e.g. ephedrine, pseudoephedrine

3- According to Spectrum of Action

Non-selective adrenergic agonist عنده Actions كثير فتصير أعراضه الجانبية أكثر	Selective adrenergic agonist:
Adrenaline (α1, α2 , β1 , β2, β3) Noradrenaline (α1, α2 , β1) *نورا وقفت عند قلبي (B1) Isoprenaline (β1, β2, β3) *isomers of peta receptor Dopamine (D1, β1, α1) Mainly D1 but affect others when increasing the dose. Ephedrine (α1, α2 , β1 , β2, and releasing agent)	Phenylephrine (α1) α-Methyldopa - clonidine (α2) Dobutamine (β1) *Do it but be Amine. والأمانة محلها القلب (B1) Salbutamol, terbutaline, ritodrine (β2)

Adrenaline = Epinephrine 0

Receptor		**Direct Action/ Non-selective α1 α2 β1 β2 β3**
Overview		**(Natural, Catecholamine) released from adrenal medulla secondary to stress, hunger, fear.** (Well, since it's a catecholamine then we know that it's polar and we shouldn't give it orally because it will be degraded by COMT/MAO etc..) Fast onset of action & Short duration of action.
Administration		Not effective orally (inactivated by intestinal enzymes). Given I.V, S.C, inhalation.
Actions		**Heart**: Inotropic,chronotropic (increase HR),dromotropic (β1) **Blood Pressure:**↑ **Systolic (β1) (α1)** (Prominent action)↓ **Diastolic (at low dose: β2, at a high dose α1** (Minorly) **Vascular:** - Vasoconstriction of blood vessels in skin + peripheral (α1) - Vasodilation of blood vessels of skeletal muscles and coronaries (β2) يقل الدم في الـskin والأطراف بسبب α1 لكن بالمقابل بيروح دم اكثر للـcoronaries والقلب بسبب β2 لأن الاحتياج أكبر هناك للأوكسجين. **Eye:** Mydriasis (α1) → no effect on accommodation **Lung**: Bronchodilation (β2) (because we need oxygen) **GIT:** ↓ motility (β2) / contract sphincter (α1) **Urinary bladder:** - Relaxation of detrusor muscle (β2) - Contraction of sphincter (α1) **CNS:** Little (rare), (Since it's a catecholamine, it has poor BBB penetration) headache, tremors & restlessness (usually in case of overdose) **Pregnant Uterus** Relaxation tocolytic effect (β2) Tocolytic action means relaxation of pregnant uterus **Metabolism:** ↓ insulin (α2), ↑ glucagon (β2) ↑ liver glycogenolysis +skeletal muscle glycolysis (β2) (Hyperglycemia) ↑ adipose lipolysis (β3)
Uses	Locally	➢ **Haemostatic (control bleeding) (by vasoconstriction):** (Stops it) Nasal packing epistaxis (nasal bleeding) & in dental practice., & as a decongestant (α1).. ➢ **Combined with local anesthetic to:** ↓ absorption of the local anesthetic and toxicity & ↑ duration of action Adrenaline causes vasoconstriction so there will be less blood flow to the area which will reduce the absorption of the local anesthetic. Therefore, the duration of the local anesthetic will be increased. امتصاص الدواء يكون بطيء ↓ side effects of local anesthetic. (Cause it's not absorbed quickly in a large amount so it will not produce prominent side effects) ↓ bleeding from the incision. (Vasoconstriction)
	Systemically	➢ **In acute asthma (status asthmaticus)** S.C., inhalation, emergency bronchodilation (β2) + ↓ mucosal edema **due to vasoconstriction (α1).** *Remember edema is caused by vasodilation due to inflammatory mediators ➢ **Anaphylactic shock (Hypersensitivity reactions)** is the drug of choice as it is the physiological antagonist of histamine (adrenaline will increase BP & bronchodilation). Hypersensitivity reactions are caused by histamine release which cause hypotension & bronchoconstriction so adrenaline is given to reverse these actions ➢ **Cardiac arrest** (i.v.). direct but now through central line N.B. Selective b1 agonists are preferred
ADR		- Tachycardia, palpitation, arrhythmias, angina pains(chest pain) (TAAP) - Headache, weakness, tremors, anxiety and restlessness. - Hypertension → cerebral hemorrhage and pulmonary edema. - Coldness of extremities (due to vasoconstriction) → tissue necrosis - Nasal stuffiness: rebound congestion if used as decongestant
Contradictions		- Coronary heart diseases (CHD), Ischemic heart disease: - Arrhythmia, Myocardial infarction - Hypertension, peripheral arterial disease (because they have low blood flow to extremities & adrenaline cause vasoconstriction) - Hyperthyroidism. (There is an increased amount of thyroxine hormone which can cause tachycardia) - Closed-angle glaucoma ciliary "iris" relaxation ↓ filtration angle) > ↑ □ IOP

Drug	Noradrenaline (Norepinephrine)	Isoprenaline
Classification	**Natural, Catecholamine** non-selective agonist It is naturally released from postganglionic adrenergic fibers. Not much used therapeutically because it causes severe vasoconstriction	**Synthetic direct acting catecholamine.** Shows no reuptake, nor does it show a breakdown by MAO (Which leads to longer action)
Administration	**ONLY administered by I.V** may cause necrosis using IM or SC (due to vasoconstriction)	**Parenteral** in cardiac arrest **inhalation** rarely in acute attack of asthma
Receptor	Mainly on α adrenoceptors **(α1, α2, β1, weak action on β2)**	Non-selective **β** agonist It Acts on **β1, β2, β3**
Pharmacological Action	- Severe vasoconstriction (α1) - Reflex bradycardia *due to severe Vasoconstriction.* (The body tries to regulate itself) - Increase force of contraction but decrease heart rate (**β1**) في البداية الـheart rate يزيد شوي بسبب زيادة الـ contraction بس في النهاية بيقل بسبب الـvasoconstriction	**β1 :** - + inotropic effect - + chronotropic effect - increase cardiac output **β2 :** - Vasodilation of blood vessels of skeletal muscles and coronaries - Bronchodilation - Relaxation of uterus - Hyperglycemia **β3 :** - lipolysis
Uses	**Locally:** As a local haemostatic with local anesthetic to reduce tachycardia & irritability, but as side effect, may produce necrosis & sloughing of the skin.	- Used mainly in cardiac arrest to increase heart rate (Parenteral) Better than adrenaline because it has less side effects - Rarely in acute attack of asthma (inhalation)
	Systemically: 1-hypotensive states : in **septic shock** (hypotension) if fluid replacement and inotropics fail. 2- Spinal anesthesia	**Contraindications**: In hyperthyroidism Thyroid gland over produces thyroxine which also increases cardiac output Congestive heart disease CHD

Drug	Dopamine	Dobutamine	Phenylephrine
Classification	**Natural catecholamine** & CNS neurotransmitter Direct acting Released from postganglionic adrenergic fibers (Renal vessels)	**Synthetic catecholamine** Direct acting Metabolized by COMT , thus has a short duration	**Synthetic NON catecholamine** Direct acting has prolonged duration of action since its not inactivated by COMT
Administration	Given **parenterally** by infusion	IV	Orally (Since it's not catecholamine)
Receptor	**D1 > β1 > α1** **(in order, depending on dose)**	**Selective β1–agonist**	**Selective α1**
Pharmacological Actions	**Low dose (Dopaminergic Receptor D1):** - **Vasodilatation** of mesenteric, coronary, renal blood vessels.(Thus improves blood flow to viscera.) + Decrease BP - Diuresis (increase excretion of urine due to vasodilation of renal blood vessels) **intermediate dose: (β1)** +ve inotropic (increased cardiac output) +ve chronotropic (increased heart rate) **high dose: (α1)** Vasoconstriction (↑BP)	**On heart:** +ve Inotropic with little chronotropic effect. as it increases cardiac output and heart contractility. **On BP:** Hardly any effect; β1 & β2 counterbalance + no α1	increased both systolic & diastolic blood pressure (hypertension) due to vasoconstriction **(α1)** **Reflex Bradycardia** **Adverse effects:** Hypertension. Thus, another drug is more preferable to produce hypertension that doesn't last for long. This drug is **Midodrine**. It peaks in 20 min, duration 30 min only. Midodrine: Selective α1, given orally
Uses	**Drug of choice in treatment of shocks**: **Septic, Hypovolemic** (after fluid replacement), **Cardiogenic** (I.V). It increases the **BP** & **CO** by **β1** receptor but **without causing renal impairment (D1)** because it increases CO and causing vasodilation (more blood goes to the kidney so it's not impaired) Can be given in acute heart failure (HF) **but Dobutamine is better** because it's selective and does not change o2 demand.	- Short term management of Cardiac decompensation after cardiac surgery - In acute myocardial infarction (AMI) & heart failure. It does not increase oxygen demand which made it preferred.	**Systemically:** - Vasopressor (anti-hypotensive) agent in hypotension & terminates atrial tachycardia by its reflex bradycardia action. **Topically:** **- Haemostatic** with Local anesthesia. **- Mydriatic** (in ophthalmic solutions to facilitate eye examination) Because it does not cause cycloplegia.(paralysis of ciliary muscles) - **Nasal decongestant** "Vasoconstriction" topically, nasal drops in allergic rhinitis, cold. (لانه بيقطع وصول الدم للخلية اللي تفرز فبينقطع الإخراج فيها) - Treatment of nasal stuffiness, But can cause a rebound.

Selective β2 agonist

*سالي راحت تلعب في بيت تيته(b2)

Drug	Salbutamol	Ritodrine	Terbutaline
Classification	**Selective β2–agonist** Synthetic, Non catecholamines Direct acting	**Selective β2–agonist** Non catecholamines	**Selective β2–agonist**
Administration	-Orally -Inhalation -injection. (preferred as inhalation for local effect on the respiratory system)	Orally injection	
Effect	Bronchodilation Little effect on heart (**β**1) (Because of its 4 hrs action, longer acting preparations exist ; Salmeterol & Formoterol)	Tocolytic drug (relaxation of uterus). Ritodrine= Tocolytic= Beta Two	Bronchodilator & Tocolytic
Uses	Acute attack of asthma & COPD.	Treat premature labor. (labour that begins before the 37th week of gestation)	

Selective α2 agonist

*أخذت توت(α2) وأكلته بالبيت

Drug	Clonidine	Brimonidine
Classification	Synthetic, Direct acting Selective **presynaptic** **α**2 agonist inhibits NE release	**Selective α2 agonist**
Administration	Orally or as patch.	
Action	- Acts centrally (**α**2) at nucleus tractus solitarius to ↓ sympathetic outflow to heart & vessels. -Inhibit sympathetic vasomotor centers. Clonidine stimulates the alpha 2 adrenergic receptors in the vasomotor center of the brain, leading to inhibition of activity of sympathetic nerves to the peripheral tissues	.
Uses	As antihypertensive in essential hypertension to lower BP. Essential hypertension = primary (unknown cause)	Glaucoma treatment (reduce aqueous humor production by the ciliary body) Note that drugs that treats glaucoma either reduce the production, or they increase the drainage of the aqueous humor

Adrenergic Stimulants

	Indirect Acting Sympathomimetics	Dual Acting Sympathomimetics	
Drug	**Amphetamine α & β**	**Ephedrine α & β**	**Pseudoephedrine**
Classification	Synthetic, Non-catecholamine.	Plant alkaloid, synthetic, Non-catecholamine	-
Administration	Given **orally** Since it's not a catecholamine, it is not destroyed by enzymes → Prolonged duration action		-
Excretion	Excreted mostly unchanged (excretion is increased by acidification of urine)	-	-
Action	Acts indirectly & Depletes vesicles from stored Norepinephrine → Tachyphylaxis (gradual decrease in the effect after a short period of time with repeated administration. Ex: the evening dose has less effect than the morning dose) While tolerance occurs after a few days. يخلي كل الـNorepinephrin اللي بالـvesicles يطلع, بحيث تصير كلها فاضية بعدين، ويصير تأثيره قوي مره على الـreceptor	1- **Direct** action on receptors 2- **Indirect** by releasing NE from adrenergic endings → depletes stores.	-
Effects	**-Has CNS stimulant effects:** Mental alertness, wakefulness, concentration & self-confidence followed by depression & fatigue on continued use. - Euphoria→ causes abuse - ↓ Weight by: ↓ appetite & ↑energy expenditure	**CNS stimulant effects** (less than amphetamine) Tachyphylaxis Facilitation of neuromuscular transmission (myasthenia gravis) & retention of urine.	Vasoconstriction of blood vessels, mainly those located in the nasal passages causing a decrease in symptoms of nasal congestion. (نفس الـ Phenylephrine)
Uses	No more used therapeutically as it induces psychic and physical dependence and psychosis.	No more therapeutically used but is abused by athletes and prohibited during games.	Used as nasal & ocular decongestant & in flu remedies.

Dr's Summary

الترتيب بأولوية الإستخدام

Hypotension

Midodrine,
Phenylephrine,
Norepinephrine,
Phenylpropanol

Cardiogenic shock & acute heart failure

Dobutamine,
Dopamine,
Epinephrine

Shock

Dopamine,
Norepinephrine

Cardiac Arrest

Dobutamine,
Epinephrine,
Norepinephrine

Bronchial asthma

Salbutamol,
Salmeterol,
Formoterol,
Terbutaline,
Isoprenaline

Premature labor

Ritodrine,
Terbutaline

Nasal decongestant

Pseudoephedrine,
Phenylephrine

Sports
Illegal use

Ephedrine,
Amphetamine

SUMMARY

Drug	Receptor	Uses
Adrenaline	**α1, α2, β1, β2, β3**	- Combined with local anesthetic - Haemostatic (Stops bleeding) - In acute asthma - Anaphylactic shock - Cardiac arrest
Noradrenaline	**α1, α2, β1, weak β2**	- local haemostatic with local anesthetic to reduce tachycardia. - in septic shock (after fluid replacement)
Isoprenaline	**β1, β2, β3**	- cardiac arrest (preferred) - Rarely in acute attack of asthma
Dopamine	**D1 > β1 > α1 (in order)**	**- Treatment of shocks**: **septic, Hypovolemic** (after fluid replacement), **cardiogenic** (I.V)
Dobutamine	**β1**	- Short term management of Cardiac decompensation. - Acute myocardial infarction (AMI) & heart failure.
Phenylephrine	**α1**	- Vasopressor (anti-hypotensive) **- Haemostatic** with Local anesthesia. - **Mydriatic** **- Nasal decongestant**
Salbutamol	**β2**	- Acute attack of asthma & COPD.
Ritodrine	**β2**	- Treat premature labor.
Terbutaline	**β2**	-
Clonidine	**α2**	- Antihypertensive
Brimonidine	**α2**	- Glaucoma treatment
Amphetamine	**-**	No more used therapeutically
Ephedrine	**-**	No more therapeutically used but is abused by athletes
Pseudoephedrine	**-**	- Nasal & ocular decongestant - In flu remedies.

MCQs

1- Which of the following is correct regarding responses mediated by adrenergic receptors?			
A- Stimulation of α1 receptors increases blood pressure.	B- Stimulation of sympathetic presynaptic α2 receptors increases norepinephrine release.	C- Stimulation of β2 receptors increases heart rate (tachycardia).	D- Stimulation of β 2 receptors causes bronchoconstriction.

2- 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

3-An elderly patient is brought to the emergency room with a blood pressure of 76/60 mm Hg, tachycardia, and low cardiac output. He is diagnosed with acute heart failure. Which of the following drugs is most appropriate to improve his cardiac function?			
A-Epinephrine	B-Fenoldopam	C-Dobutamine	D-Isoproterenol

4-As a cardiovascular drug, dopamine can produce different effects depending on the dose administered. At high doses the predominant effect of dopamine would be to cause vasoconstriction by stimulating?			
A- Alpha 1 receptor	B-Beta 1 receptor	C-Beta 2 receptor	D-Dopaminergic Receptor

5-A drug that produces a pressor response is administered repeatedly by i.v. injection over a short period of time. After several injections, a tachyphylaxis occurs. What drug was most likely administered?			
A-ephedrine	B-epinephrine	C-norepinephrine	D-phenylephrine

6- The drug to treat 49 years old patient brought to the emergency room having a cardiac arrest. Her husband reported that she's on methimazole, an anti thyroid medication.			
A-dobutamin	B- Isoprenaline	C- Phenylephrine	D-Ephedrine

Answers

1	2	3	4	5	6
A	D	C	A	A	A

SAQ

Q1) During a prolonged surgical procedure performed under halothane general anesthesia, a patient experiences a fall in blood pressure. The anesthesiologist administers a drug that avoids kidney impairment. What is the drug? How does that happen? Mention another use.

Dopamine. It increases the BP & CO by β1 receptor but without causing renal impairment (D1). It is also the Drug of choice in treatment of shocks: Septic, Hypovolemic, and Cardiogenic.

Q2) Enumerate the uses of adrenaline

- Combined with local anesthetic
- Haemostatic (Stops bleeding)
- In acute asthma
- Anaphylactic shock
- Cardiac arrest

Q3) Dopamine has different effects depending on the dose. Name each receptor for each dose and mention its pharmacological effect.

Low dose (Dopaminergic Receptor D1):
Vasodilatation of mesenteric, coronary, renal blood vessels.
Diuresis
intermediate dose: (β1)
+ve inotropic effect
+ve chronotropic effect
high dose: (α1)
Vasoconstriction

GOOD LUCK!

Team Leaders

Tarfa Alsharidi

Khaled Alsubaie

Revised by

Dana Naibulharam

Bandar Alharbi

Team Members

Ghada Alothman
Ghadah Alsuwailem
Rawan Bagader
Noura Bamarei
Sadem Alzayed
Yasmin Alqarni
Ghaida Almarshoud
Shayma Alghanoum
Leen Almadhyani
Noura Alsalem
Noura alsalem
May barakah
Banan Alqady
Reem Aldossari
Nouf Alsubaie
Duaa Alhumoudi

Mohamed Aquhidan
Meshal Alhamed
Abdulaziz Alsalem
Feras Alqaidi
Musab Alamri
Mohammed Alkathiri
Abdullah Alburikan
Mohammed Al-Shamrani
Abdulrhman Alsuhaibany

Any suggestions or Complaints :

TeamPharma439@gmail.com

Pharmacology439