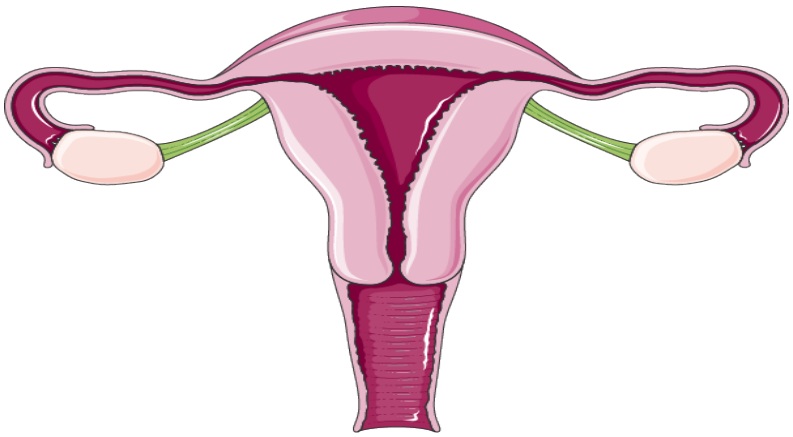
MEDICINE
KING SAUD UNIVERSITY

436 team
pharmacology



1: Drug Affecting Erectile Dysfunction

Objectives

1. Revise the haemodynamic changes inducing normal erection
2. Interpret its different molecular control mechanisms
3. Define erectile dysfunction [ED] and enumerate its varied risks
4. List drugs inducing ED and reflect on some underlying mechanisms
5. Correlate drugs used in treatment of ED to the etiopathogenesis
6. Classify oral 1st line therapy relevent to; Mechanism / Utility / ADRs
7. Compare the pharmacological difference of PDE_5 inhibitors
8. Study the transurethral, intracavernous or topical 2nd line therapies; Mechanism / Utility / ADRs
9. Enumerate lines of treatment of priapism

Color index

- **Doctors' notes**
- **Drugs names**
- **Extra information and further explanation**
- **Important**
- **Mnemonics**

تمت المراجعة
شوق الأحمري & عبدالعزيز العنقري

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Recall

Adrenergic receptors

Receptor	Location	Effect
α_1	Post synaptic	On blood vessels: vasoconstriction
α_2	Presynaptic	Inhibition of transmitters release
β_2	Post synaptic	On blood vessels: vasodilatation

هذي المحاضرة راح نتكلم كثير عن الأوعية الدموية وتأثير هذي المستقبلات عليها:

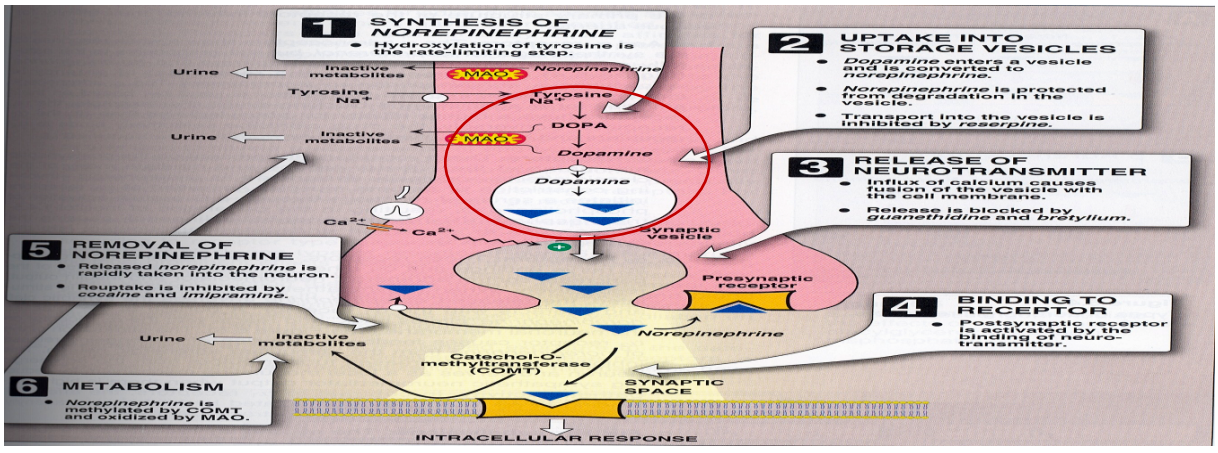
1. بالنسبة لمستقبلات α_1 هذي راح يسوون عندي انقباض للأوعية الدموية، فلما نستخدم أدوية تسكر هذي المستقبلات معناته راح يصير عندي أيش؟ بالضضضبط ارتخاء للأوعية الدموية
2. مستقبلات α_2 موجودة على الخلايا العصبية، هي راح تمنع إفراز الناقلات العصبية (زي نورابينيفرين والدوبامين) فبالتالي بتقلل عندي arousal، طيب لو جبنا أدوية تسكرها أيش راح يصير عندي؟ زيادة إفراز الناقلات العصبية بالتالي arousal
3. مستقبلات β_2 وهم موجودين على أعضاء كثير منها الأوعية الدموية، وظيفتهم يسوون ارتخاء للأوعية الدموية، إذا سكرنا هذي المستقبلات أيش راح يصير؟ انقباض للأوعية الدموية

Methyl-dopa

Mechanism of action:

1. Decrease dopamine release by forming methyl dopa
2. Acts centrally as α2 receptor agonist to inhibit NE release

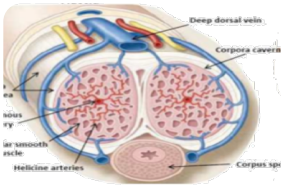
في خلال تصنيع الدوبامين المفترض بعدين يتحول إلى نورابينيفرين، هذا الدوا يحول لي الدوبامين إلى ميثايل دوبا بالتالي يقل عندي الدوبامين و نورابينيفرين



Pathophysiology of Erection*

Stages

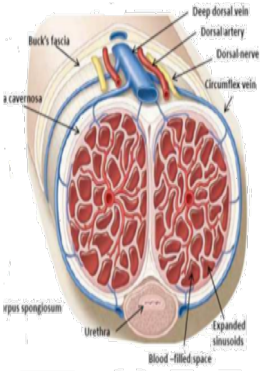
When does erection occur? it occurs when the **amount of blood rushing to the penis is greater than the amount of blood flowing from it**



1- FLACCID State

(blood inflow = venous outflow)

2- ERECT State



A massive influx of blood accumulates in the sinusoidal spaces due to **relaxation of smooth muscle & dilatation of arteries** → corpora cavernosa to swell **(tumescence)**

(blood inflow > venous outflow) (explanation : there is a deep artery in the middle of each carpora cavernosa , when parasympathetic works, this artery gets dilated and the fill the whole carpora cavernosa sinuses with blood → erection of the penis → dilation of carpora cavernosum → compress the vein on top of it → decrease out flow)

Tumescence compresses the veins that normally drain the penis → reduces **venous outflow** & maintains penile rigidity

A normal erection relies on the coordination:

- Vascular (blood vessels)
- Neurological
- Hormonal
- Psychological (self psychosis)

Stimulation

An erection can occur following direct*:

- Genital stimulation
- Auditory or visual stimulation
- Aspects that contribute to the influx of blood to the penis

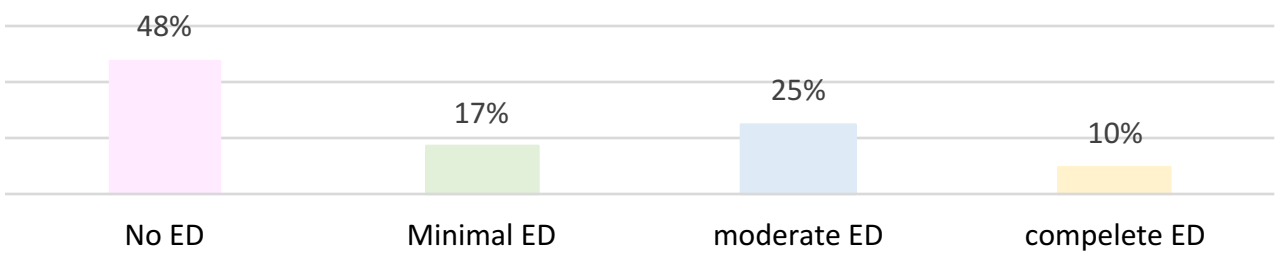
*خلونا نتفق أول إن erection يصير عن طريق تحفيز الماغ "arousal" عن طريق محفز سمعي أو نظري فيفرز الدوبامين ويتحفز الدماغ ويروح يرسل إشارات عن طريق parasympathetic للأعصاب اللي تغذي penis، هذي الإشارات راح تسبب زيادة تدفق الدم لpenis، ليش؟ لأن parasympathetic يسوي انبساط للشرايين، يعني عشان يصير erection نحتاج نسوي **vasodilatation** بشكل عام نقدر نسوي vasodilatation عن طريق: ١- إغلاق مستقبلات الفا١ الموجودة في عضلات الشرايين الملساء أو ٢- عن طريق إغلاق باثواي راح نشرحه في السلايدات الجايه ☺

Erectile Dysfunction

Erectile dysfunction: Persistent or recurrent inability to attain (acquire) & maintain (sustain) an erection (rigidity) sufficient for satisfactory sexual performance

Impotent

Definition: is reserved for those men who experience erectile failure during attempted intercourse more than 75 % of the time.
Prevalence: Endothelial Dysfunction → Commonest Cause (cases of Endothelial Dysfunction)



Other causes: collected in monic IMPOTENTCE

اللي بالأحمر مهمة

Inflammatory	Prostatitis, urethritis
Mechanical	Peronei's disease, chordee
Psychological	Depression, performance anxiety, stress, relationship difficulties
Occlusive vascular	Arterial: hypertension, smoking, hyperlipidemia hyperlipidemia (lead to atherosclerosis), DM, peripheral vascular disease (Oxidative stress →endothelium damage → lose the vasodilator) Venous: venous occlusion due to anatomical or degenerative changes
Trauma	Pelvic fracture, SC injection, penile truma
Endocrine	Hypogonadism, hyperprolactinemia, hypothyroidism, hyperthyroidism
Neurologic	Parkinsons, multiple sclerosis, spina bifida, pelvic surgery, peripheral neuropathy
Chemical	Anti-HTN, anti-arrhythmics, antidepressant, anxiolytics, anti-androgens, anticonvulsants, alcohol, marijuana, anti-parkoson drugs, LHRH analogues
Extra factors	Prostatectomy (may damage the nerve), old age (hormonal embalance) , CRF, cirrhosis

Drugs Adversely Causing ED

Drug Class	Specific drug examples دي بس أقروها
Beta-blockers Calcium-channel blockers Alpha-adrenergic agonists Cardiac glycosides	propranolol, metoprolol, atenolol verapamil, nifedipine clonidine digoxin
Thiazide diuretics Aldosterone antagonists	hydrochlorothiazide spironolactone
Fibric acid derivatives	gemfibrozil, clofibrate
Selective serotonin reuptake inhibitors Tricyclic antidepressants Other antidepressants	fluoxetine, sertraline, paroxetine, citalopram amitriptyline, desipramine, nortiptyline lithium
Benzodiazepines	lorazepam, alprazolam, diazepam
Histamine (H_2) receptor antagonists	ranitidine, cimetidine
Butyrophenones and phenothiazines	haloperidol, prochlorperazine, chlorpromazine
Hydantoin anticonvulsants	phenytoin
Cytotoxic agents	cyclophosphamide, methotrexate
Recreational drugs	alcohol, cocaine, marijuana

Central acting drugs	
Main action	Dopamine more than epinephrine promote arousal. 5HT (serotonin) action on $5HT_2$ → decrease dopamine release → decrease arousal (there are some neurotransmitters affect the Arousal, one of them is Dopamine, so any agonist of Dopamine will increase Arousal & any antagonist of Dopamine will decrease it).
anti-Depressant Drugs	e.g. non-selectively as TCAs, selectively as SSRIs ADD (anti-Depressant Drugs) decrease 5HT uptake which lead to ↑ 5HT in synapse act on $5HT_2$ → decrease dopamine release → decrease arousal Peripheral effect: antagonize NO actions / decrease genital sensation → Delay ejaculation (use for Treat of Premature Ejaculation)(SSRI)
Anti-psychotic drugs	DA (dopamine) antagonist + hyperprolactinemia
Anti-epileptic drug	e.g. phenytoin, they have GABA effect (inhibitory neurotransmitter) → antagonize excitatory Amino acid. → increase sedation → decrease arousal.

SSRI and other Anti-depressant drugs are the most common class of drugs which cause ED in male.

Anti-hypertensive

Anti-hypertensive	
Central hypotensive	Methyl-dopa, Reserpine: they decrease DA by depleting dopamine → decrease arousal Clonidine (α_2 agonist): decrease arousal centrally, Vasoconstriction peripherally by anticholinergic action (blocking alpha receptors) → ED
Other hypotensive	• β_2 blockers: -ve vasodilating β_2 + potentiate α_1 effect (vasoconstriction) • Thiazide diuretics: decrease spinal reflex controlling erection + decrease arousal

Anti-androgen (Decrease the Desire)

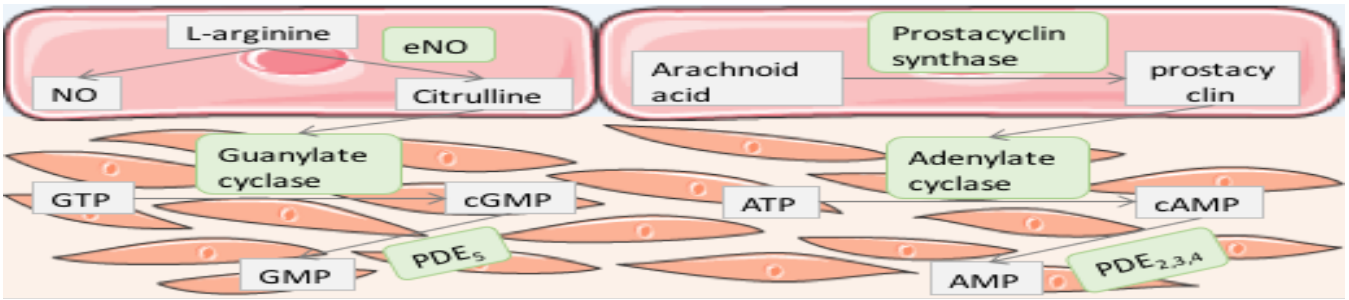
Anti-androgen	
Drug	Finasteride: (used in Benign prostatic hyperplasia , male androgenic alopecia (during first puberty they have a lot of testosterone lead to acne and hair loss → α reductase inhibitor (prevent production of active testosterone → irreversible erectile dysfunction وصلنا نهاية (Finasteride=Final=end) الطريق خلاص ما فيه رجعه منه (irreversible)
Drug	Cyproterone acetate: synthetic steroidal antiandrogen
Drug	Cimetidine* (H_2 blocker used in peptic ulcer), Ketoconazole, Spironolactone: (used in HF by working on angiotensin system) but it cause hyperprolactinemia + gynecomastia * Long acting
Drug	Estrogen-containing medications

Habituating Agent

Habituating agent	
Smoking	Cigarette smoking cause vasoconstriction + penile venous leakage (blood flow to penis will not be maintained due to vasoconstriction and wall damaged → coordination is disrupted blood in <or = out flow)
Alcohol	Small amount: increase desire + decrease anxiety + vasodilatation Big amount: increase sedation + decrease desire Chronic alcoholism: hypogonadism + polyneuropathy

Drugs Treat ED

- **Peripheral**
  - selective PDE_5 inhibitors
    - Sildenafil
    - Vardenafil
    - Tadalafil
    - Avanafil
  - Oral
    - Phentolamine
    - Yohimbine
    - Trazodone
    - Korean Ginseng
  - transurethral[1]
    - Alprostadil
  - Topical[1]
    - Papaverine
    - Minoxidil
    - Nitroglycerine
  - Intracavernosa[1]
    - Alprostadil
    - Papaverine
    - Phentolamine
- **Central**
  - Androgens (Testosterone) ↑ desire
  - Apomorphine (dopamine agonist) (↑ arousal)



المقصود من الصورة إن عندنا مادتين يطلعون من الإندوثيليم حقت الشرايين وهم: (١) **NO** (٢) prostacyclin و هم يطلعون خلال **sexual activity**، بيروحون للعضلات الملساء ويحفزون أنزيمات وهم: (امشوا مع الصورة عشان يسهل عليكم الفهم)

أ- **NO يحفز انزيم اسمه GC اللي بيعطيني cGMP هذا لحاله بسوي vasodilation بس يجي بعدين انزيم ثاني يكسره اسمه PDE_5 وبيحول cGMP إلى GMP**

ب- prostacyclin راح يحفز انزيم اسمه AC "تونا دارسينه في الإندوكراين ياويلكم لو نسيتوه ☺ " هذا الأنزيم يعطيني cAMP اللي بيسبب **vasodilation** بعدين انزيم ثاني يكسره اسمه $PDE_{2,3,4}$ بيحول cAMP إلى AMP

بالمختصر: عشان أسوي ارتخاء للعضلة أحتاج cAMP or cGMP و هم بعدين بيتكسرون ويتحولون إلى GMP و AMP طيب المشكلة عندنا في هذي المحاضرة إن العضلة منقبضة فما فيه دم يوصل لذلك نحتاج نسوي **vasodilation**[2] عشان يوصل الدم بكمية كبيرة، لذلك أبغى أزود شغل الأنزيم (AC+ **GC**) اللي يعطيني المادة المسببة لارتخاء العضلة (cGMP + cAMP) أو بنمنع الأنزيمات اللي تكسر هذي المواد .

[1] These way are more favorable to decrease the affect of systemic affect (e.g. lead to sever hypotension → angina)

[2] So vasodilatation mechanism is so important in sexual life and the way to maintain it by keep exercising to increase blood flow to all of your body even it help to improve memory

Selective PDE_5 Inhibitors

Nafil = Number five

	Sildenafil	Vardenafil	Tadalafil	Avanafil

M.O.A

- Inhibit PDE_5 → prevent breakdown of cGMP → pertain vasodilatation → erection.
- They do not affect the libido, so sexual stimulation is essential to a successful (this drug prevent PDE_5 from degenerate cGMP, but we need NO to produce cGMP, and NO excreted during sexual stimulation)

P.D

Pharmacodynamics action relevant to PDE_5 inhibition:

- Vascular smooth muscle cells (VSMCs) of Erectile Tissue of Penis
- Other vascular smooth muscle cells (VSMCs) (lung, brain….) / heart (so we use them in pulmonary hypertension)
- Other non-VSMCs (prostate, bladder, seminal vesicle, GIT….)
- Platelets (so we can use them to treat benign prostate hyperplasia)
- Other tissues; testis, sk. muscles, liver, kidney, pancreas, …..
- **All types have similar efficacy**: Sildenafil: 74-84% , Vardenafil: 73-83% , Tadalafil: 72-81%

Uses

- Erectile dysfunction; **1st line therapy.**
- Pulmonary hypertension (they cause smooth muscle relaxation,
- BPH & premature ejaculation (SSRI as well) (rare)

Selectivity

10-fold selective	16-fold selective	>200-fold selective

Selectivity on PDE_5 is not absolute and vary with each drug:

- Can partially act on PDE targeting cGMP (6, 11, 9, 1)
- In higher doses it can act on PDE targeting cAMP (2,3,4, 10,…)

❖ **PDE types and locations:**

- PDE1: Heart, brain, lung, smooth muscle (main ADRs: IH3D / AMI)
- PDE2: Adrenal gland, heart, lung, liver, platelets (main ADRs: IH3D / AMI)
- PDE3: Heart, lung, liver, platelets, adipose tissue, inflammatory cells (main ADRs: IH3D / AMI)
- PDE4: Sertoli cells, kidney, brain, liver, lung, inflammatory cells ((main ADRs: IH3D / AMI)
- PDE5: Lung, platelets, vascular smooth muscle, heart. (Main ADRs: Headache/Flush nasal congestion)
- PDE6: Photoreceptor (main ADR: Altered VISION)
- PDE7: Skeletal muscle, heart, kidney, brain, pancreas, T lymphocytes
- PDE8: Testes, eye, liver, skeletal muscle, heart, kidney, ovary, brain, T lymphocyte
- PDE9: Kidney, liver, lung, brain, possibly heart
- PDE10: Testes, brain
- PDE11: Skeletal muscle, prostate, kidney, liver, pituitary gland and salivary glands, testes (main ADR: Back Pain)

المقصود هنا إن الأودية هذي مب بس تأثر على انزيم PDE_5 إنما عندها تأثير على انزيمات ثانية، إذا أخذناها بالجرعة المعتادة راح تأثر على اللي لونهم سماوي (كونها بتأثر عليهم يعني بتعطي تأثير على الأعضاء الموجودة فيهم بالتالي بتعطيني آثار جانبية) أما إذا أخذناها بجرعة عالية راح تأثر على كل الأنزيمات اللي بالوردي والسماوي
لا تحفظون شي من الأنزيمات غير اللي محدد بالعنابي

ADRs of PDE_5 Inhibitors

Major common ADRs:

	Sildenafil	Vardenafil	Tadalafil
 Headache	14	10	15
	Most of them have these because they are all vasodilation		
 Flushing	12	11	3
 Nasal	Congestion	Rhinitis	Congestion
 Dyspepsia	7	3	15
 Abnormal vision	4 Bc It acts on PDE_6: Photoreceptor. السلايدز Sildenafil الزرقاء دايم تعمي العين	< 2	
 Myalgia & Back pain	-	-	5 Tadalafil تدل الطريق ؟ تكسر ظهري ورجلي وأنا أدور
 Sperm functions	-	-	Less than (?) Tadalafil; Adal is infertile
 Q-T prolongation	-	Increase Vardenafil يوم شفت الفأر وقف قلبي من الخوف	-

Major less common:

1. IHD & AMI: patients on big dose or on nirates
2. Hypotension: patients on α-blockers than other antihypertensives (as nitrate is contraindication)
3. Bleeding: epistaxsis (bleeding from nose # in blood disorder).....etc.
4. Priapism: if erection lasts longer than 4 hours → emergency situation

Major rare ADRs:

1. Ischemic Optic Neuropathy: can cause sudden loss of vision or herring loss
2. Hearing loss

With high selective PDE_5 the adverse effects is less (ADRs depend on the selectivity)

Selective PDE_4 Inhibitors

	Sildenafil	Vardenafil	Tadalafil	Avanafil
Absorption	Fatty food interferes with Sildenafil & Vardenafil absorption, so taken on empty stomach at least 2 hours after food		Tadalafil & Avanafil are not affected by food	
Interactions	All by hepatic CYT3A4; Tadalafil > the rest, thus: ✓ Increase ADRs with enzyme inhibitors; erythromycin & clarithromycin, ketoconazole, cimetidine, tacrolimus, fluvoxamine, amiodarone…etc. ✓ Decrease efficacy with enzyme inducers; rifampicin, carbamazepine, phenytoin			
Dose	50 - 100	10 - 20	10 - 20	
Time of admin.	1 hour before intercourse	1 hour before intercourse	• 1-12 hours before intercourse • Must be given every 72 hrs if used with **enzyme inhibitors**	Has the advantage of been given 30 min before intercourse
Onset	30 - 60 min	30 - 60 min	< 30 - 45 min	
Duration	4 (shortest)	4 - 5	36 Usually if the drug with long duration, that's mean used for pulmonary hypertension or BPH	
C.I	• Nitrates: total contraindication (the less selective PDE_5 may Induce hypotension so we need to know if the patient take antihypertensive drugs as nitrate because if the patient take the two medication he will develop sever hypotension and it's emergency station and enter ICU also may lead to the death of the patient). • Hypersensitivity to drug • Patients with history of AMI, stroke, fatal arrhythmias <6 month			
Precautions	• With α blockers (except tamsulosin), bc they cause orthostatic hypotension • With hepato/renal insufficiency • With bleeding tendencies (leukemia's, hemophilia, Vit K deficiency, antiphospholipid syndrome,…etc) • **With quinidine, procainamide, amiodarone (class I & III antiarrhythmic) especially with (Vardenafil) because it cause prolong QT interval** • Dose adjustment; when using drugs that have interaction on hepatic liver microsomal enzymes i.e. inhibitors or inducers. • Retinitis pigmentosa			

Oral Drugs to Treat ED

Testosterone (Androgens)	• Given to those with hypogonadism or hyperprolactinemia. (when prolactin increase in male will contribute with receptors in gonads and inhibit testosterone synthesis). • Given for promotion of **desire** الرغبة. أنا آدري إنك تبي(ترغب)
Apomorphine Central acting → increase Arousal	• A dopamine agonist on D_2 receptors. • Activates **arousal** الإثارة centrally; Erectogenic + Little promotion of desire. أبو المورفين مسوي فيها إثارة • Given sublingual, so Acts quickly. • Not FDA approved, Weaker than PDE_5 Is • Given in mild-moderate cases, psychogenic, PDE_5 Is contraindication • ADRs: nausea, headache, and dizziness **but safe with nitrate**
Oral Phentolamine	• α_1 blocker / debatable efficacy
Yohimbine	• Central and peripheral presynaptic α_2 agonist (vasoconstrictor) (Aphrodetic + Erectogenic) but low efficacy and many CV side effects, such as hypertension. (not used any more)
Trazodone	• Antidepressant, a 5HT reuptake inhibitor which cause priapism. In beginning it was used to treat premature ejaculation but later was noticed that may lead to priapism
Korean Ginseng	• Questionable / may be a NO donner.

Topical Drugs to Treat ED

Combination:

- 20% Papaverine (Direct smooth muscle relaxant): increase cAMP + cGMP
- 2% Minoxidil (antihypertensive): NO donner + K channel opener
- 2% Nitroglycerine: a drug absorption enhancers

Disadvantage:

- Low efficacy / No FDA approval
- Female Partner can develop hypotension, headache, due to vaginal absorption.

Transurethral Drugs to Treat ED

Alprostadil	
M.O.A	PGE1 → increase cAMP (PGE1 increase AC activity → more cAMP → vasodilatation)
P.K	• Synthetic + more stable • Applied by a special applicator into penile urethra & acts on corpora cavernousa which lead to Erection • Low - Intermediate Efficacy • Minimal systemic effects / Rarity of drug interactions. (Advantages upon $PDEI_5$)
ADRs	• Variable penile pain urethral bleeding, urethral tract infection • Vasovagal reflex, Hypotension • Priapism or Fibrosis (rare)

Intracavernosal Drugs to Treat ED

Alprostadil	• Needs training: Erection → after 5-15 min • Lasts according to dose injected • May develop fear of self injury, so Discontinuation • ADRs: Pain or bleeding at injection site, cavernosal fibrosis, Priapism
Papaverine	• PGE1 → increase cAMP
Phentolamine	• α_1 blocker

3 combined in severe cases

First line of treatment is PDE_5I , if there is no effect we can start multi-treatment such as transurethral Alprostadil. If it is sever we can give them the injection form.

Treatment of Priapism

Priapism: A medical emergency (if erection lasts longer than 4 hours)

Treatment:

I. Aspirate blood to decrease intracavernous pressure.

II. Intracavernous injection of Phenylephrine (local α_1 agonist) ⟶ Detumescence

Summary

Drugs adversly causing ED	
Centrally Acting Drugs	Antidepressant drugs, SSRI (Treat premature ejaculation.), Anti-psychotic drugs and Anti-epileptic drugs (phenytoin).
antihypertensives	• Centrally acting antihypertensives: Methyldopa, Reserpine, Clonidine • Other antihypertensives: β2 blockers and Thiazide diuretics
Antiandrogens leads to ↓ desire	• Finasteride: α reductase inhibitor (cause irreversible erectile dysfunction) • Cyproterone acetate • Cimetidine / Ketoconazole /Spironolactone: leads to hyperprolactinemia & gynecomastia .
Habituating Agents	e.g. Cigarette smoking and alcohol intake.

Drugs used in treatment of ED

Selective PDE5 Inhibitor.

Sildenafil, Vardenafil, Tadalafil, Avanafil

- Inhibit PDE5 which prevent breakdown of cGMP.
- They don't affect libido (don't produce NO)
- It target multiple tissues in the body
- Used as treatment in Erectile dysfunction, Pulmonary hypertension and BPH

ADRs:

- Headache, flushing, nasal irritation
- Abnormal vision, more with Sildenafil
- Myalgia, back pain and decreased sperm function with Tadalafil
- QT prolongation, prolonged with vardenafil
- IHD and AMI, Bleeding; epistaxis, Priapism.
- Ischemic Optic Neuropathy, hearing loss (both rare)

C.I:

- Patient using nitrate
- Hypersensitivity to drug.
- Patients with history of AMI, stroke or fatal arrhythmias

Precautions:

With alpha blocker, hepato/renal insufficiency.
Retinitis pigmentosa.
With Quinidine, procainamide, amiodarone
Dose adjustment when using drugs that have interaction on hepatic liver microsomal enzymes

oral drugs

Testosterone: given to those with hypogonadism or hyperprolactinemia for promotion of desire.
Apomorphine: A dopamine agonist on D2 receptors, safe with nitrate. Given if PDE5 is contraindicated.
ADRs: Nausea, headache, and dizziness
Oral phentolamine: α1 blocker / debatable efficacy.

❖ Trans-urethraloral (Alprostadil): Prostaglandin E1 analogue

ADRs: Variable penile pain Urethral bleeding, infection.
Vasovagal reflex, Hypotension and priapism.

❖ Intra-cavernosal (Alprostadil, Papaverine, Phentolamine)

❖ Topical (20% Papaverine, 2% Minoxidil, 2% Nitroglycerine) Female Partner can develop: Hypotension, headache because of vaginal absorption.

Treatment of Priapism:

- Aspirate blood to decrease intracavernous pressure
- Intracavernous injection of Phenylephrine (α_1 agonist)

MCQs

Q1: Which of the following statements is CORRECT regarding the mechanism of action of phosphodiesterase-5 (PDE-5) inhibitors?

A. PDE-5 inhibitors increase prostaglandin production.
B. PDE-5 inhibitors enhance the effect of nitric oxide.
C. PDE-5 inhibitors cause vasoconstriction of the erection chamber.
D. PDE-5 inhibitors antagonize cyclic GMP.

Q2: A patient who is taking a PDE-5 inhibitors for ED, is diagnosed with angina. Which of the following antianginal medications would be of particular concern in this patient?

A. Metoprolol. B. Diltiazem. C. Amlodipine. D. Nitroglycerin.

Q3: Which of the following BEST describes the mechanism of action of alprostadil?

A. Alprostadil blocks cGMP.
B. Alprostadil blocks nitric oxide.
C. Alprostadil increases PDE-5.
D. Alprostadil increases cAMP.

Q4: Alprostadil is administered locally. Which of the following is CORRECT regarding local administration of alprostadil? *

A. Local administration of alprostadil allows for low systemic absorption.
B. Local administration of alprostadil increases the chance of drug interactions.
C. Local administration of alprostadil is accomplished by way of a cream.
D. Local administration of alprostadil causes changes in color vision.

Q5:Which of the following is CORRECT regarding finasteride ?

A. α reductase inhibitor.
B. cause irreversible erectile dysfunction.
C. Decrease the desire.
D. All of them.

Q6: 39 years old male who has erectile dysfunction, his doctor prescribe to him Viagra. Which of the following is incorrect about Viagra (Sildenafil) ?**

A. They block the conversion of cGMP into GMP.
B. They inhibit PDE-5.
C. Pulmonary hypertension
D. They affect libido.

Q7: Which one of the following PDE-5 inhibitors may lead to prolonged QT intervals ?

A. Sildenafil. B. Vardenafil. C. Tadalafil. D. Avanafil.

* Local administration of alprostadil allows for minimal systemic absorption. This makes alprostadil associated with few drug interactions. Alprostadil is administered by injection or urethral suppository, not a cream. Because there is little systemic absorption, and alprostadil does not affect PDE-6, changes in color vision are not likely.

** PDE5 inhibitors are ineffective without sexual stimulation. So sexual stimulation is essential to be successful

1) B
2) D
3) D
4) A
5) D
6) D
7) B

MCQs

Q8: which one of the following drugs need sexual stimulation to be effective in treatment of erectile dysfunction ?

A. Korean Ginseng. B. Nitroglycerine. C. Vardenafil. D. Papaverine.

Q9: Which one of the following adverse effects/complications explain the highly contraindication to combine quinidine with Vardenafil ?

A. Anaphylactic reaction due to Hypersensitivity to these drugs.
B. fatal arrhythmias due to prolonged QT interval.
C. Sever hypotension due to potentiated effect.
D. Both A & B.

Q10: A patient who is taking a PDE-5 inhibitors for ED, later he is diagnosed with angina and be treated with Nitroglycerin. Which of the following medications would be the safest to be used in this patient to treat erectile dysfunction ?

A. Nitroglycerin . B. Vardenafil . C. Phentolamine. D. Apomorphine.

Q11: Which one of the following drugs can be safe to be used in combination with PDE-5 inhibitors ?

A. Prazosin. B. procainamide. C. Nitroglycerin. D. Tamsulosin.

Q12: Which one of the following PDE-5 inhibitors may lead to visual disturbance due to its action on PDE-6 in photoreceptors ?

A. Sildenafil. B. Vardenafil. C. Tadalafil. D. Avanafil.

Q13: Which one of the following adverse effects/complications explain the highly contraindication to combine nitrates with any drug from PDE_5 inhibitors class ?

A. Anaphylactic reaction due to Hypersensitivity to these drugs.
B. fatal arrhythmias due to prolonged QT interval.
C. Sever hypotension due to potentiated effect.
D. Both A & B.

Q14: 44 years old male came to ER with Priapism persist for 6 hours. Which one of the following drugs is highly recommended in his case ?

A. Trazodone. B. Phenylephrine . C. Phentolamine. D. Apomorphine.

Q15: Which one of the following can be used to Treat the Premature Ejaculation in male* ?**

A. Trazodone. B. fluoxetine. C. Phentolamine. D. Vardenafil.

*** SSRI and other Anti-depressant drugs are the most common class of drugs which cause ED in male. Also SSRI such as fluoxetine can be used in case of Premature Ejaculation in male

8) C
9) B
10) D
11) D
12) A
13) C
14) B
15) B

إِنَّ فِي ذَٰلِكَ لَآيَاتٍ لِّقَوْمٍ يَتَفَكَّرُونَ ﴿٣﴾

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