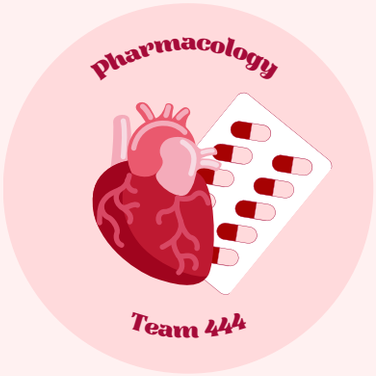
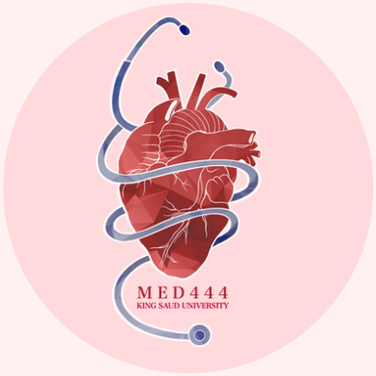




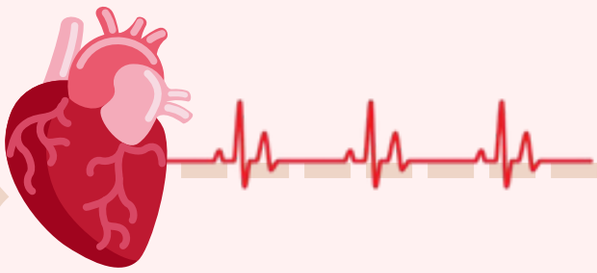
L3.

Drug therapy of heart failure [1] & [2]

EDITING FILE

COLOR INDEX :

- MAIN TEXT
- IMPORTANT
- GIRL'S SLIDES
- BOY'S SLIDES
- NOTES
- EXTRA



فشل قلب المريض وودّه يرجع
خلِ بري لود وأفتر لود صيدك
وبعدين الدجُكسن خلّه يفزع
يخلّي قبضته تشبه حديدك

✍️ علي العبدالعظيم

Objectives:

- Describe the different classes of drugs used for treatment of acute & chronic heart failure & their mechanism of action.

- Understand their pharmacological effects, clinical uses, adverse effects & their interactions with other drugs.

First video
Intro min 4:20
Left heart failure min 8:33
Heart sounds min 15:00
Right heart failure min 17:00
Pathological classification min 25:00
Pathophysiology min 28:30

Second video(only about digoxin)
Intro first 10 mins
Digoxin min 10:00
Brain bridge reflex min 29:00
Conductivity min 31:00
Rhythmicity min 32:30
ECG min 39:30
Uses of digoxin min 49:30
Atrial fibrillation min 50:30
Contraindications min 54:00
Toxicity min 1:20:00
Cardiac and non cardiac 1:24:30

Third video
Intro first 5 mins
PDE inhibitors min 6:00
ACEI & Diuretics min 17:00
Spironolactone min 21:00
lasilactone min 24:00
Venodilators min 26:00
Beta blockers min 30:00
Cardiogenic pulmonary edema & Loop diuretics min 37:00
Nitroglycerine min 49:00

What is Heart Failure?

Definition:

Inability of the heart to maintain an adequate cardiac output to meet the metabolic demands of the body. (progressive)

Causes (acute or chronic)

- Heart valve disorder.
- High blood pressure.
- Disorder of coronary arteries.
- Cardiomyopathy.
- Abnormal heart rhythm

Disease in coronary arteries will decrease the blood flow and blood supply to the heart which leads to decrease in O2 supply to the heart , and cause heart failure

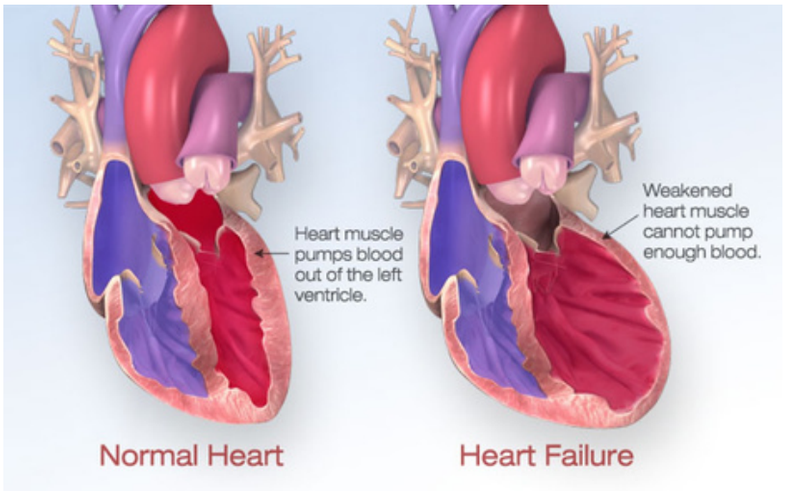
Symptoms

- Tachycardia.
- Cardiomegaly. (Abnormal enlargement of heart)
- Decreased exercise tolerance. (Rapid Fatigue)
- Peripheral edema. (Right-sided heart failure)
  Due to poor circulation
- Dyspnea (Pulmonary congestion). (Left-sided heart failure)

the causes could be due to

- Extrinsic factors : factors that out of the heart
- intrinsic factors: factors that related to the heart itself

Extrinsic factors: High BP - Disorder of coronary arteries . Rest of the causes are Intrinsic factors.



Pathophysiology of Congestive Heart Failure (CHF)

When there is low CO it will cause the heart to undergo compensatory responses:

Factors affecting cardiac output and heart failure:
1- Preload
2- Afterload
3- Cardiac contractility
الادوية اللي بناخذها كلها راح تأثر على هذه العوامل
442

↓ Force of contraction

↓ C.O (Cardiac output)

↓ Arterial Pressure

28:20

Baroreceptors in carotid artery sense changes in arterial BP

↓ Renal blood flow ← Compensatory mechanism → **↓ Carotid sinus firing**

Activate renin-angiotensin Aldosterone system (RAAS)

Activate sympathetic system ↑ Sympathetic discharge

Aldosterone | Remodeling | Angiotensin ll

↑ Force of cardiac contraction | ↑ Heart rate

Salt & water retention

Vasoconstriction (VC)

Blood volume expansion

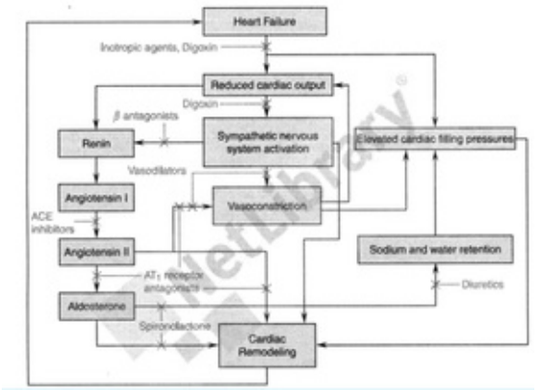
Venous VC | Arterial VC

↑ Preload | ↑ Afterload

Drugs used in treatment of heart failure

The ultimate goal of therapy:

1- Alleviate symptoms
2- Slow disease progression
3- Improve survival



Site of action

|- Drugs that decrease **preload** **(venous return)**	
1- Diuretics (used to decrease accumulation of fluid).	Chlorothiazide, Furosemide
2- Aldosterone antagonists	Spironolactone, Eplerenone
3- Venodilators	Nitroglycerine, Isosorbide dinitrate
||- Drugs that decrease **afterload** **(peripheral resistance)**	
1- Arteriodilators	Hydralazine
|||- Drugs that decrease both **preload and afterload** (combined arteriolo- &venodilators)	
1- Angiotensin converting enzyme (ACE) inhibitors	Captopril, Enalapril, Ramipril
2- Angiotensin receptor antagonists	Losartan, Valsartan, Irbesartan
3- α1- adrenoceptor antagonists	Prazosin
4- Direct vasodilators	Sodium nitroprusside
5- Beta blockers	Bisoprolol, Metoprolol, Carvedilol, Nebivolol

lV- Drugs that increase **heart contractility**	
1- Cardiac glycosides (digitalis)	Digoxin
2- (selective) β- adrenoreceptor agonists	Dobutamine
3- Phosphodiesterase inhibitors	Milrinone, Enoximone, Vesnarinone
β- adrenoceptors blockers in heart failure	
Second generation	Bisoprolol, Metoprolol
Third generation	Carvedilol, Nebivolol
New drugs for heart failure	
Natriuretic peptides	Nesirtide
Calcium sensitisers	Levosimendan

I- Drugs that decrease preload

1. Diuretics
2. Aldosterone antagonists
3. Venodilators

1- Diuretics (↓ Congestion & edema)

Drug	Chlorothiazide	Furosemide (lasix)
Subgroup	Thiazides	Loop diuretics
M.O.A in HF	-Reduce water and salt retention (↑ excretion) → decrease ventricular preload and venous pressure → reduction of cardiac size → improvement of cardiac performance. -different diuretics function on different parts of a nephron of the kidney, and because each part is responsible for a certain percentage of salt & water excretion, the part a diuretic drug functions on determines how strong it is (it's efficacy). Therefore, a drug that works on a part that secrets 60% of salt & water is strong, but a drug that works on a part that secrets 5% is weak. -A nephron is divided into 4 parts: proximal convoluted tubule→loop of henle→thick ascending limb of loop of henle→ distal convoluted tubule. Cause HyperCalcEmia → Thiazide diuretics Cause HyperKalEmia → K+ Sparing diuretics ***Important***	

MOA	Works on distal convoluted (secretion of 5% of water &salt). Not a strong diuretics (mild).	Works on Na-Cl concentrationes in cells of the thick ascending limb of loop of henle (secretion of 25% of water & salt) (5 times stronger than chlorothiazide). A potent diuretic.
Use	-First line agent in heart failure therapy. (to control edema) -Used in volume overload (Pulmonary and/or peripheral edema). (↓ Pulmonary congestion). -Used in mild congestive heart failure. (mild because it works on the part that secretes 5%of water and salt) بالبداية فقط لما تكون حالة المريض مستقرة وليست طارئة. It is commonly used with losartan and valsartan	-loop is better than thiazide in HF, used in emergency & acute case -Used for immediate reduction of pulmonary congestion (edema) & severe edema associated with: 1-Acute heart failure 2- Moderate & severe chronic failure. - ↑ urine output, cause hypotension and hypokalemia
ADRs	- ↑ urine output, cause hypotension and hypokalemia	

2- Aldosterone antagonists

Drugs	**Spironolactone (most famous)**	**Eplerenone (better)**
MOA	Non-selective antagonist of aldosterone receptor. (non-selective: it can bind to other steroid hormones receptors,like androgen & estrogen. ممكن يسبب تثدي "gynecomastia") A potassium sparing diuretic (K+ is not--excreted→hyperkalemia) .so it's drug of choice when the patient has hypokalemia	A new selective Aldosterone receptor antagonist (does not inhibit other hormones such as estrogen & androgens).

Use	Improves survival in advanced heart failure	Indicated to improve survival of stable patients with congestive heart failure

3- Venodilators

Drug	**Nitroglycerine**	**Isosorbide dinitrate**
PK	-Used IV. -Used in emergency.	
M.O.A	- ↑ cGMP in smooth muscles of vessels→**Dilates venous blood vessels & reduce preload.**	
Use	-Used IV for severe heart failure when the main symptom is dyspnea due to pulmonary congestion and edema. -Dyspnea is often a symptom associated with increased preload. (Venodilators are used in Angina too).	

II- Drugs that decrease afterload

Arteriodilators

(mainly used in hypertension & HF)

Drug	**Hydralazine** حيدر الحزين
M.O.A	Direct relaxation of vascular smooth muscle cells in resistance Arterioles→reduce peripheral vascular resistance
Use	Used when the main symptom is rapid fatigue due to low cardiac output
ADRs	Hypotension, lupus- like syndrome

III- Drugs that decrease both Afterload & preload

1. ACE Inhibitors (Angiotensin converting enzyme)
2. ARBs (Angiotensin receptors blockers)
3. α-Adrenoceptors blockers
4. Direct acting vasodilators
5. β- blockers

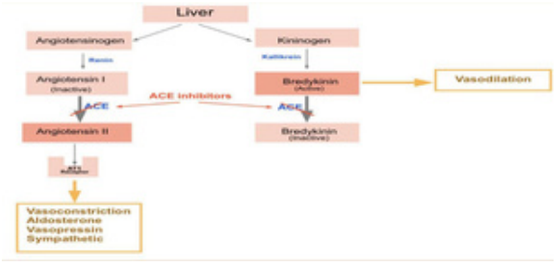
Angiotensin converting enzyme (ACE) inhibitors (first line therapy).

Drug	Captopril (causes dry cough, must not take it with losartan)	Enalapril	Ramipril
Use	-Considered as first-line drugs for chronic heart failure along with diuretics. -First-line drugs for hypertension therapy.		
PK	-Rapidly absorbed from GIT after oral administration. -Food reduce their bioavailability. Therefore, the patient is advised to take these medication in an empty stomach or 2 h b4 the meal		
PK	-	-prodrugs, converted to their active metabolites in liver. -Have long half-life & given once daily.	

M.O.A

-inhibiting ACE, will achievethe opposite of all angiotensin II normal actions in his results in increase in CO.

Helpful video:

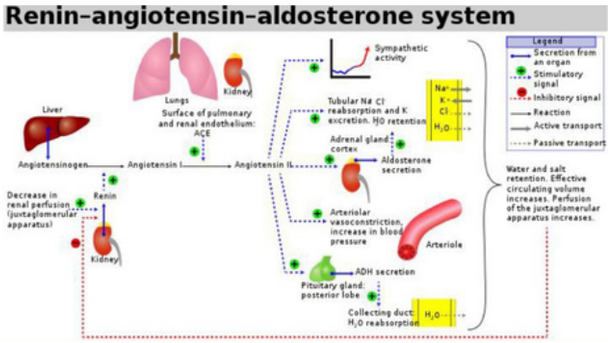


RAAS System Effects

-plasma protein Angiotensinogen (synthesized in the liver) is converted to angiotensin I by renin (enzyme in juxtaglomerular cells of the kidney & then released in the circulation).
ACE enzyme converts Angiotensin 1 → Angiotensin 2

Angiotensin II effects:
→extremely powerful vasoconstrictor (constriction of arterioles)
→ ↑total peripheral resistance → ↑arterial pressure (mild constriction of veins)
→ ↑venous return
→ stimulating the secretion of aldosterone (sodium and water retention).
→stimulating the secretion of Vasopressin (antidiuretic hormone) →water retention
→stimulating the sympathetic nervous system
→causes hypertrophy of vascular & cardiac cells & increases synthesis & disposition of collagen by cardiac
fibroblast (remodeling).
→Increased renin in the body is mainly responsible for cardiac & vascular remodeling.
→ACE (kininase II) is also essential for the breakdown of bradykinin (additive vasodilation effects).

EXTRA
*text in red is in slides

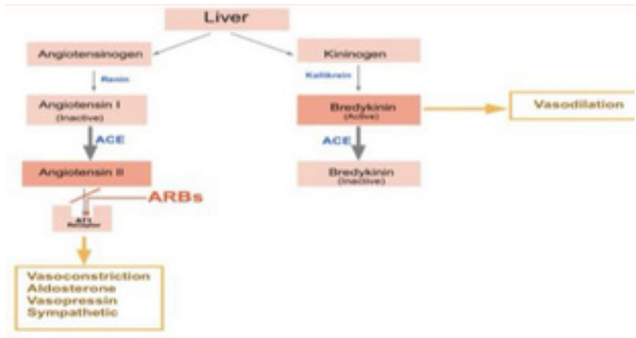


Pharmacological actions	-Decrease peripheral resistance (Afterload) (arteriodilation). -Decrease venous return (preload) (venodilation). -Decrease sympathetic activity. -Inhibit cardiac and vascular remodeling associated with chronic heart failure (protect to the heart) →decrease in mortality rate.
ADRs	-Acute renal failure, especially in patients with renal artery stenosis. -Hyperkalemia, especially in patients with renal insufficiency or diabetes. (Aldosterone will increase Na retention and K excretion so when we inhibit it that will lead to increase in K in our body. And when the patient has renal insufficiency the K will increase more because he has already poor excretion of K). -Severe hypotension in hypovolemic patients →hypovolemic due to diuretics, salt restriction or gastrointestinal fluid loss e.g. severe vomiting or diarrhea. -Dysgeusia (reversible loss or altered taste). (reversible: if we stop the drug the side effects will disappear). *the last 2 ADRs are associated with Bradykinin accumulation (important):- -Dry cough sometimes with wheezing because of Bradykinin accumulation  ***Important*** -Angioneurotic edema (swelling in the nose, throat, tongue, laryn). if in the layrnx this is a severe case
Contraindications	-During the second and third trimesters of pregnancy (not recommended for the first trimester too). (due to high risk of: fetal hypotension,renal failure, and malformation) -Renal artery stenosis (will cause renal failure).

III- Drugs that decrease both Afterload & preload

Angiotensin receptor blockers (ARBs)

Used if ACE inhibitors are contraindicated or untolerated. (just affect receptor not production

Drug	Losartan	Valsartan	Irbesartan
M.O.A	**- Block angiotensin 1 (AT1) receptors.** Less ADRs due to bradykinin accumulation **- Decrease action of angiotensin II.** - AT1 mediates most of the known actions of Ang & predominate in vascular smooth muscle → renal sodium reabsorption, vasoconstriction, cell growth and proliferation (remodeling). - AT2 → natriuresis, vasodilation, anti proliferation. 		

α-Adrenoceptor BLOCKERS

Drug	Prazosin
M.O.A	- Blocks α- receptors in arterioles and venules. - Decrease both afterload & preload.

Direct acting vasodilators (by ↑ cGMP)

drug	Sodium nitroprusside
P.K	- Acts immediately and effects lasts for 1-5 min. (it doesn't affect the receptors it acts directly on blood vessels , so the action will be very fast)
Uses	- Given I.V. for acute or severe heart failure.

4-Drugs that increase contractility

1. Cardiac glycosides (digitalis)
2. β-Adrenoceptor AGONIST
3. Phosphodiesterase - III inhibitors

Cardiac glycosides (digitalis)

drug	Digoxin
M.O.A	**Inhibits Na+ / K+ ATPase enzyme (the sodium pump)→** **-Increases the force of myocardial contraction (+ve inotropic effect).** Normal function of Na/K pumps -number 1 in the picture- : 1. Na/K pump, works during repolarization: K in, Na out (against gradient) 2. Na/Ca pump : Ca out, Na in Abnormal function of pumps due to presence of Digoxin : 1. Blocked Na/K channels 2. Reversed Na/Ca pumps (Na out ,Ca in)

Uses	- Congestive heart failure - has narrow therapeutic index ONLY if patient has decrease in contractility (438).	
ADRs	Cardiac: digitalis-induced arrhythmias: - Extrasystoles. - Coupled beats (Bigeminal rhythm). - Ventricular tachycardia or fibrillation. - Cardiac arrest (toxic dose).	Non-cardiac: -GIT anorexia (loss of appetite), nausea, vomiting, diarrhea -CNS: headache, visual disturbances, drowsiness. visual disturbances: Can see halos in light
Factors that Increase its toxicity **(check for ion balance before starting therapy)**	1. Renal diseases (because it's excreted through it & because the kidney is responsible for ion balance in the body and any imbalance in ions will affect digoxin toxicity). 2. Electrolyte disturbances: ● Hypokalemia (↓ K so easier binding of digoxin,could happen by taking non-K sparing diuretics). ● Hypomagnesemia Mg is cofactor of sodium pump so if it not present the pump won't function achieving what digoxin is already trying to achieve). ● Hypercalcemia (↑ ↑ intracellular Ca). it already causes calcium influx so having too much calcium entering the cells with cause problems	

β-Adrenoreceptor Agonist

drug	**Dobutamine**
M.O.A	**Selective β1 agonist.** **Remember: You have one heart so β1, And you have two lungs so β2**

Uses	Treatment of acute heart failure in cardiogenic shock

Phosphodiesterase-III inhibitors (↑ cAMP)

DRUG	**Milrinone**	**Enoximone** **Vesnarinone**
M.O.A	Inhibit phosphodiesterase -III (cardiac & B.vessels) → ↑ cAMP. - ↑ cAMP in cardiomyocytes → increases cardiac contractility. - ↑ cAMP in vascular smooth muscles→ dilatation of arteries & veins (reduction of preload & afterload). Extra explanation from the doctor *click here*	
Uses	-Used only IV for management of acute heart failure. -Not safe for effective in the longer (>48 hours) treatment of patients with heart failure.	New drugs in clinical trials.
ADRs	- Hypotension and chest pain (angina) (so it's not use at more than 2 days)	-
Chemical interactions	- Furosemide (Diuretic drug) shouldn't be administered in IV lines containing milrinone due to formation of a precipitate. راسب كيميائي :Precipitate فالدوائين ال2 مايشتغلون لأنه لازم الدواء يكون ذائب في المحلول وليس مترسب DI is either 1- PK , 2- PharmacoD ,3- Chemical Extra explanation *click here*	-

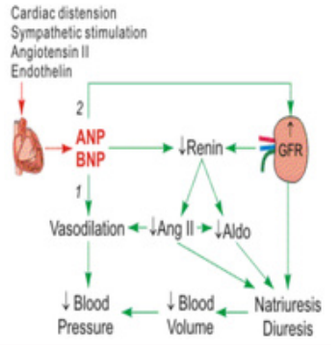
β-adrenoceptor blockers

The elevated adrenergic activity in chronic heart failure patients cause structural remodeling of the heart (cardiac dilatation & hypertrophy)

Generation	**Second generation**	**Third generation**	
Drug	**Eg: Bisoprolol Metoprolol**	**Eg: Carvedilol**	**Eg: Nebivolol**
	Cardioselective (β1-receptor)	β-1 receptor blocker + have additional vasodilator action (a blocking effect).	
		Non selective vasodilators (alpha and beta blocker) (a1, β2) (selective a and non selective β-blocker).	β1- selective with vasodilating properties not mediated by a blockade due to increase in endothelial release of NO via indication of eNOS.
M.O.A in HF	1.Attenuate/slows down cardiac remodeling. 2.Slow heart rate, which allows the left ventricle to fill more completely. **3.Decrease renin release** ***57:41 R>B*** ***Important*** → reduce mortality & morbidity of patients with HF.		
Use	- Reduce the progression of chronic heart failure. For stable patient. - NOT used in acute heart failure.		

New Drugs for Heart Failure:

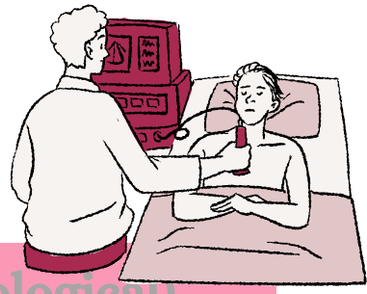
1-Natriuretic peptides

Drug	Nesiritide	
Definition	- A purified preparation of human BNP, manufactured by recombinant DNA technology - BNP is a hormone secreted by cardiomyocyte in the heart ventricles in response to stretch caused by increased ventricular blood. - Elevated BNP and ANP are associated with advanced HF (compensatory mechanism in HF)	
M.O.A	- Physiological effects of ANP and BNP: - Vasodilation. - Natriuresis (excretion of sodium in urine) - Inhibition of RAAS (inhibitory effects on renin secretion, inhibit the action of ANG II & aldosterone). - ↑ cyclic-GMP in vascular smooth muscle leading to: ● smooth muscle relaxation (vasodilation). ● reduction of preload and afterload.	
Uses	Indicated (IV) for the treatment of patients with (ADHF "acute decompensated heart failure") who have dyspnea at rest or with minimal activity (not given in stable cases).	

2-Calcium sensitisers

Drug	Levosimendan
M.O.A	- Calcium sensitization (improves cardiac contractility Without increasing oxygen consumption) (no extra work on heart). - Potassium-ATP channel opening (cause vasodilation, improving blood flow to vital organs). - These effects reduce the risk of worsening ADHF or death compared with dobutamine.
uses	Used in management of ADHF (not given in stable cases).

Management of heart failure:



Management of Chronic Heart Failure (Non-pharmacological).

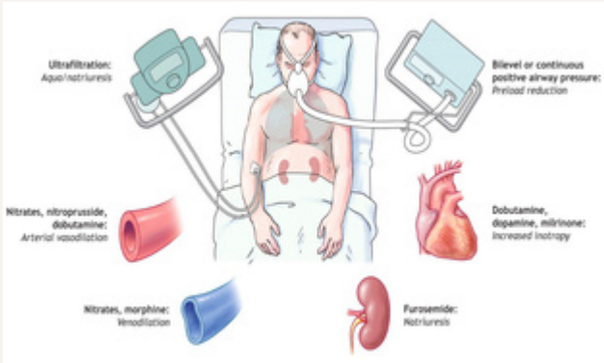
- Reduce workload of the heart ex:
  A)Limit patient activity.
  b)Reduce weight
  C)Control hypertension
- Restrict sodium because ↑ sodium → ↑ BP → ↑ edema.
- Stop smoking.

Management of acute heart failure

ADHF is a common and potentially serious cause of respiratory distress.

Acute Decompensated Heart failure (ADHF): A sudden worsening of signs and symptoms of heart failure,which typically includes:

- difficulty breathing (dyspnea)
- leg or feet swelling
- 3- fatigue



Congestive heart failure in black patients

Hydralazine (Arterial Dilator)/ isosorbide dinitrate (venodilators) fixed dose combination.

●FDA approved to add to standard therapy for black Americans with Congestive heart failure.

(due to poor response to ACE inhibitors).

● Should be considered for patient intolerant to ACE inhibitor & ARBs due to renal dysfunction.

Heart Failure Functional Classification and Management of Chronic Heart failure:

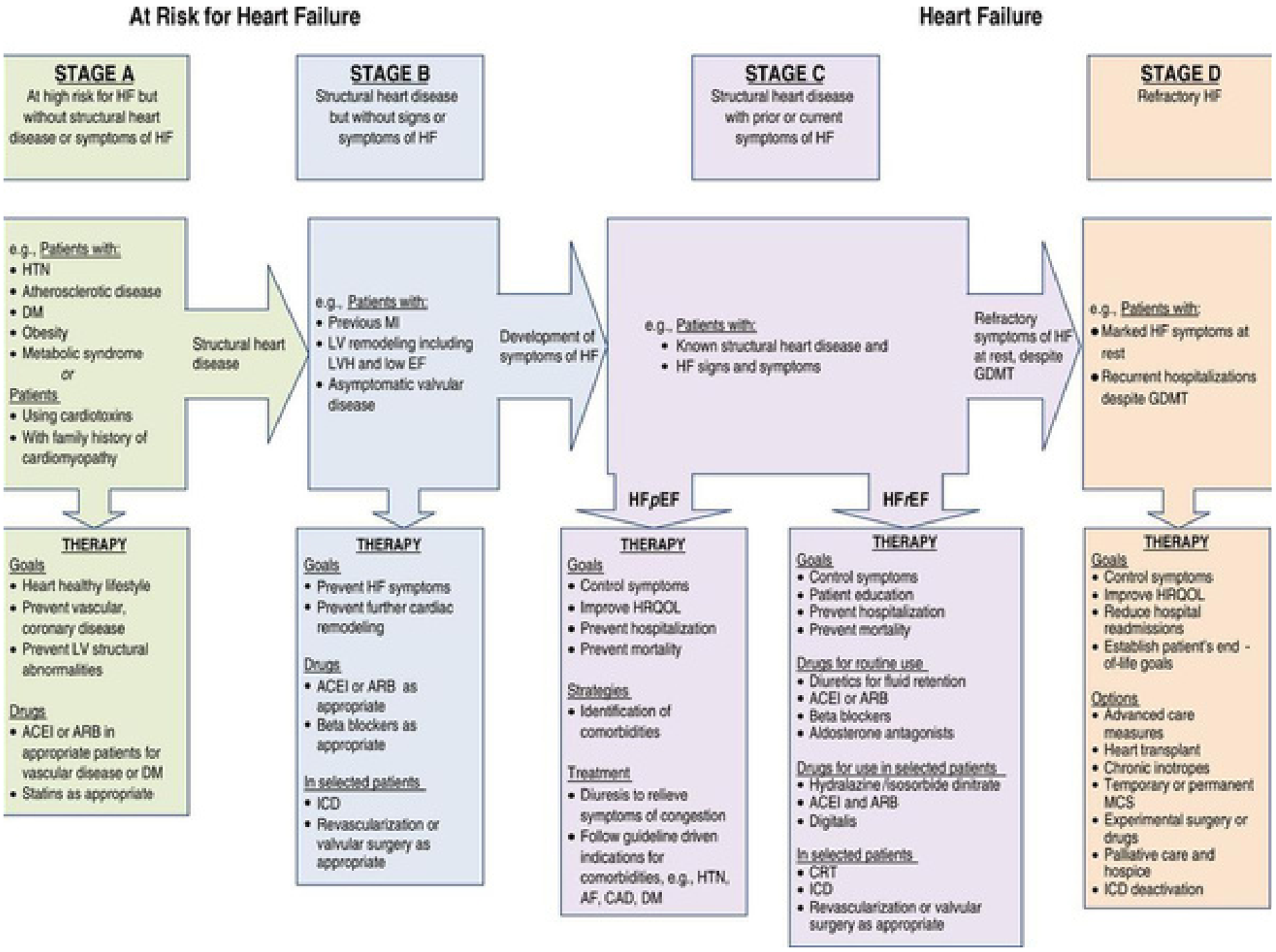
The new guidelines classify heart failure (HF) into three subgroups based on the ejection fraction (EF):

- HF with reduced EF (HFrEF)
- HF with mid-range EF (HFmrEF)
- HF with preserved EF (HFpEF).

◆ The severity of heart failure is usually described according to a scale devised by the New York Heart Association (NYHA):

NYHA Class:		I	II	III	IV
Functional classification	Symptoms	Cardiac disease, but no symptoms & no limitation in ordinary physical activity, eg: no shortness of breath when walking, climbing stairs etc. -symptoms occur only with greaterthan ordinary exercise.	Mild symptoms (mild shortness of breath &/or angina), slight limitation during activity which result in fatigue and palpitations.	Marked limitation in activity due to symptoms (fatigue,etc), even during less-than ordinary activity, eg: walking short distance (20-100m). Comfortable only at rest (no symptoms).	Severe limitation, experiences symptoms even while at rest.Mostly bed bound patients.
MAnagement of chronic heart failure	for survival (morbidity)	Continue ACE inhibitor/ARB if ACE inhibitor intolerant, continue aldosterone antagonist If post-MI and add beta-blocker if post MI used as prophylaxis so it doesn't get worse	ACE inhibitor as first line treatment/ARB if ACE inhibitor intolerant add beta blocker and aldosterone antagonist if post-MI.	ACE inhibitor + ARB or ARB alone if ACE intolerant Beta blocker Add aldosterone antagonist	-Continue ACE inhibitor/ARB -Beta blocker -aldosterone antagonist
	for symptoms	Reduce/stop diuretic	+/- diuretic depending on fluid retention	+diuretic +Digitalis if still symptomatic	+Diuretic +Digitalis +Consider temporary inotropic support

EXTRA SLIDE


At Risk for Heart Failure
Heart Failure
STAGE A
At high risk for HF but without structural heart disease or symptoms of HF
STAGE B
Structural heart disease but without signs or symptoms of HF
STAGE C
Structural heart disease with prior or current symptoms of HF
STAGE D
Refractory HF
e.g., Patients with:
• HTN
• Atherosclerotic disease
• DM
• Obesity
• Metabolic syndrome
or
Patients
• Using cardiotoxins
• With family history of cardiomyopathy
Structural heart disease
e.g., Patients with:
• Previous MI
• LV remodeling including LVH and low EF
• Asymptomatic valvular disease
Development of symptoms of HF
e.g., Patients with:
• Known structural heart disease and
• HF signs and symptoms
Refractory symptoms of HF at rest, despite GDMT
e.g., Patients with:
• Marked HF symptoms at rest
• Recurrent hospitalizations despite GDMT
HFpEF
HFrEF
THERAPY
Goals
• Heart healthy lifestyle
• Prevent vascular, coronary disease
• Prevent LV structural abnormalities
Drugs
• ACEI or ARB in appropriate patients for vascular disease or DM
• Statins as appropriate
THERAPY
Goals
• Prevent HF symptoms
• Prevent further cardiac remodeling
Drugs
• ACEI or ARB as appropriate
• Beta blockers as appropriate
In selected patients
• ICD
• Revascularization or valvular surgery as appropriate
THERAPY
Goals
• Control symptoms
• Improve HRQOL
• Prevent hospitalization
• Prevent mortality
Strategies
• Identification of comorbidities
Treatment
• Diuresis to relieve symptoms of congestion
• Follow guideline driven indications for comorbidities, e.g., HTN, AF, CAD, DM
THERAPY
Goals
• Control symptoms
• Patient education
• Prevent hospitalization
• Prevent mortality
Drugs for routine use
• Diuretics for fluid retention
• ACEI or ARB
• Beta blockers
• Aldosterone antagonists
Drugs for use in selected patients
• Hydralazine /isosorbide dinitrate
• ACEI and ARB
• Digitalis
In selected patients
• CRT
• ICD
• Revascularization or valvular surgery as appropriate
THERAPY
Goals
• Control symptoms
• Improve HRQOL
• Reduce hospital readmissions
• Establish patient's end - of-life goals
Options
• Advanced care measures
• Heart transplant
• Chronic inotropes
• Temporary or permanent MCS
• Experimental surgery or drugs
• Palliative care and hospice
• ICD deactivation

“ study smarter , not harder “

Active recall

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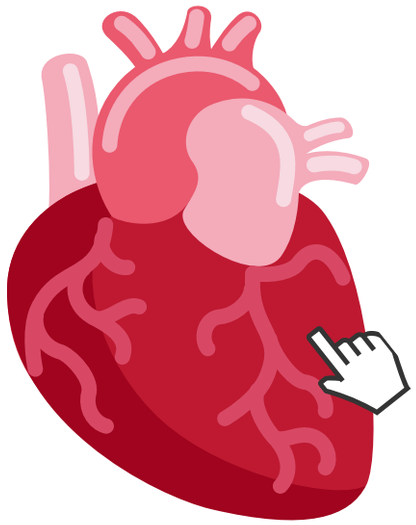
summary



MCQs

1 **What is the primary indication for the use of digoxin in the treatment of heart failure?**

- A: Preserving sinus rhythm and atrial fibrillation
- B: Reducing blood pressure in hypertension
- C: Improving myocardial contractility
- D: Preventing ventricular arrhythmias

2 **Which of the following electrolyte disturbances increases the risk of Digoxin's toxicity?**

- A: Hypocalcemia
- B: Hypomagnesemia
- C: Hyponatremia
- D: Hyperkalemia

3 **ACE inhibitors can cause dry cough in some patients .This ADR is due to?** ***Important***

- A: Histamine release
- B: Increased sympathetic activity
- C: Direct irritation of respiratory tract
- D: Bradykinin accumulation

4 **Which of these drugs could cause dry cough?**

- A: Procainamide
- B: Losartan
- C: Verapamil
- D: Captopril

C,B,D,D

MCQs

5 **Which of the following is an ADR in loop diuretics and thiazide drugs?**

A Hypokalemia
B Hyperkalemia
C Hypoglycemia
D Hyperglycemia

6 **Which of the following drugs increases contactility and causes vasodilation?**

A Milrinone
B Ranolazine
C Niacin
D Adenosine

7 **All of the following are ADRs of digoxin except?**

A Diarrhea
B Bigeminal rhythm
C visual disturbances
D Fluid discoloration

8 **What is used when the main symptom is rapid fatigue due to low cardiac output?**

A Hydralazine
B Nitroglycerine
C Digoxin
D Spirolactone

A,A,D,A

MCQs

Important

9 **Which of the following causes Hypercalcemia**

Important

A Digoxin

B Chlorothiazide

C Bisoprolol

D Captopril

10 **Which of the following causes Hyperkalemia**

Important

A Milrinone

B Ranolazine

C Niacin

D K+ sparing diuretics

11 **Patient presented to the ER with Pulmonary Edema. Which of the following drugs is considered First Line Therapy for control of edema**

Important

A Diuretics

B Beta Blockers

C Digitalis

D ACE Inhibitors

12 **Which of the following drugs decrease renin release**

Important

A Captopril

B Losartan

C Carvedilol

D Digoxin

B,D,A,C

SAQs

1. **What categories are considered as the first line agents used to treat heart failure? And mention 2 examples for each .**

Diuretics+ ACEIs
(examples in slide 6)

2. **A 53 year old patient came to the ER with acute heart failure .Milrinone was given to manage his case.Discuss its MOA,its role on preload and/or afterload and the route of administration used.**

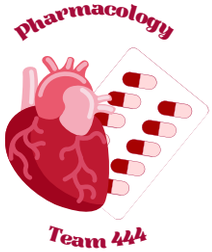
Slide 17

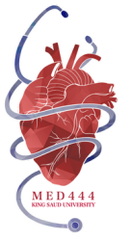
3. **A 62-year-old male patient with a history of hypertension and diabetes mellitus presents to the primary care clinic for a routine follow-up visit. His blood pressure readings have been consistently elevated, despite being on a thiazide diuretic. His current blood pressure is 150/90 mmHg. The doctor considered ACEIs as an option for management.List 4 ADRs that patient should be aware of .**

Slide13

4. **A 54 year old has severe dyspnea due to pulmonary congestion and edema along with severe heart failure. Venodilators were given to relief his symptoms. Mention 2 drugs that can be used with their MOA and route of administration .**

Slide 10





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