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Children's Cancer Hospital Foundation - Egypt
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Heart Failure

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Outlines

- ❑ What's Heart Failure ?
- ❑ Risk factors
- ❑ Staging of HF
- ❑ HF Compensatory Mechanism
- ❑ ECHO
- ❑ Background Therapy
- ❑ Add-on therapy
- ❑ Drugs should be avoided in HF

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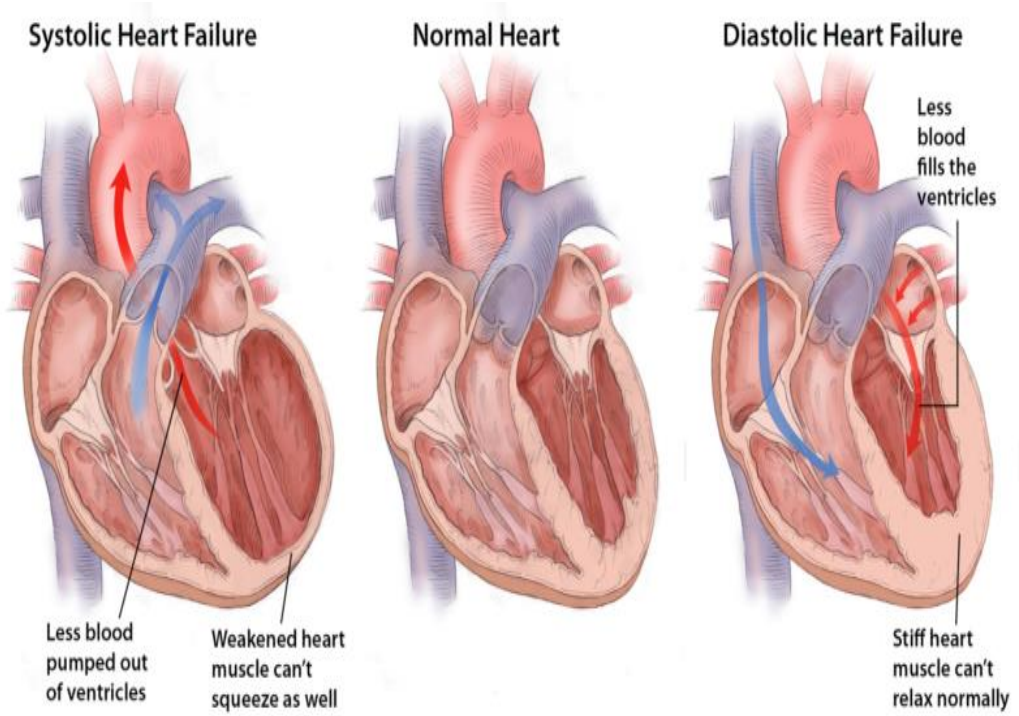
Heart Failure

- ✓ Heart failure, also known as **CHF**
- ✓ Condition that develops when **your heart doesn't pump enough blood for your body's needs**.
- ✓ This can happen if your heart **can't fill up with enough blood**.
- ✓ It can also happen when your heart **is too weak to pump properly**.



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HEART DISEASE RISK FACTORS

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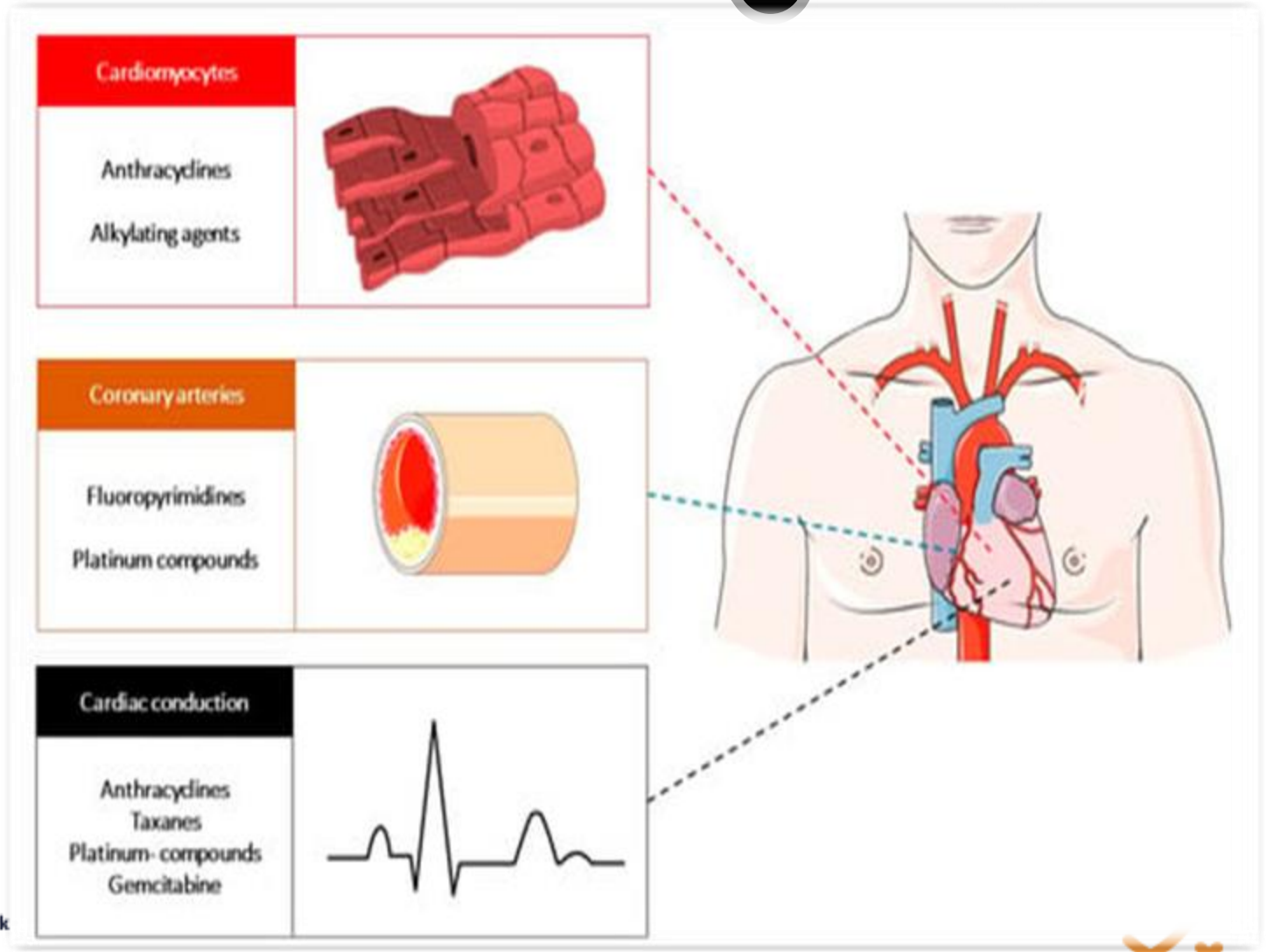
Anthracyclines

- Anthracyclines are a class of drugs used in cancer chemotherapy that are extracted from Streptomyces bacterium.
- These compounds are used to treat many cancers, including **leukemias**, **lymphomas**, **breast**, **stomach**, **uterine, ovarian, bladder cancer, and lung cancers.**
- Cumulative dose ind. Cardiotoxicity **250mg/m2**



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Reactive Oxygen Species (ROS) !!!

- ✓ Redox reactions generate reactive oxygen species in the presence of cytochrome P450 reductase,
- ✓ NADH dehydrogenase, and xanthine oxidase.
- ✓ **The excess ROS cannot be detoxified, resulting in oxidative stress**
- ✓ DNA damage, and lipid peroxidation triggering cell apoptosis.

Heart Failure

Systolic failure (HF*r*EF)

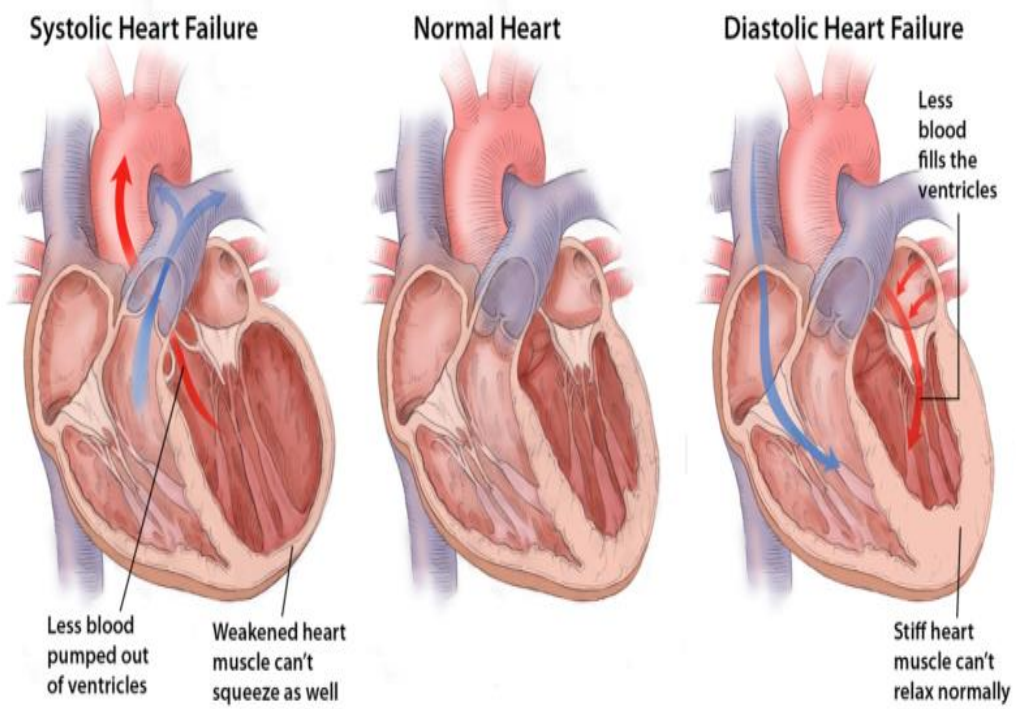
- EF < 40%
- Majority of HF cases.
- 2/3 of the cases is related to CHD
- 1/3 cases is related to non-ischemic causes :

➢ **(HTN, thyroid diseases, obesity, stress, myocarditis, tachycardia, cardiotoxin-induced)**

✓ **Cardiotoxins include (alcohol, cocaine, anthracyclines, high-dose cyclophosphamide, 5-FU, Trastuzumab, and mitoxantrone).**

Diastolic failure (HF*p*EF)

- LVEF ≥ 50% (the borderline between 41-49%)
- Normal wall motion, impaired ventricular relaxation.
- Most common in HTN.



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Staging of HF

STAGE A: At-Risk for Heart Failure

Patients at risk for HF but without current or previous symptoms/signs of HF and without structural/ functional heart disease or abnormal biomarkers

Patients with hypertension, CVD, diabetes, obesity, exposure to cardiotoxic agents, genetic variant for cardiomyopathy, or family history of cardiomyopathy

STAGE B: Pre-Heart Failure

Patients without current or previous symptoms/signs of HF but evidence of 1 of the following:

Structural heart disease

Evidence of increased filling pressures

Risk factors and
- increased natriuretic peptide levels or
- persistently elevated cardiac troponin

in the absence of competing diagnoses

STAGE C: Symptomatic Heart Failure

Patients with current or previous symptoms/signs of HF

STAGE D: Advanced Heart Failure

Marked HF symptoms that interfere with daily life and with recurrent hospitalizations despite attempts to optimize GDMT

New Onset/De Novo HF:	Resolution of Symptoms:	Persistent HF:	Worsening HF:
• Newly diagnosed HF • No previous history of HF	• Resolution of symptoms/ signs of HF Stage C with previous symptoms of HF with persistent LV dysfunction HF in remission with resolution of previous structural and/or functional heart disease*	• Persistent HF with ongoing symptoms/signs and/or limited functional capacity	• Worsening symptoms/ signs/functional capacity

Heart Failure Compensatory mechanism

NPS↑

1- Elevated BNP and NT-pro BNP

2- Naturitic diuresis (BV)

3- Dec BP

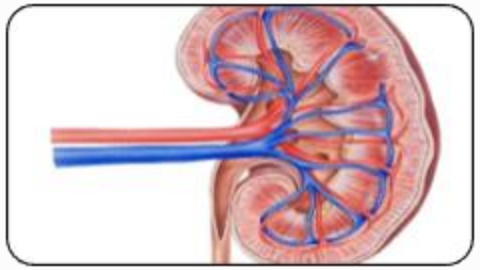
BR

1- SNS activation

2- HR ↑

3- BP ↑

3- CO ↑



RAAS

1- Renin sec ↑

2- Na/H2o retention

3- BP ↑

4- Co ↑

Cardiac Remolding

1- Ventricular wall distention

2- Myocyte growth

3- Hypertrophy

Cause of death !!

Cardiomegaly !

- Metabolic needs inc.
- Waste product inc.
- Acc. cell death Arrythmia

NPS : decreases blood pressure (BP) , lowers the sympathetic tone, and reduces aldosterone levels. antagonistically to the RAAS and has favorable effects on the pathogenesis of HF

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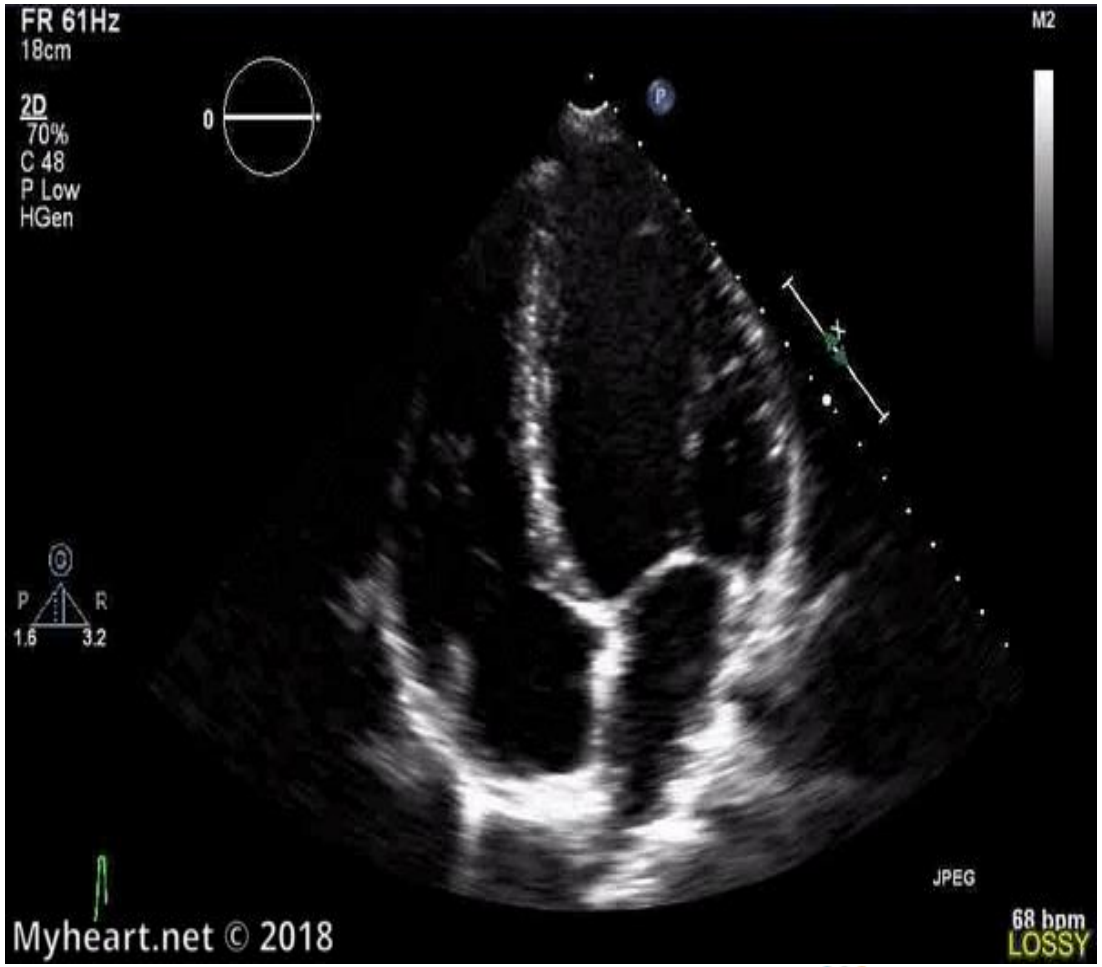
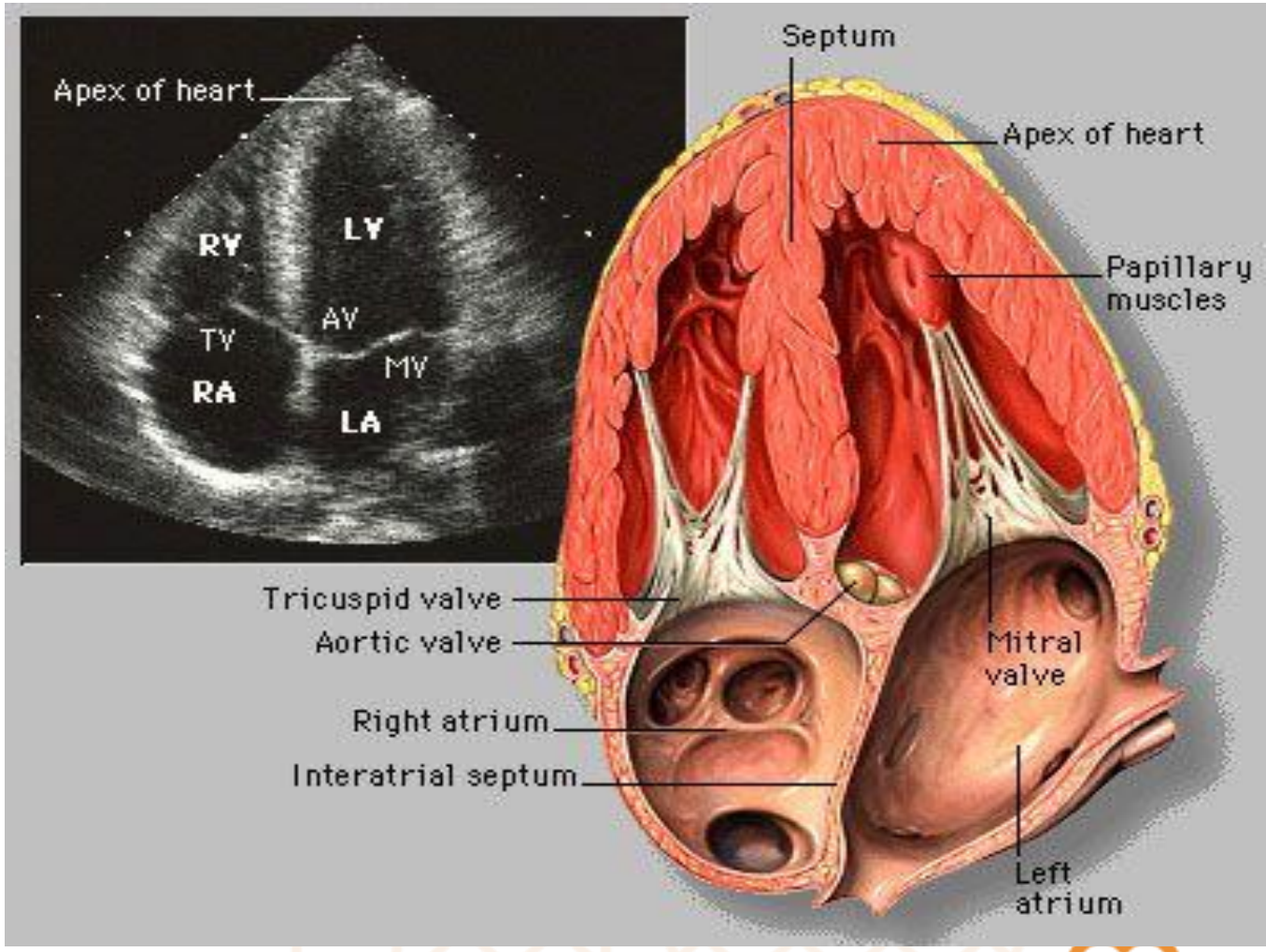
ECHO : to identify hypertrophy or blood accumulation in heart





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Background Therapy

ACEI

ARBs

Beta blockers

Digoxin ??

Sacubitril/valsartan

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Monitoring parameters

ACEI	ARBS	ARNI	B-Blocker	Digoxin
➢ Renal functions (SCR). ➢ K levels. ➢ BP and symptoms of hypotension	➢ Renal functions (SCR). ➢ K levels. ➢ BP and symptoms of hypotension	➢ Signs/sympt angioedema/ hypotension ➢ Renal functions ➢ K levels ➢ **Fluid status ?** ➢ **BNP not costant** so dep on : ❖ **pro-BNP**	➢ HR, symptoms of hypotension ➢ **Edema/fluid retention Usually resolve!!** spontaneously within 2 weeks ➢ Fatigue/weakness (1-2%)	❑ Avoid abrupt discontinuation.! ❑ Maintain drug concentrations between 0.5-0.9 ng/ml. ❑ Concomitant drugs could ↑ drug concentrations. ✓ Amiodarone We should (↓ digoxin dose by 30-50% or increase dosing interval)

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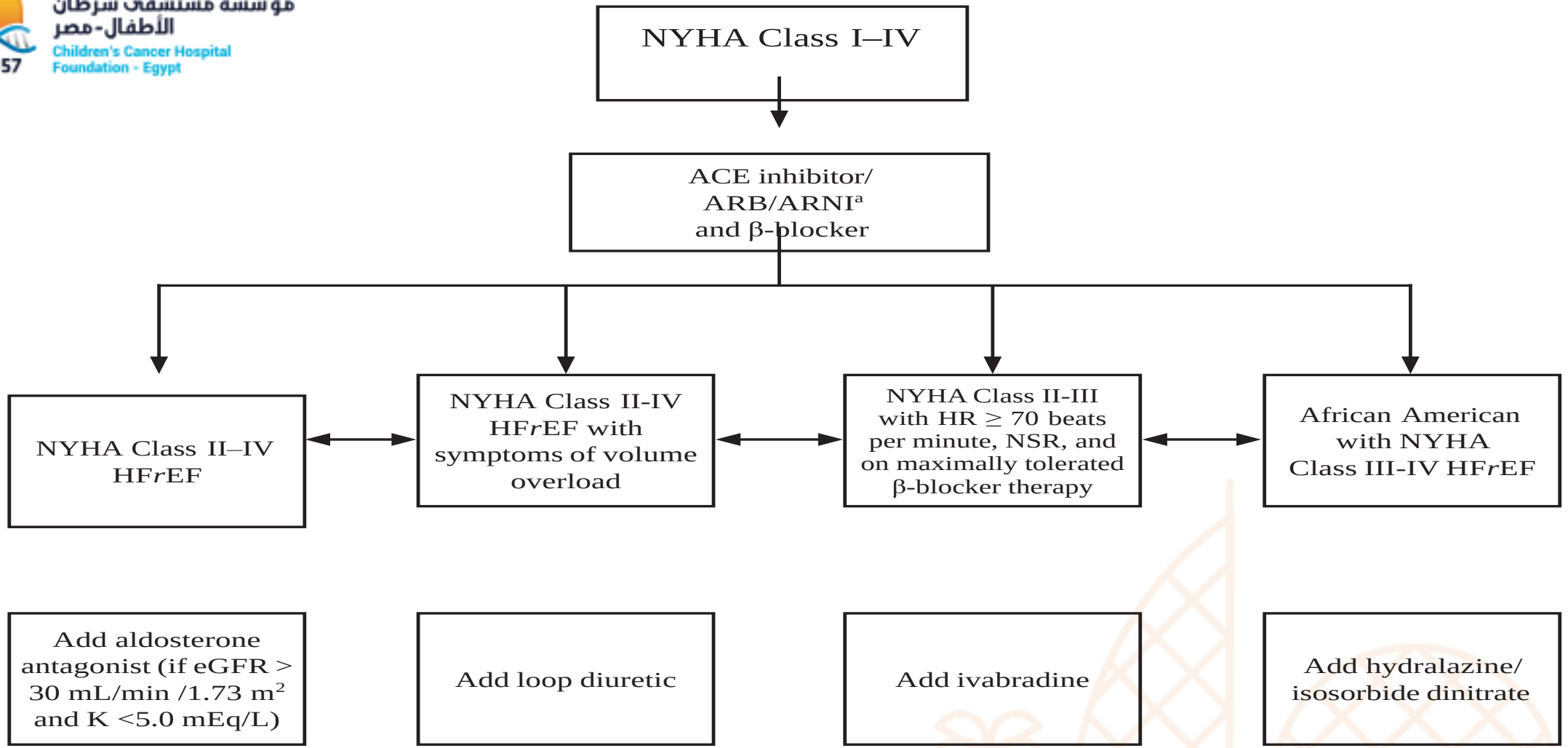
Add-on Therapy

Aldosterone antagonist

Diuretics

Ivabradine


NYHA Class I–IV
ACE inhibitor/ ARB/ARNI[a] and β-blocker
NYHA Class II–IV HFrEF
NYHA Class II-IV HFrEF with symptoms of volume overload
NYHA Class II-III with HR ≥ 70 beats per minute, NSR, and on maximally tolerated β-blocker therapy
African American with NYHA Class III-IV HFrEF
Add aldosterone antagonist (if eGFR > 30 mL/min /1.73 m² and K <5.0 mEq/L)
Add loop diuretic
Add ivabradine
Add hydralazine/ isosorbide dinitrate

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1. **2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines**
2. ECC Guidelines
3. ASHPP
4. American board for cardiology