Heart Failure Management and Prognosis

Dr. Rashed Alfagih MBBS MHSc
Consultant Cardiologist KFCC

Presentation

- Chronic or Subacute.
- **Acute.**

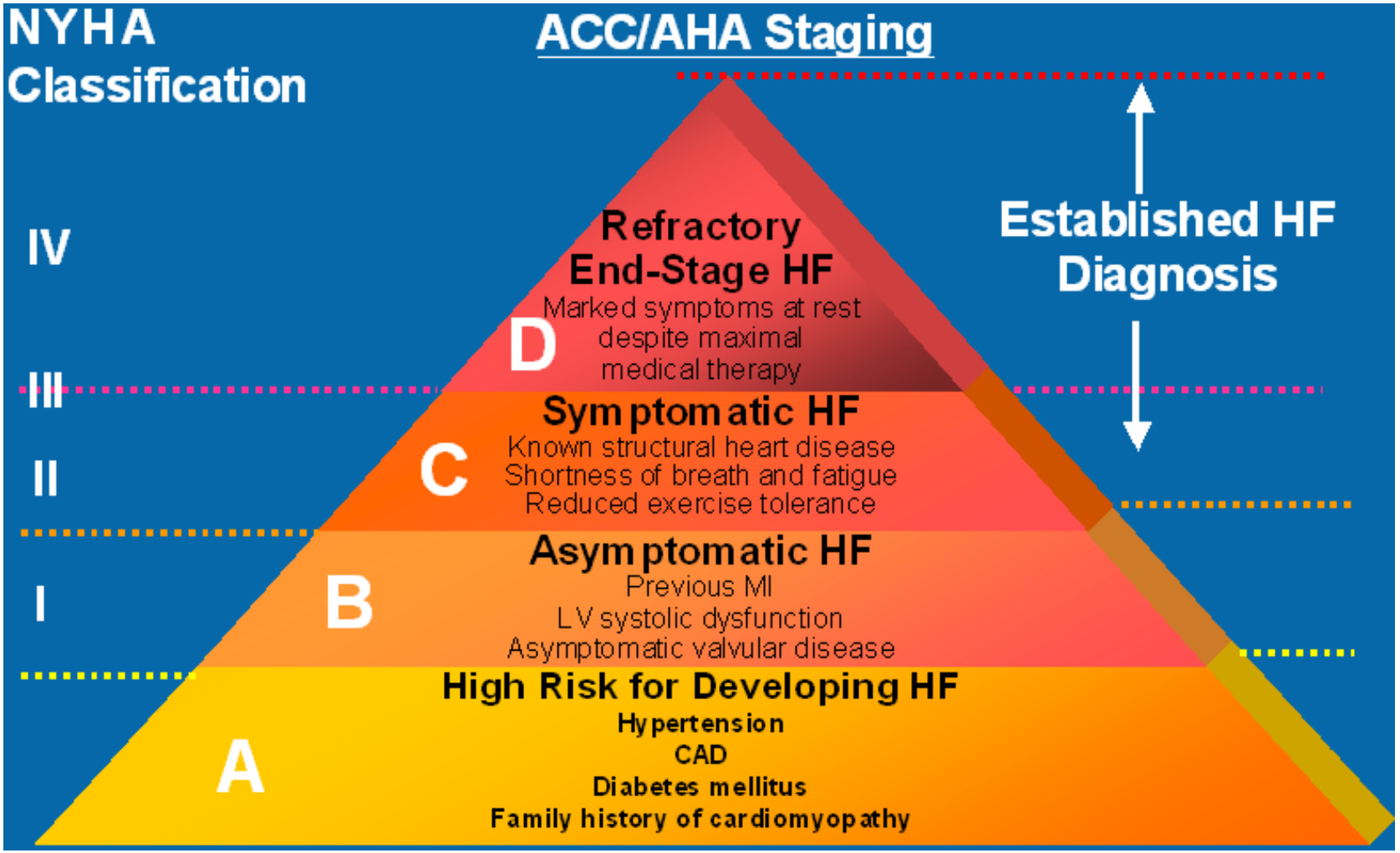
Management

- Correction of reversible causes
  - Ischemia
  - Valvular heart disease
  - Thyrotoxicosis, anemia and other high output status
  - Shunts
  - Arrhythmia
    - Tachy. Like : A fib, flutter or Brady. Like : CHB.
  - Medications
    - Ca channel blockers, some antiarrhythmics, NSAIDs,

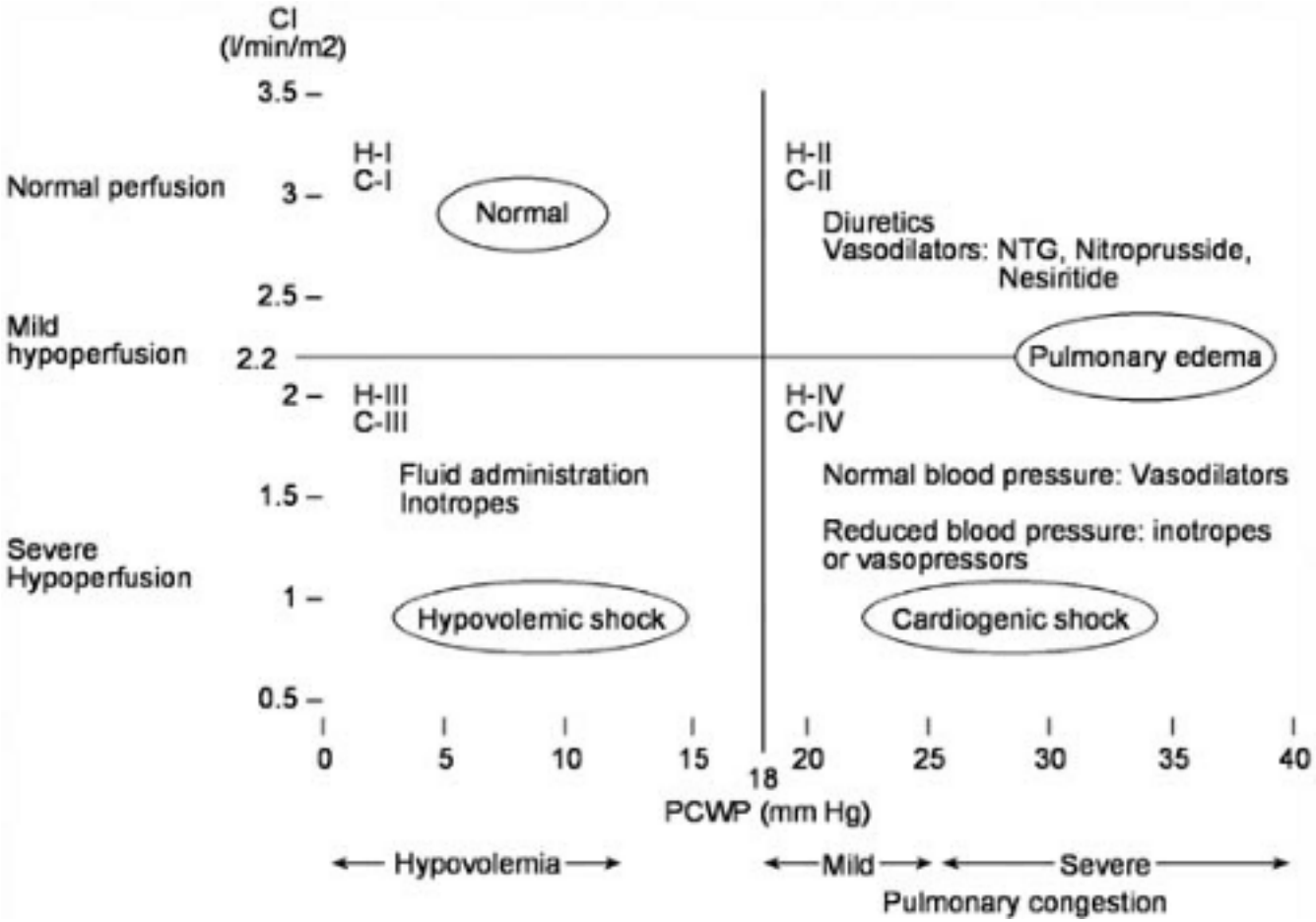
Management tools

- Life style modifications:
  - Diet, Fluid intake, exercise, Smoke cessation, Wt.
- Pharmacological interventions:
  - Oral medication.
  - Immunization.
- Surgical interventions:
  - Device therapy.
  - Assisting devices.
  - Transplant.

Stages of Heart Failure



Forrester Classification



Am J Cardiol 1977;39:137–145

Forrester Classification

		Congestion at Rest	
		No	Yes
Low Perfusion at rest	No	Warm & Dry	Warm & Wet
	Yes	Cold & Dry	Cold & Wet

Eur J Heart Fail. 1999 Aug;1(3):251-7.

Definitions

- Heart failure with reduced Ejection Fraction (HFrEF).
- Heart failure with mildly reduced Ejection Fraction (HFmrEF).
- Heart failure with preserved Ejection Fraction (HFpEF).

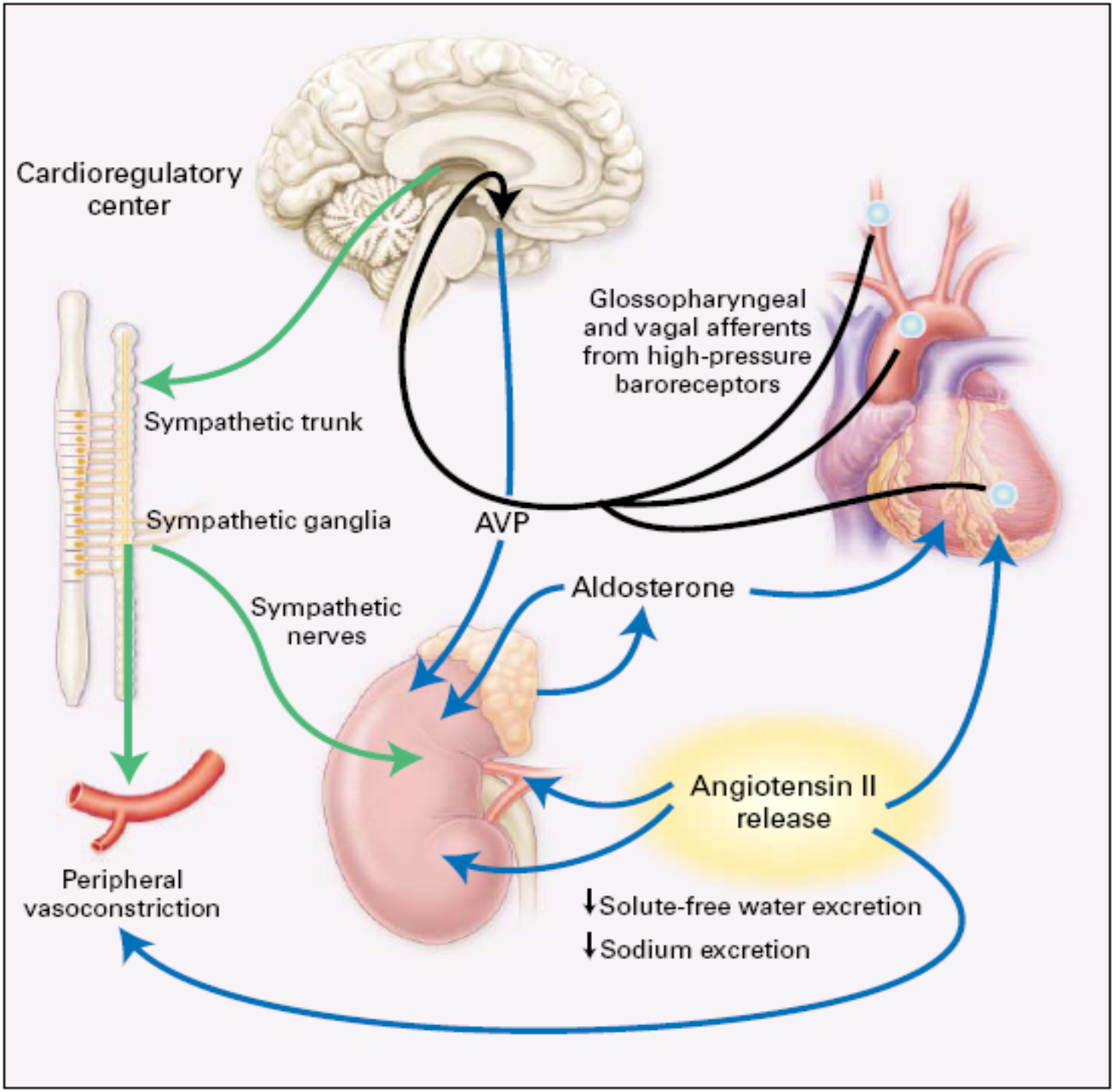
LVEF : Left Ventricular Ejection Fraction;
Determined by Echocardiography

LVEF	<= 40% HFrEF	41-49% HFmrEF	>= 50% HFpEF

Diet and Activity

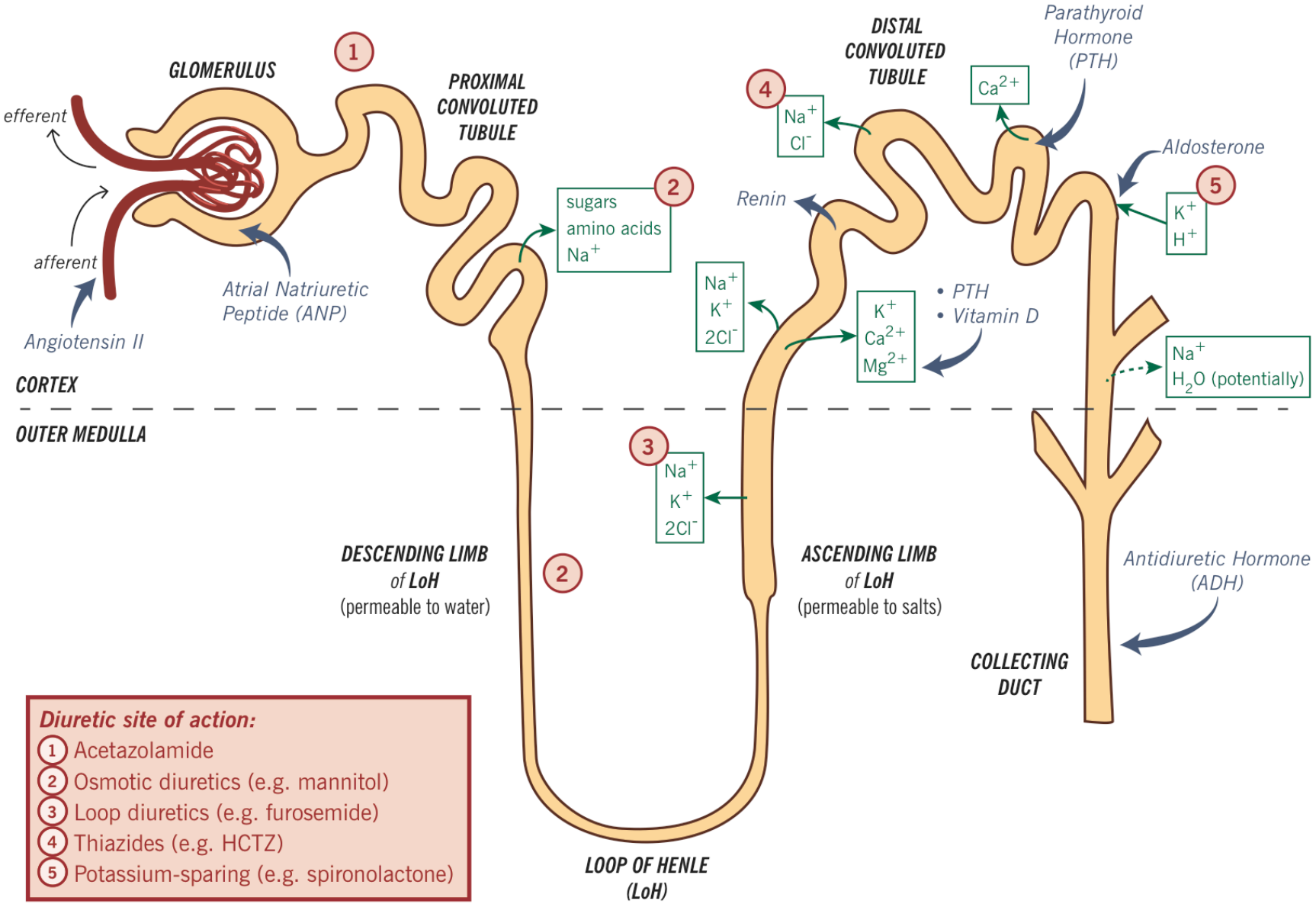
- Salt restriction (2g of Na = 5 g NaCL = ½ table spoon)
- Fluid restriction (1.5 to 2 L / day) about 8 cups
- Daily weight (tailored therapy)
- Gradual exertion programs (rehabilitation program)


5.0
0:00


Cardioregulatory center
Glossopharyngeal and vagal afferents from high-pressure baroreceptors
Sympathetic trunk
Sympathetic ganglia
AVP
Sympathetic nerves
Aldosterone
Angiotensin II release
Peripheral vasoconstriction
↓Solute-free water excretion
↓Sodium excretion

	Starting dose	Target dose
ACE-I		
Captopril[a]	6.25 mg *t.i.d.*	50 mg *t.i.d.*
Enalapril	2.5 mg *b.i.d.*	10—20 mg *b.i.d.*
Lisinopril[b]	2.5—5 mg *o.d.*	20—35 mg *o.d.*
Ramipril	2.5 mg *b.i.d.*	5 mg *b.i.d.*
Trandolapril[a]	0.5 mg *o.d.*	4 mg *o.d.*
ARNI		
Sacubitril/valsartan	49/51 mg *b.i.d.*[c]	97/103 mg *b.i.d.*
Beta-blockers		
Bisoprolol	1.25 mg *o.d.*	10 mg *o.d.*
Carvedilol	3.125 mg *b.i.d.*	25 mg *b.i.d.*[e]
Metoprolol succinate (CR/XL)	12.5—25 mg *o.d.*	200 mg *o.d.*
Nebivolol[d]	1.25 mg *o.d.*	10 mg *o.d.*
MRA		
Eplerenone	25 mg *o.d.*	50 mg *o.d.*
Spironolactone	25 mg *o.d.*[f]	50 mg *o.d.*
SGLT2 inhibitor		
Dapagliflozin	10 mg *o.d.*	10 mg *o.d.*
Empagliflozin	10 mg *o.d.*	10 mg *o.d.*
Other agents		
Candesartan	4 mg *o.d.*	32 mg *o.d.*
Losartan	50 mg *o.d.*	150 mg *o.d.*
Valsartan	40 mg *b.i.d.*	160 mg *b.i.d.*
Ivabradine	5 mg *b.i.d.*	7.5 mg *b.i.d.*
Vericiguat	2.5 mg *o.d.*	10 mg *o.d.*
Digoxin	62.5 µg *o.d.*	250 µg *o.d.*
Hydralazine/ Isosorbide dinitrate	37.5 mg *t.i.d.*/20 mg *t.i.d.*	75 mg *t.i.d.*/40 mg *t.i.d.*

Hormones Acting on the Nephron / Diuretics and Their Site of Action



Diuretic site of action:

1. Acetazolamide
2. Osmotic diuretics (e.g. mannitol)
3. Loop diuretics (e.g. furosemide)
4. Thiazides (e.g. HCTZ)
5. Potassium-sparing (e.g. spironolactone)

Diuretic Therapy

- The most effective symptomatic relief
- Mild symptoms
  - HCTZ
    Thiazides are ineffective with GFR < 30/min
  - Chlorthalidone
  - Metolazone
  - Block Na reabsorbtion in loop of henle and distal convoluted tubules

Diuretics (cont.)

- **Side Effects**
  - Pre-renal azotemia
  - Skin rashes
  - Neutropenia
  - Thrombocytopenia
  - Hyperglycemia
  - ↑ Uric Acid
  - Hepatic dysfunction

Diuretics (cont.)

- **More severe heart failure → loop diuretics**
  - **Lasix** (20 – 320 mg QD), Furosemide
  - **Bumex** (Bumetanide 1-8mg)
  - **Torsemide** (20-200mg)
- **Mechanism of action**: Inhibit chloride reabsortion in ascending limb of loop of Henle results in natriuresis, kaliuresis and metabolic alkalosis
- **Adverse reaction:**
  - Pre-renal azotemia
  - Hypokalemia
  - Skin rash
  - Ototoxicity

K^+ Sparing Agents

- **Triamterene & amiloride** – acts on distal tubules to ↓ K secretion
- **Spironolactone/ Eplerenone** (Aldosterone inhibitor)

Evidence suggests that it may improve survival in CHF patients due to the effect on renin-angiotensin-aldosterone system with subsequent effect on myocardial remodeling and fibrosis

Inhibitors of renin-angiotensin-aldosterone system

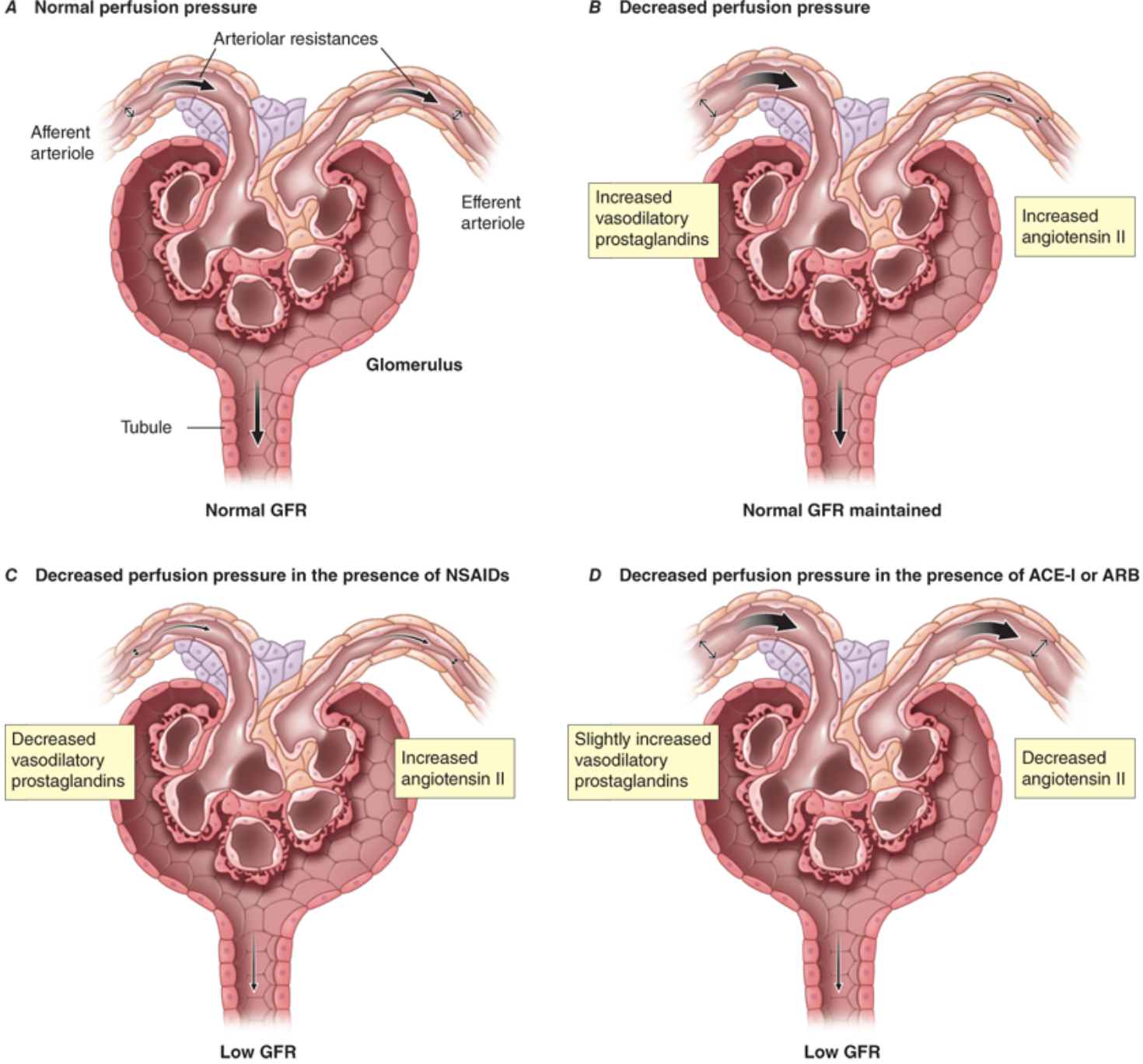
- Renin-angiotensin-aldosterone system *activation is early in the course of heart failure and plays an important role in the progression of the syndrome*
- Angiotensin converting enzyme inhibitors
- Angiotensin receptors blockers
- Spironolactone

Angiotensin Converting Enzyme Inhibitors

- They block the R-A-A system by inhibiting the conversion of angiotensin I to angiotensin II → vasodilation and ↓ Na retention
- ↓ Bradykinin degradation ↑ its level → ↑ PG secretion & nitric oxide
- ACE Inhibitors were found to improve survival in CHF patients
  - Delay onset & progression of HF in pts with asymptomatic LV dysfunction
  - ↓ cardiac remodeling

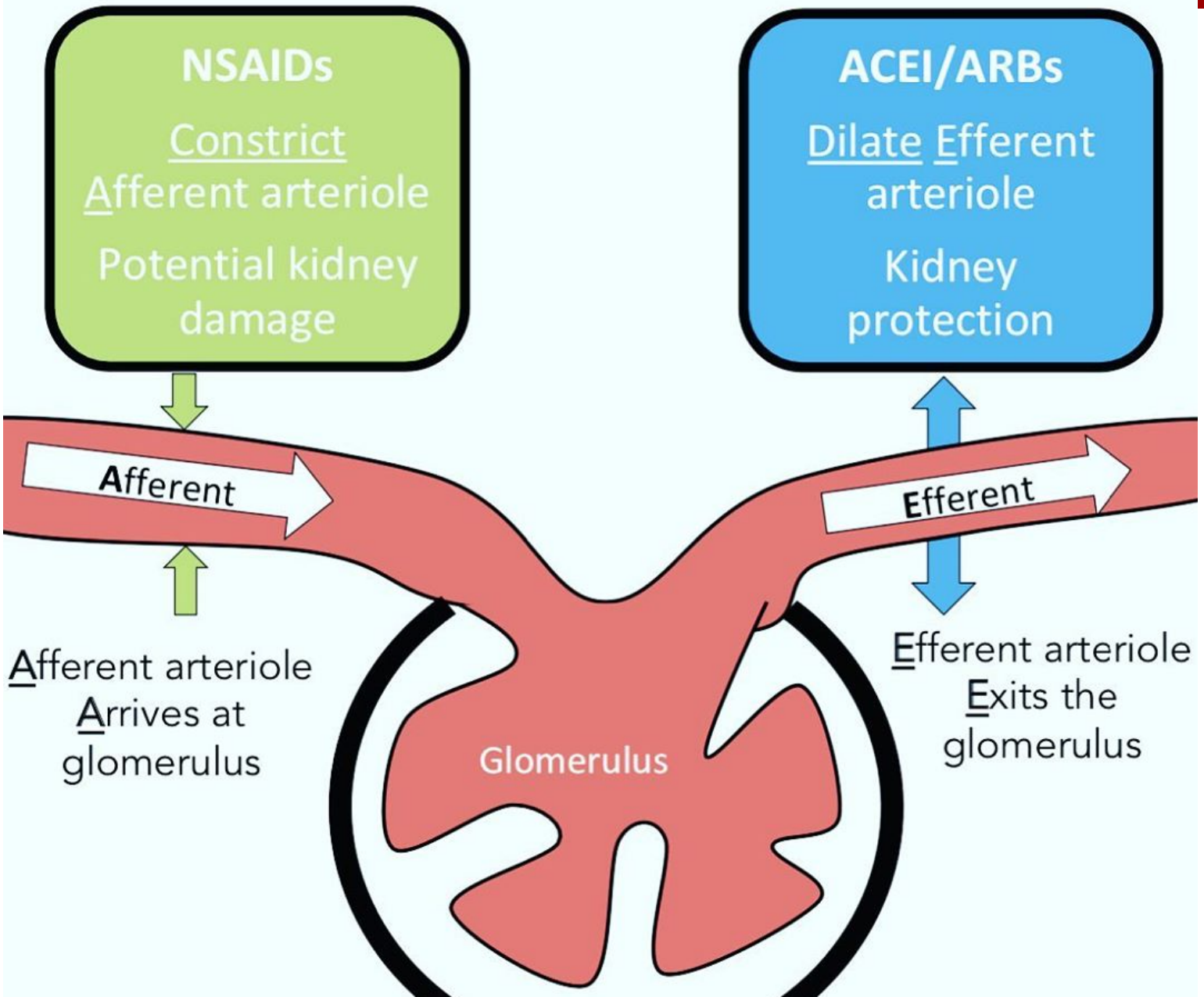
Side effects of ACE inhibitors

- Angioedema
- Hypotension
- Renal insuffiency
- Rash
- Cough



Source: D. L. Kasper, A. S. Fauci, S. L. Hauser, D. L. Longo, J. L. Jameson, J. Loscalzo: Harrison's Principles of Internal Medicine, 19th Edition.
www.accessmedicine.com

NSAID vs ACEI/ARB on Kidneys



Angiotensin II receptor blockers

- Has comparable effect to ACE I
- Can be used in certain conditions when ACE I are contraindicated (angioneurotic edema, cough)

Angiotensin Receptor- Neprilysin inhibitor (ARNi)

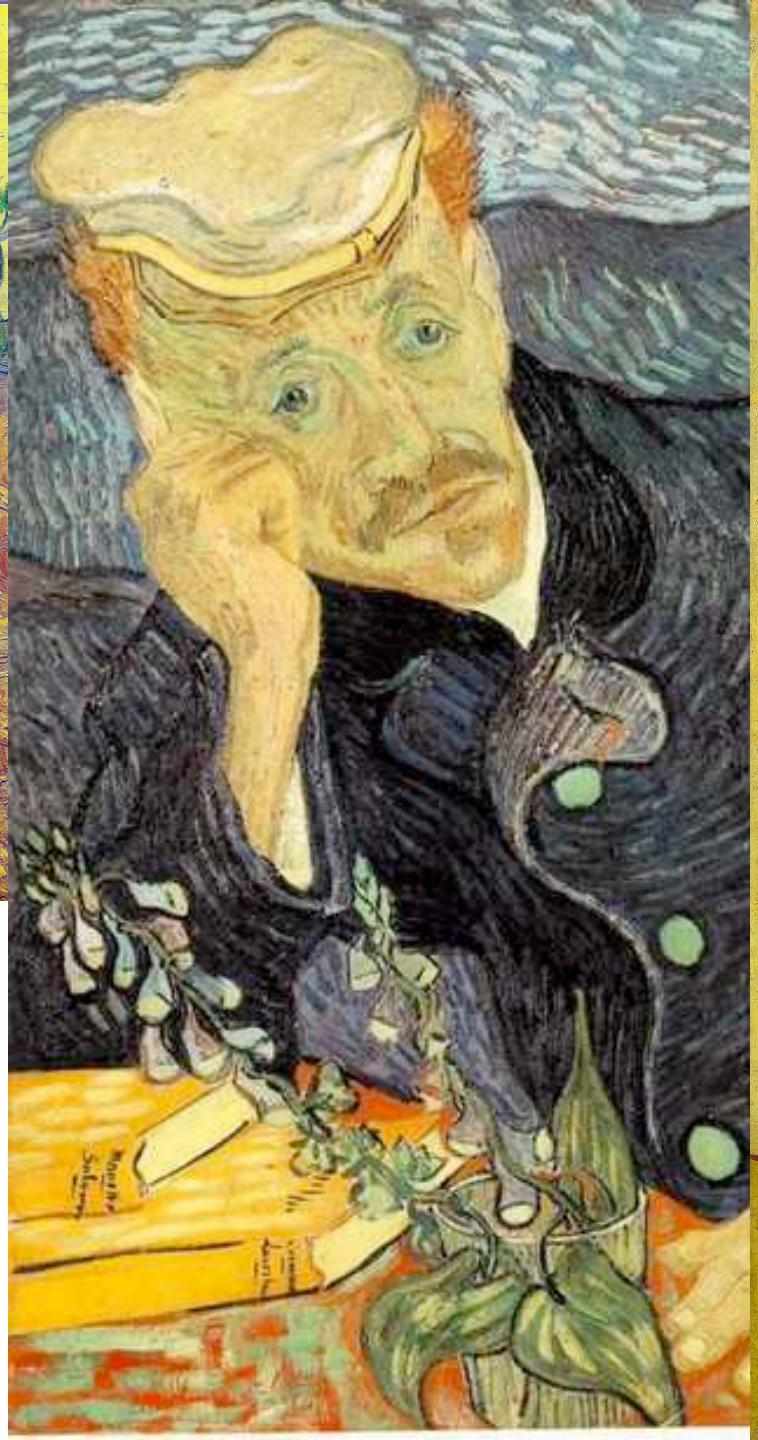
- Recent FDA approval (2015)
- The only product available (valsartan/sacubitril)
- Valsartan = ARB
- Sacubitril = prodrug for sacubitrilat
  Inhibit neprilysin which breakdown the vasoactive peptides.
- Used if patient LVEF <= 35% and still symptomatic with ACE/ARB
- In this specific group of patients it improves M&M.

I_f- Channel blocker

- Ivabradine ; Inhibit the Na inflow during the SA nodel action potential phase 4.
- Decrease the heart rate.
- Only use it if HR not controlled by BB and remains > 70 bpm and the patient has sinus rhythm.
- In this group if patients it improve M&M.

Digitalis Glycosides (Digoxin, Digitoxin)

- The role of digitalis has declined somewhat because of safety concern
- Studies have shown that digitals does not affect mortality in CHF patients but causes significant:
  - Reduction in hospitalization
  - Reduction in symptoms of HF




VINCENT

Digitalis (cont.)
Mechanism of Action

- +ve inotropic effect by ↑ intracellular Ca & enhancing actin-myosin cross bride formation (binds to the Na-K ATPase → inhibits Na pump → ↑ intracellular Na → ↑ Na-Ca exchange
- Vagotonic effect
- Arrhythmogenic effect

Digitalis Toxicity

- Narrow therapeutic to toxic ratio
- Non cardiac manifestations
  - Anorexia,
  - Nausea, vomiting,
  - Headache,
  - Xanthopsia sotoma,
  - Disorientation

Digitalis Toxicity

- **Cardiac manifestations**
  - Sinus bradycardia and arrest
  - A/V block (usually 2nd degree)
  - Atrial tachycardia with A/V Block
  - Development of junctional rhythm in patients with a fib
  - PVC's, VT/ V fib (bi-directional VT)

Digitalis Toxicity Treatment

- Hold the medications.
- Observation.
- In case of A/V block or severe bradycardia → atropine followed by temporary PM if needed.
- In life threatening arrhythmia → digoxin-specific fab antibodies.
- Lidocaine and phenytoin could be used – try to avoid D/C cardioversion in non life threatening arrhythmia.

β Blockers

- Has been traditionally contraindicated in pts with CHF
- Now they are the main stay in treatment on CHF & may be the only medication that shows substantial improvement in LV function
- In addition to improved LV function multiple studies show improved survival
- The only contraindication is severe decompensated CHF

Vasodilators

- **Reduction of afterload** by arteriolar vasodilatation (hydralazin) → reduce LVEDP, O_2 consumption,improve myocardial perfusion, ↑ stroke volume and COP
- **Reduction of preload** By venous dilation (Nitrate) → ↓ the venous return →↓ the load on both ventricles.
- Usually the maximum benefit is achieved by using agents with both action.

SGLT 2 inhibitors (originally for diabetes)

- Work on the proximal convoluted tubule, by inhibiting reabsorption of the glucose.
- Reduce the blood glucose, systolic and diastolic blood pressure and work as diuretic.
- Mortality benefit in patients with heart failure HFrEF, HFmrEF and HFpEF.
- Dabagliflozin, Empagliflozin
- UTIs, DKA and Osteoporosis are the major side effects.

To reduce mortality - for all patients

ACE-I/ARNI | BB | MRA | SGLT2i

To reduce HF hospitalization/mortality - for selected patients

Volume overload

Diuretics

SR with LBBB ≥ 150 ms

CRT-P/D

SR with LBBB 130–149 ms or non LBBB≥ 150 ms

CRT-P/D

Ischaemic aetiology

ICD

Non-ischaemic aetiology

ICD

Atrial fibrillation

Anticoagulation

Atrial fibrillation

Digoxin | PVI

Coronary artery disease

CABG

Iron deficiency

Ferric carboxymaltose

Aortic stenosis

SAVR/TAVI

Mitral regurgitation

TEE MV Repair

Heart rate SR>70 bpm

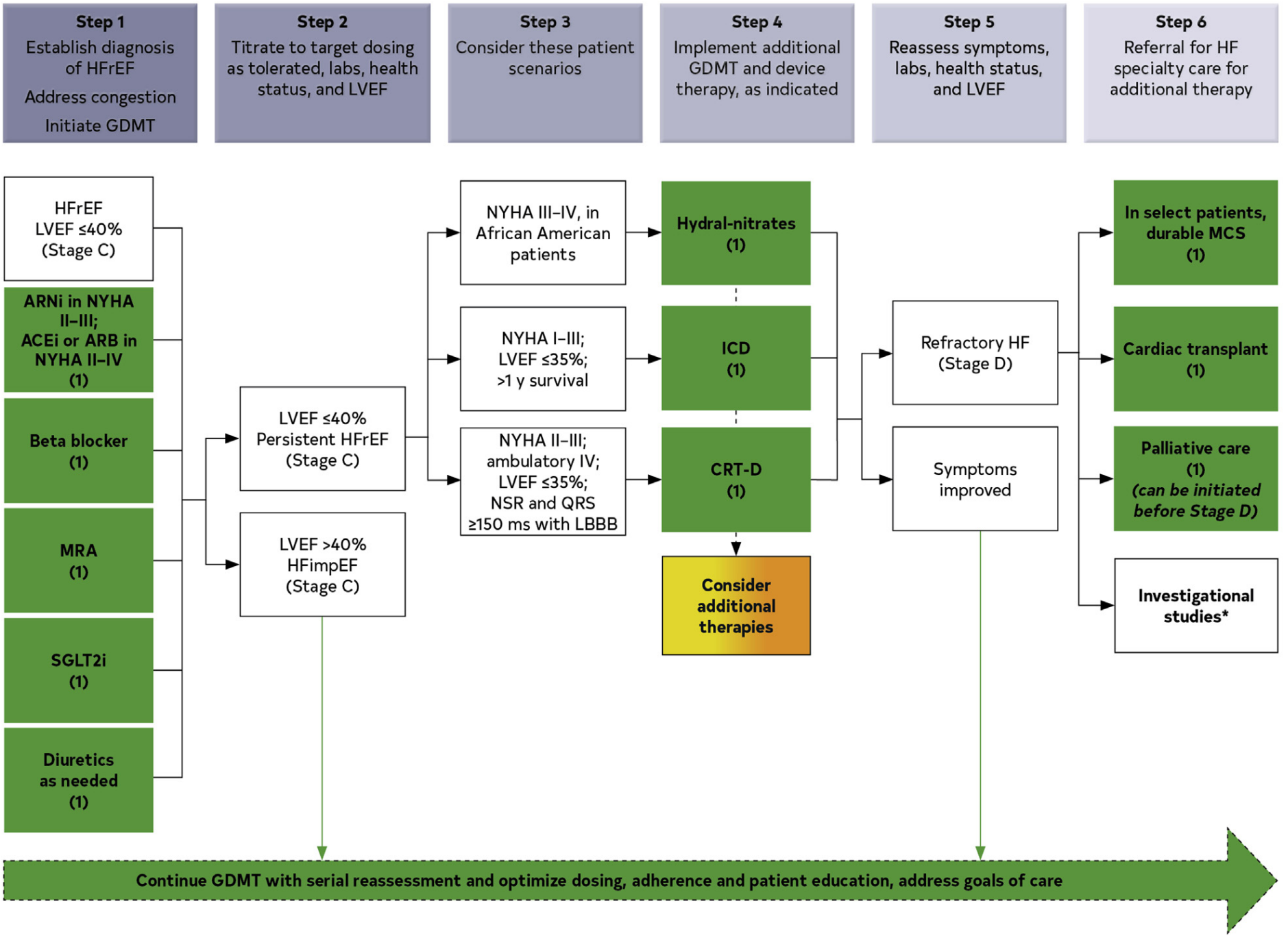
Ivabradine

Black Race

Hydralazine/ISDN

ACE-I/ARNI intolerance

ARB


Step 1
Establish diagnosis of HFrEF
Address congestion
Initiate GDMT
Step 2
Titrate to target dosing as tolerated, labs, health status, and LVEF
Step 3
Consider these patient scenarios
Step 4
Implement additional GDMT and device therapy, as indicated
Step 5
Reassess symptoms, labs, health status, and LVEF
Step 6
Referral for HF specialty care for additional therapy
HFrEF LVEF ≤40% (Stage C)
ARNi in NYHA II–III; ACEi or ARB in NYHA II–IV (1)
Beta blocker (1)
MRA (1)
SGLT2i (1)
Diuretics as needed (1)
LVEF ≤40% Persistent HFrEF (Stage C)
LVEF >40% HFimpEF (Stage C)
NYHA III–IV, in African American patients
NYHA I–III; LVEF ≤35%; >1 y survival
NYHA II–III; ambulatory IV; LVEF ≤35%; NSR and QRS ≥150 ms with LBBB
Hydral-nitrates (1)
ICD (1)
CRT-D (1)
Consider additional therapies
Refractory HF (Stage D)
Symptoms improved
In select patients, durable MCS (1)
Cardiac transplant (1)
Palliative care (1) (can be initiated before Stage D)
Investigational studies*
Continue GDMT with serial reassessment and optimize dosing, adherence and patient education, address goals of care

Positive inotropic agents

- These are the drugs that improve myocardial contractility (β adrenergic agonists, dopaminergic agents, phosphodiesterase inhibitors),

  Dopamine, Dobutamine, Milrinone, Amrinone

- Several studies showed ↑ mortality with oral inotropic agents
- So the only use for them now is in acute sittings as cardiogenic shock

Anticoagulation (Warfarin)/ NOAC

- Atrial fibrillation
- H/o embolic episodes
- Left ventricular apical thrombus

Antiarrhythmics

- Most common cause of SCD in these patients is ventricular tachyarrhythmia

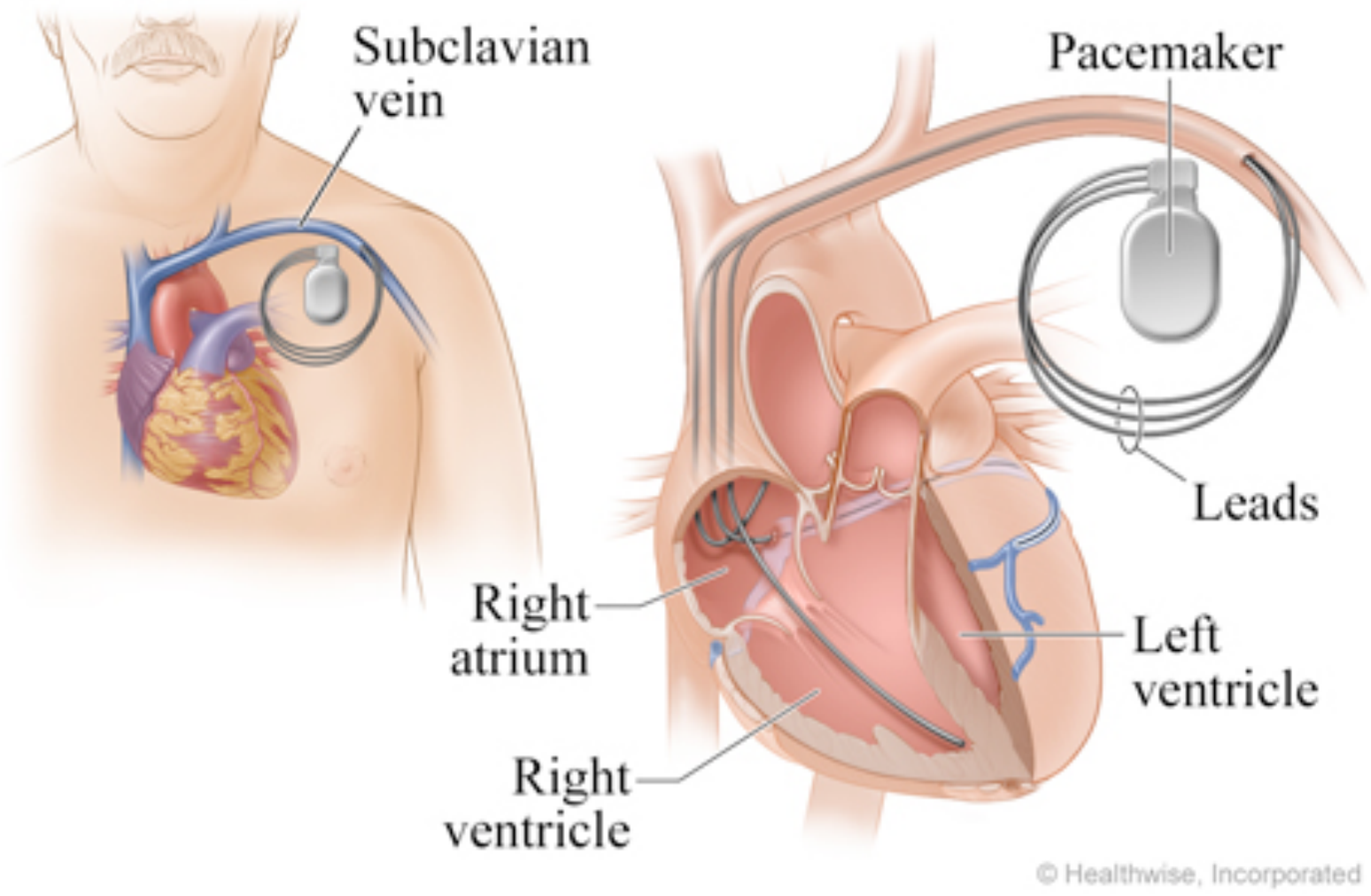
- Patients with h/o sustained VT or SCD → ICD implant

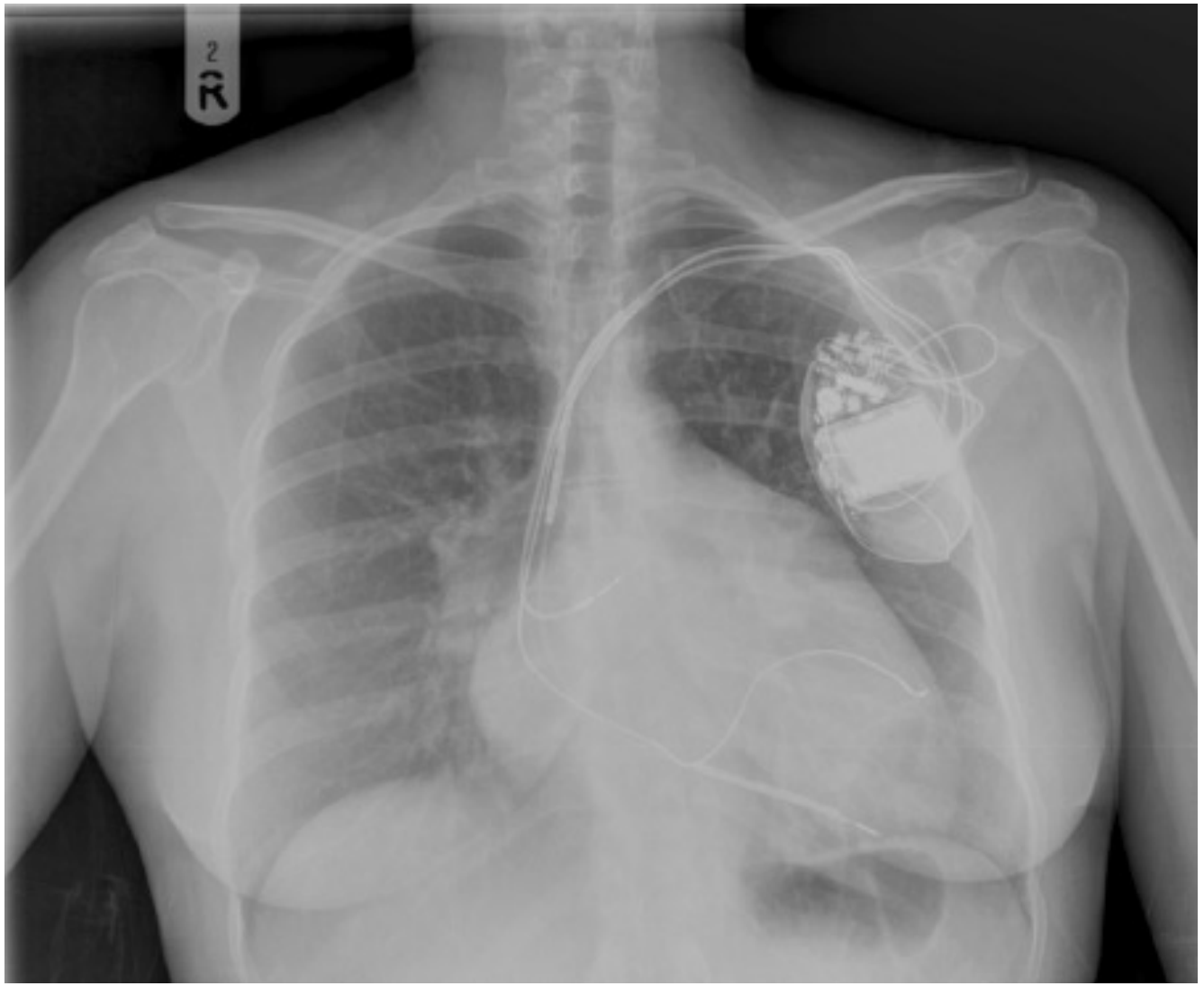
Antiarrhythmics (cont.)

- Patients with non sustained ventricular tachycardia
  - Correction of electrolytes and acid base imbalance.

Biventricular Pacing

- **Biventricular pacing** (only in patient with Wide QRS complexes & CHF).


Subclavian vein
Pacemaker
Leads
Right atrium
Left ventricle
Right ventricle
© Healthwise, Incorporated


2
R

Assisting devices

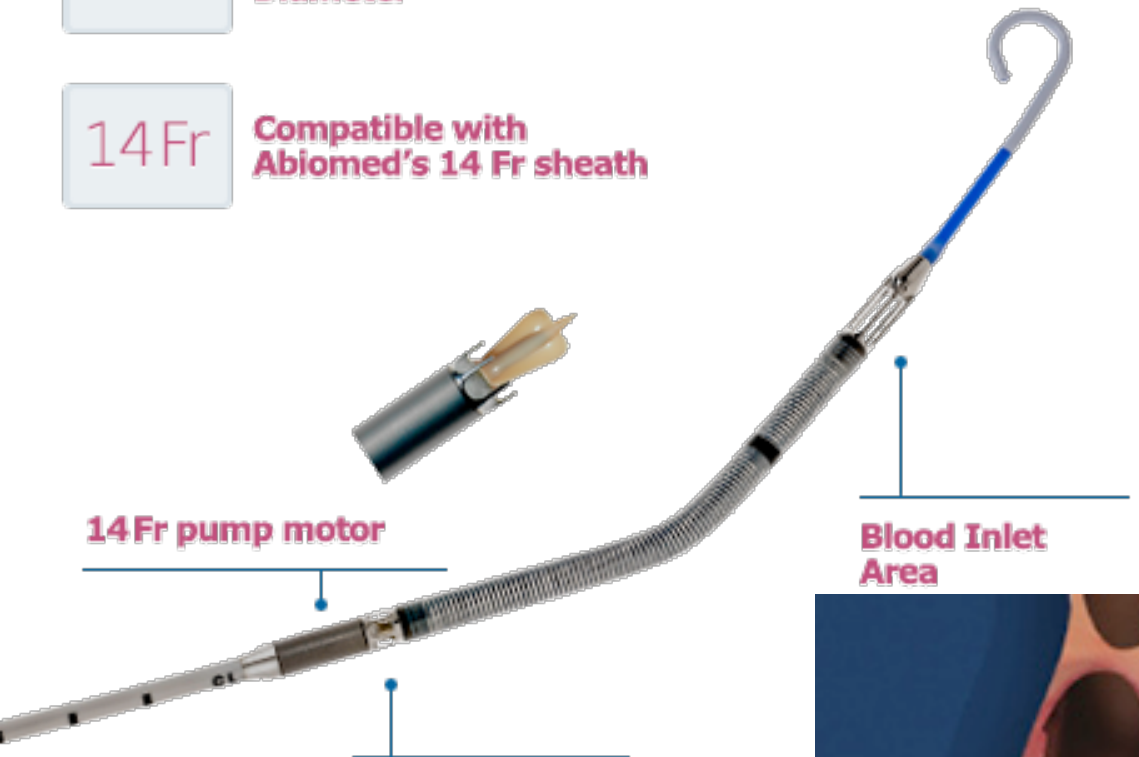
- Temporary ventricular assist devices.
- Implantable ventricular assist devices.

9Fr

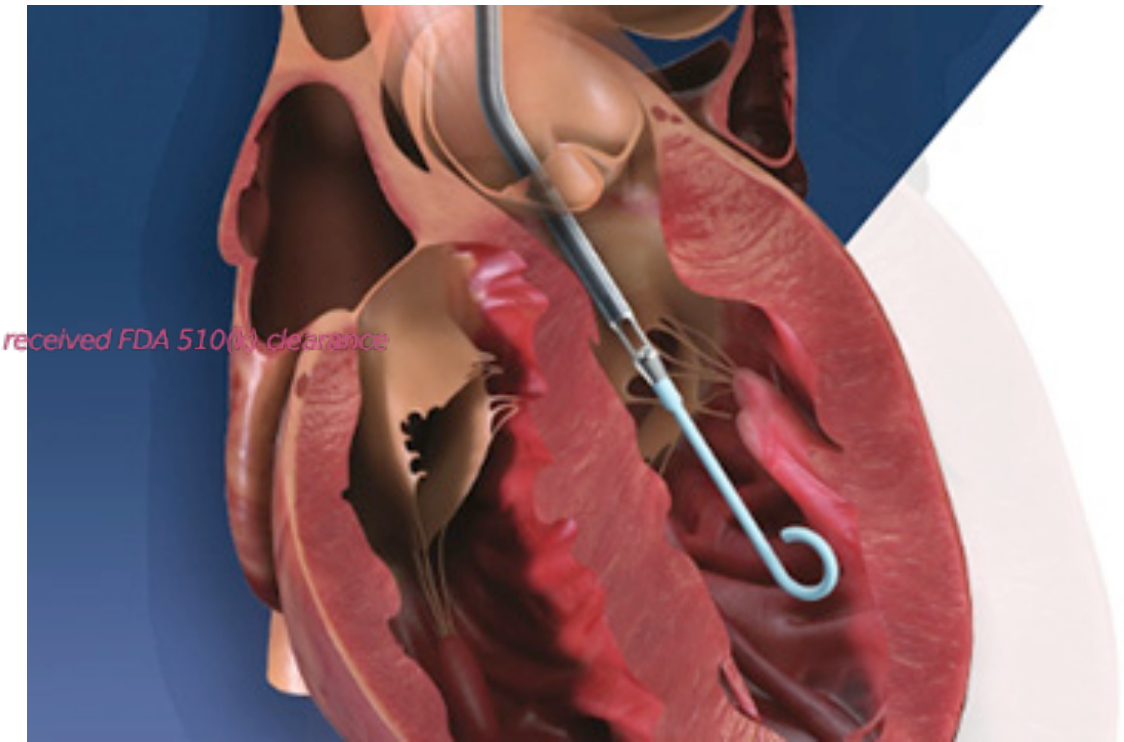
Catheter Diameter

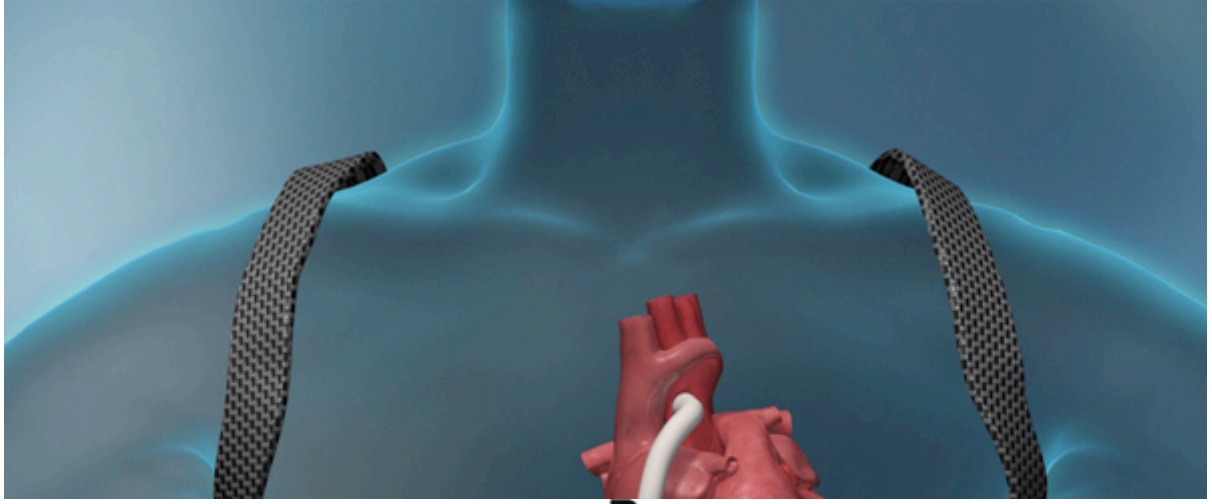
14Fr

Compatible with Abiomed's 14 Fr sheath



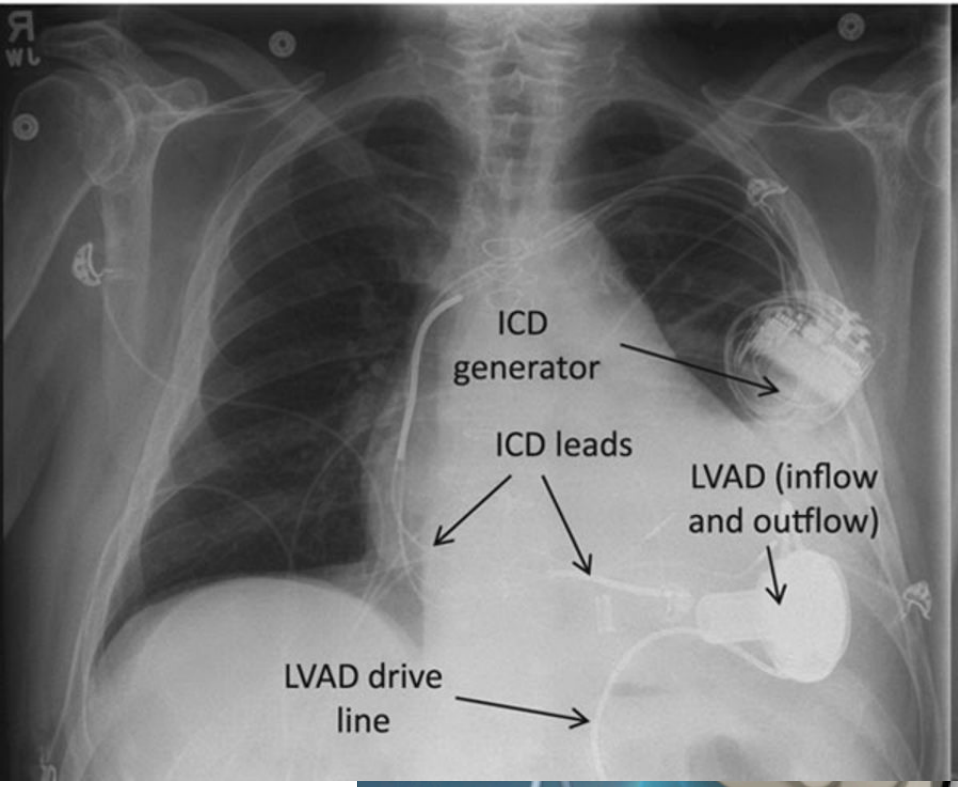
September 2012, received FDA 510(k) clearance

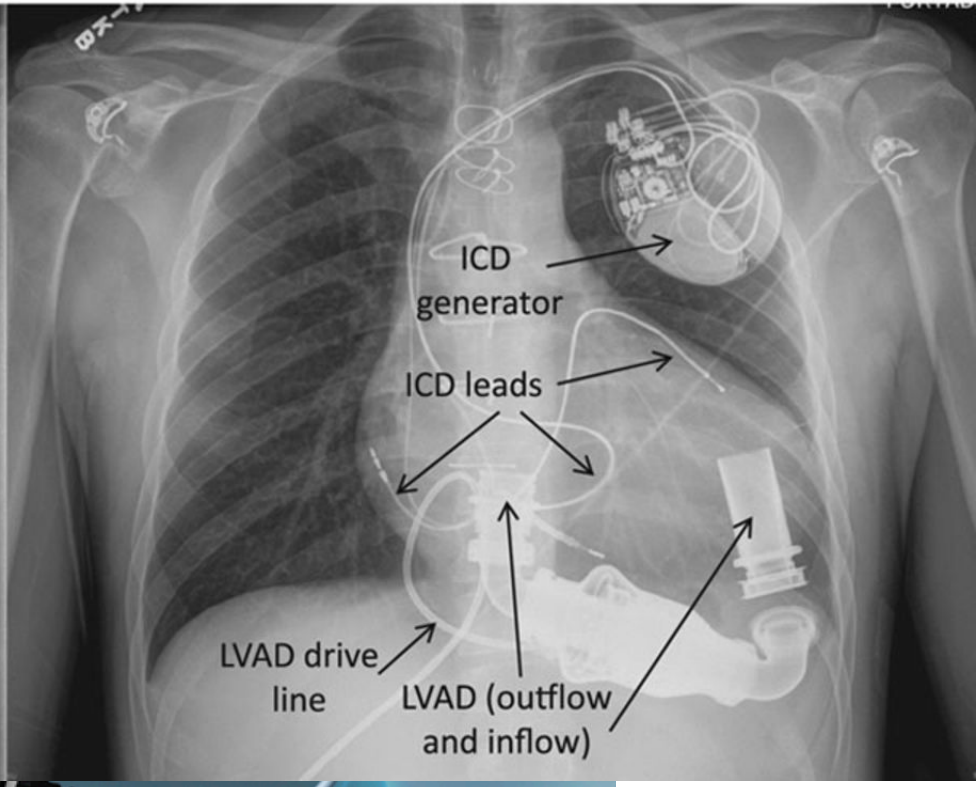




A

B


ICD
generator
ICD leads
LVAD (inflow
and outflow)
LVAD drive
line

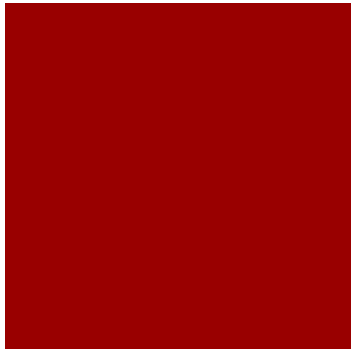

ICD
generator
ICD leads
LVAD drive
line
LVAD (outflow
and inflow)

Cardiac Transplant

- It has become more widely used since the advances in immunosuppressive treatment.
- Survival rate:
  - 1 year 80% - 90%
  - 5 years 70%

Prognosis

- Annual mortality rate depends on patients symptoms and LV function.
- 5% in patients with mild symptoms and mild ↓ in LV function.
- 30% to 50% in patient with advances LV dysfunction and severe symptoms.
- 40% – 50% of death is due to SCD.



Good luck,
Questions ….