

A red ECG line graphic runs horizontally across the middle of the slide. On the left side, it features a prominent, tall, narrow peak that resembles a stylized tower or spire. The rest of the line consists of several smaller, regular heartbeats before and after the tall peak.

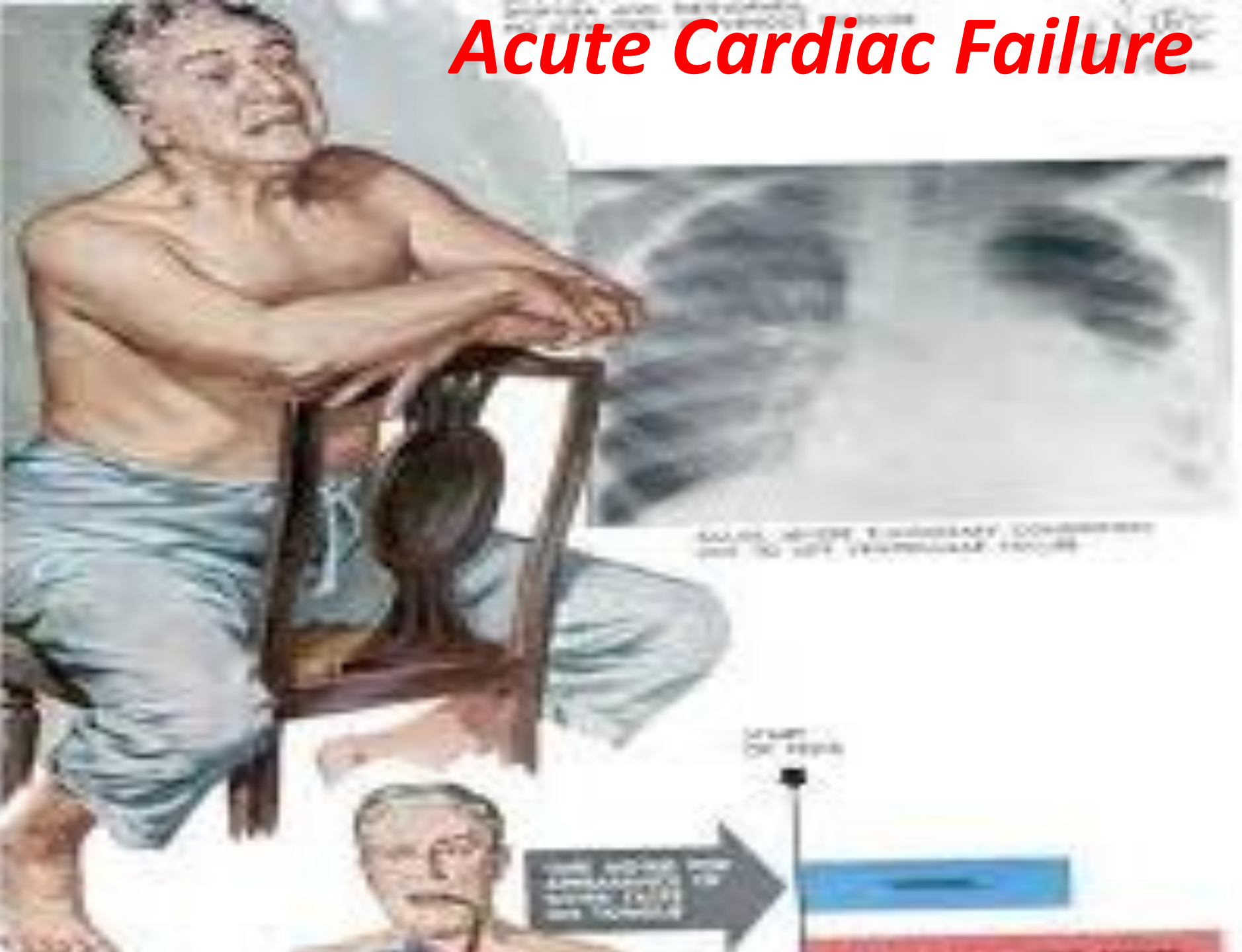
HEART FAILURE **UPDATE 2020**

Cardiogenic shock

VIRTUAL
CONFERENCE

A small, stylized grey arc logo, resembling a partial circle or a stylized letter 'C', located in the bottom right corner of the slide.

Acute Cardiac Failure



Heart failure in the United States

- Is the primary cause of hospitalization in the elderly.
- An estimated one in eight deaths is from heart failure (about 309,000 deaths each year)
- Accounts for 8.5% of cardiovascular-related deaths
- Approximately 960,000 new cases are diagnosed each year

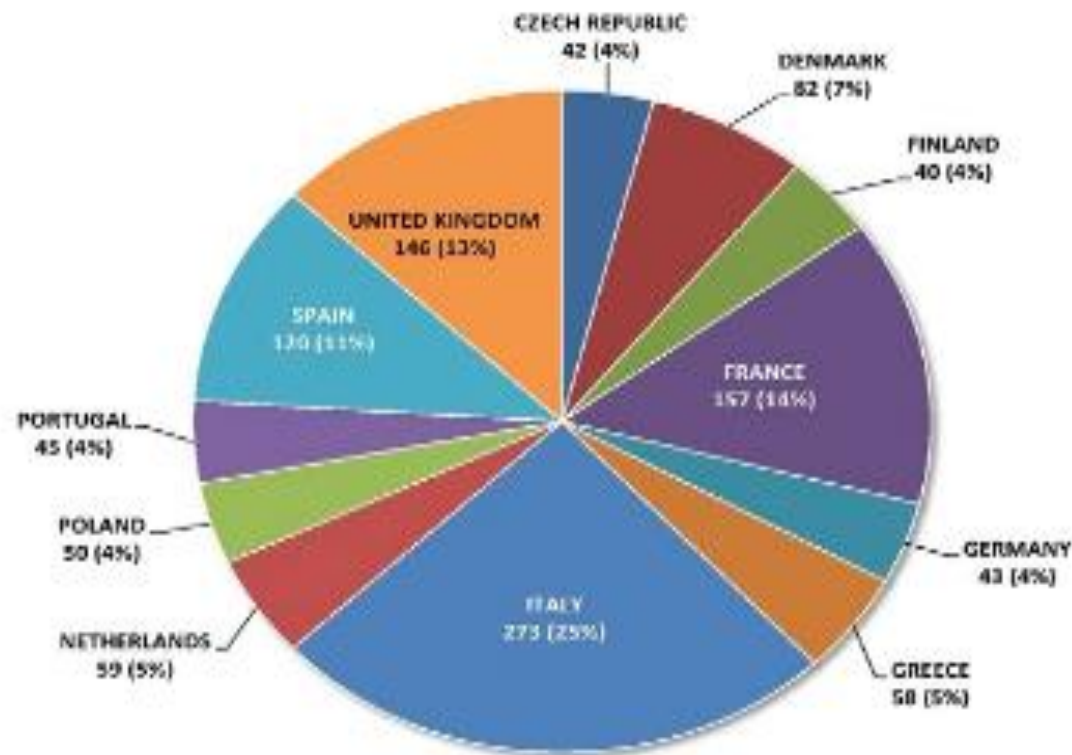
Heart failure in USA

- **Rehospitalization rates during the 6 months following discharge are 50%.**
- **2019, the estimated cost of heart failure in USA was \$30.7 billion (68% of which were direct medical costs);**
- **2030, the total cost is projected to rise to \$69.7 billion, a nearly 127% increase.**

International statistics

- Heart failure is a **worldwide problem**.
- The most common causes are:
- **Ischemic cardiomyopathy**
- **Including Chagas disease and**
- **Valvular cardiomyopathy.**
- Increased rates of diabetes and hypertension.

EURO HEART FAILURE 2020



Phenotypes



T2DM



CRS/Stalled



Wet
Hypertensive



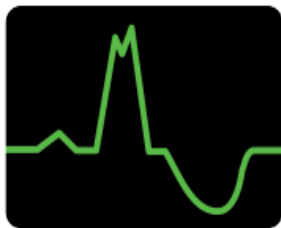
Elderly



Afib



High
Heart Rate



LBBB



HFpEF



HFmrEF

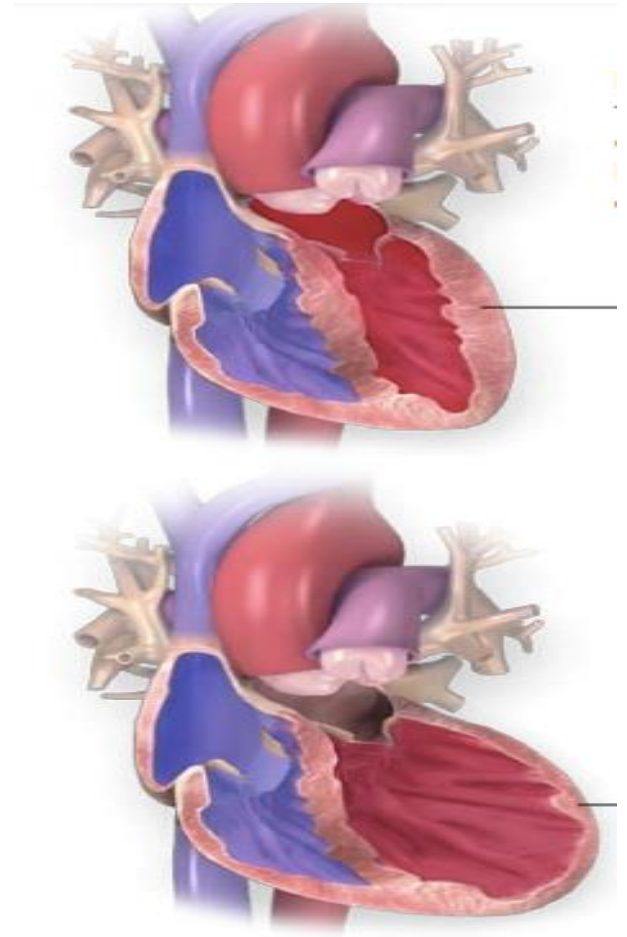


De novo

Acute Cardiac Failure

Definition

- Develops when the heart fails to pump blood at a rate commensurate with the requirements of the metabolizing tissues OR
- Is able to do so only with an elevated diastolic filling pressure.



Acute Cardiac Failure

- To maintain the pumping function of the heart, **compensatory mechanisms:**
 - Increase blood volume
 - Cardiac filling pressure
 - Heart rate, and
 - Cardiac muscle mass.
- However, despite these mechanisms, there is a progressive decline in the ability of the heart to contract and relax, resulting in worsening heart failure.

The increase in hospital admissions is due to different reasons:

- *“The aging of the population -which carries with it an increase in comorbidities that increase the incidence of this pathology-;*
- The increased survival of acute heart failure that, receive better treatment, reduce mortality but can cause a chronic alteration of heart function.
- And to the decrease in mortality from heart failure it self in the short and medium term ”.

Signs and symptoms of heart failure include

- **Tachycardia** and
- Manifestations of **venous congestion (edema)**
- Low cardiac output (fatigue).
- Breathlessness is a cardinal symptom of left ventricular (LV) failure that may manifest with progressively increasing severity.

Heart failure

- Can be classified according to a variety of factors
- The New York Heart Association (NYHA)
- Classification for heart failure comprises four classes, based on the relationship between symptoms and the amount of effort required to provoke them,

New York Heart Association (NYHA)

- Class I. Patients **have no limitation** of physical activity
- Class II. have **slight limitation** of physical activity
- Class III **marked limitation** of physical activity
- Class IV pts **have symptoms even at rest** and are unable to carry on any physical activity without discomfort

ACC/AHA and Heart Failure Guidelines complement the NYHA classification to reflect ***the progression of disease and are divided into four stages,***

- **Stage A** patients are at **high risk for heart failure** but **have no structural heart disease** or symptoms
- **Stage B** pts **have structural heart disease** but have no symptoms of heart failure
- **Stage C** patients have **structural heart disease** and have **symptoms of heart failure**
- **Stage D** patients **have refractory heart failure** requiring **specialized interventions**

- The Usefulness of the MEESI Score for Risk Stratification of Patients With Acute Heart Failure at the Emergency Department





- The MEESSEI risk score successfully stratifies AHF patients at the ED according to the 30-day mortality risk,
- Potentially helping clinicians in the decision-making process for hospitalizing patients.

The main clinical impact? Meessi Scale

- The tool could help to better make the admission or discharge decision.
- The majority of discharges from the emergency department should correspond to low-risk patients (currently only half are) and
- Most of the admissions should correspond to patients at high risk (and currently only two thirds of patients have it).

ER



VIII World Congress in

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Emergency Room

- Why do we want to predict risk of hospitalization?

To intervene sooner

-  Mortality
-  Quality of Life
-  Cost
-  Days lost from work/life

Risk Prediction Scores
Monitoring tools

Diagnosis AHF

- Is based on **clinical assessment**,
- **Measurement of natriuretic peptides**, and
- Imaging modalities.

Simultaneously, emphasis

- should be given in rapidly identifying
- the underlying trigger of AHF and
- assessing severity of AHF, as well as
- in recognizing end-organ injuries.

Diagnosis HF

- **Tissue congestion and hypoperfusion are the two leading mechanisms of end-organ injury and dysfunction, which are associated with worse outcome in AHF**

Benefit of short time to treatment in AHF

- **The first hours of hospitalization are marked by a high risk for complications, including death, and represent a “golden moment” for intervention.**
- **Indeed, a high number of AHF die in the emergency department (ED) before ICU/cardiac care unit (CCU) admission.**
- **Earlier diagnosis, triage, and initiation of specific treatment for AHF are associated with reduced mortality as well as shorter lengths of hospital stay**

Initial management of AHF without cardiogenic shock

- The large majority of AHF patients are, however, hemodynamically stable, and the primary diagnostic work-up and **early treatment can therefore be initiated in the ER.**

Image & Laboratory in HF

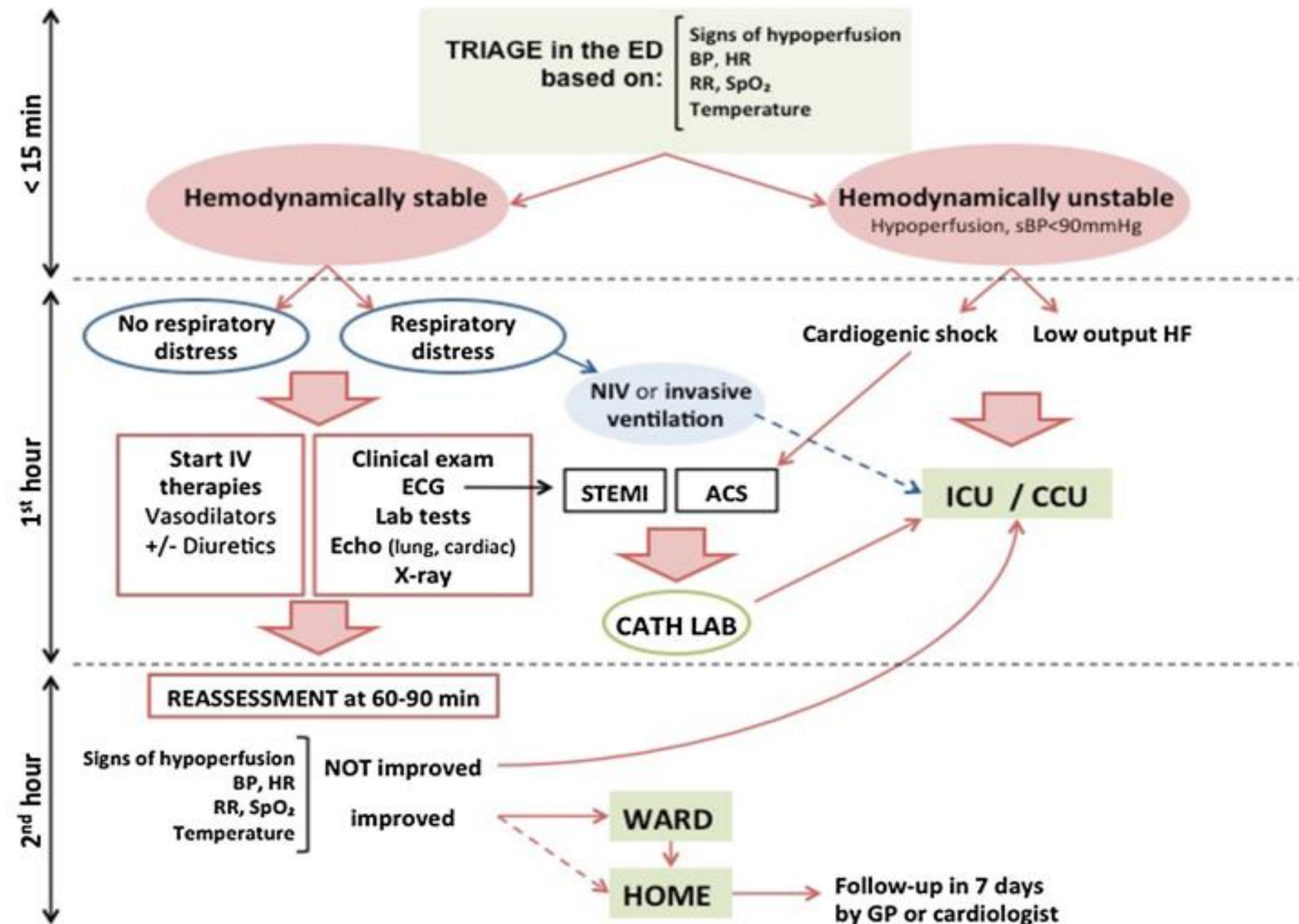
- Complete blood count (CBC)
- Electrolyte levels, and hepatorenal function.
- Chest xR and ECHO are recommended in the initial evaluation of patients with known or suspected HF.
- **B-type natriuretic peptide (BNP)** and N-terminal pro-B-type natriuretic peptide (NT-proBNP) levels ***can be useful in differentiating cardiac and noncardiac causes of dyspnea.***

What is the difference between BNP and NT Pro BNP?

- There is **no meaningful difference** between them.
- They reflect haemodynamic myocardial stress independent of the underlying pathology, thus
- **They are not specific for a distinct pathology such as heart failure but for cardiovascular diseases in general.**

In recent years

- **Biomarkers have emerged as important tools for diagnosis,**
- **Risk stratification and**
- **Therapeutic decision making in cardiovascular diseases**



Acute Heart Failure in ER

- **Consists of stabilizing the patient's clinical condition;**
- **Establishing the diagnosis, etiology, and precipitating factors; and**
- **Initiating therapies to provide rapid symptom relief and survival benefit.**

Surgical options for heart failure



- Revascularization procedures
- Electrophysiologic intervention
- Cardiac resynchronization therapy (CRT)
- implantable cardioverter-defibrillators (ICDs)
- Valve replacement or repair,
- Ventricular restoration,
- Heart transplantation, and
- Ventricular assist devices (VADs).



The goals of pharmacotherapy are

- **Increase survival and to prevent complications.**
- Along with oxygen,
- Medications with symptom relief include
- Diuretics, Digoxin, Inotropes, and Morphine.

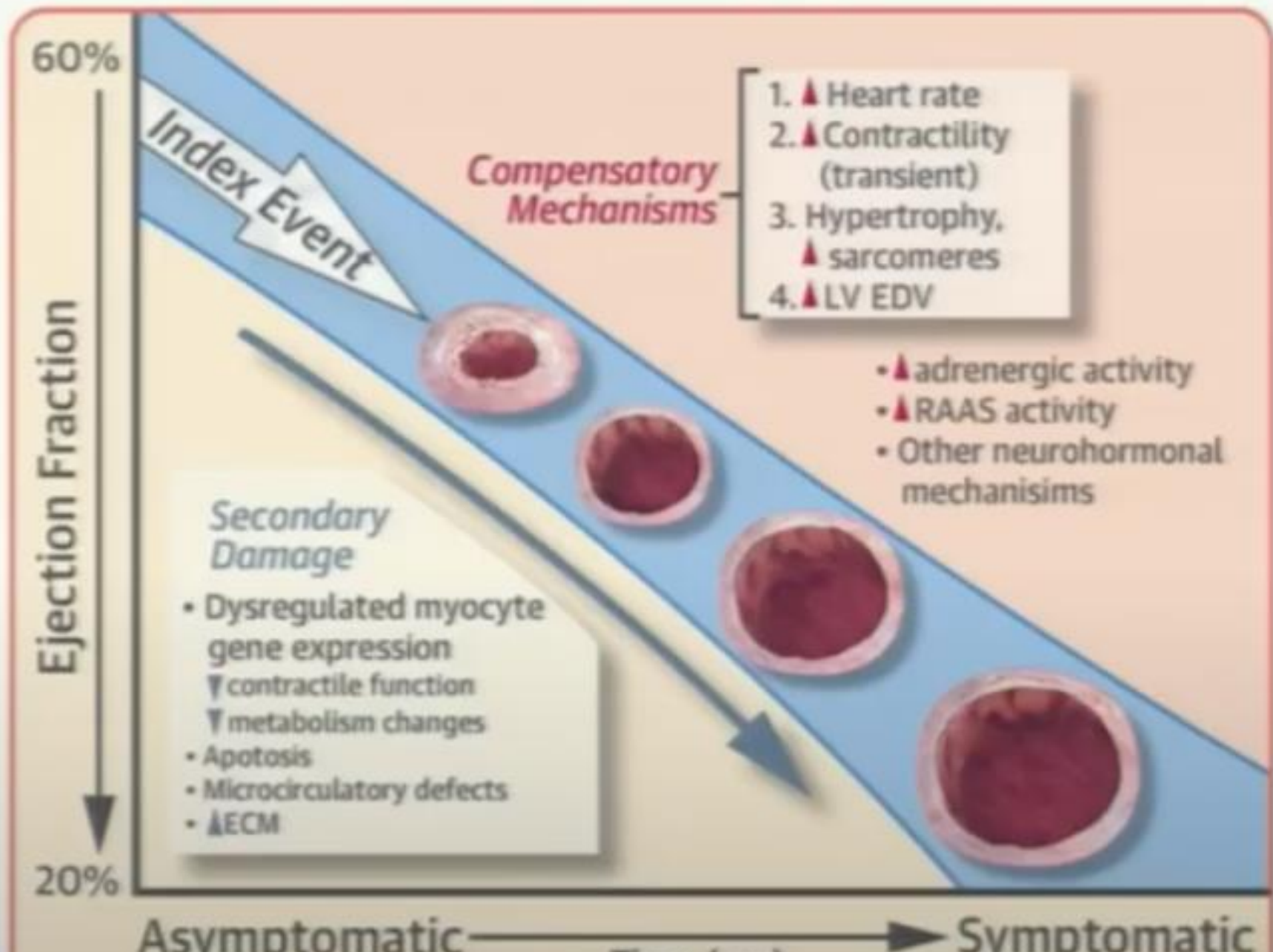
Clinical trials have shown a benefit in terms of mortality in this population.

- **HFr_{educe}EF** is defined by the clinical syndrome of HF with an **LVEF $\leq 40\%$** ,
- While **HFpEF** include patients with HF and LVEF **$\geq 50\%$**
- Recently, a **third group** has been characterized, HF with midrange ejection fraction (HFmr) (LVEF = 40% to 49%).

Guideline Similarities	 ACC/AHA	 ESH/ESC
Diagnosis testing	Transthoracic echocardiography for initial evaluation	
Prevention	cMRI to assess for myocardial scarring [ischemic etiology]	
HFrEF REDUCE	<ul style="list-style-type: none"> • Triple neurohormonal blockade (start ACEI [or ARB if ACEI intolerant] and BB, then add MRA if NYHA functional class II-IV and LVEF $\leq 35\%$) • Ivabradine for persistently symptomatic HF with sinus rhythm, LVEF $\leq 35\%$ and a resting heart rate ≥ 70 beats/min despite evidence-based dosing of beta-blocker (or maximally tolerated dose). 	
HFpEF PRESERVE	Diuretics for volume control, HBP management, relief of ischemia	
Implantable cardioverter-defibrillator (ICD) therapy	Primary prevention for (LVEF $\leq 35\%$, NYHA functional class II-III on GDMT) or (NYHA functional class II, LVEF $\leq 30\%$ on GDMT) and secondary prevention	
CRT Therapy	NYHA functional class II-IV HF, LVEF $\leq 35\%$, LBBB with QRS ≥ 150 ms	

Guideline Differences	 American College of Cardiology/American Heart Association (ACC/AHA)	 European Society of Hypertension/European Society of Cardiology (ESH/ESC)
Diagnosis testing	Cardiac MRI for myocardial scar or infiltrative process	Cardiac MRI (cMRI) for tissue characterization
HF with reduced ejection fraction (HFrEF)	<ul style="list-style-type: none"> • Specific ARB/ Beta blockers (BB) • ARNI for ACEI or ARB for chronic New York Heart Association (NYHA) functional class II/III symptoms 	<ul style="list-style-type: none"> • Class recommendation for ACEI inhibitors or ARB/BB • ARNI for persistent symptoms despite triple neurohormonal blockade • Broader indications for ivabradine (patients who cannot tolerate or have contraindication for BB)
HF with preserved ejection fraction (HFpEF) and diabetes, or high blood pressure (HBP)	<ul style="list-style-type: none"> • No recommendation • Guideline-directed medical therapy (GDMT), blood pressure goal < 130 mm Hg 	<ul style="list-style-type: none"> • Metformin for initial diabetic control • "Stepped care" approach with GDMT agents for hypertension
Cardiac resynchronization therapy (CRT) <ul style="list-style-type: none"> • Symptomatic HF, LVEF \leq 35% • QRS \geq 150 ms, non-left bundle branch block (LBBB) 	COR IIa recommendation for NYHA functional class III and IIb recommendations for NYHA functional class II	COR IIa recommendation
CRT Therapy <ul style="list-style-type: none"> • Symptomatic HF, left ventricular ejection fraction (LVEF) \leq 35% • LBBB with intermediate QRS duration 	COR IIb recommendation (QRS 120–149 ms)	COR I recommendation (QRS 130–149 ms)

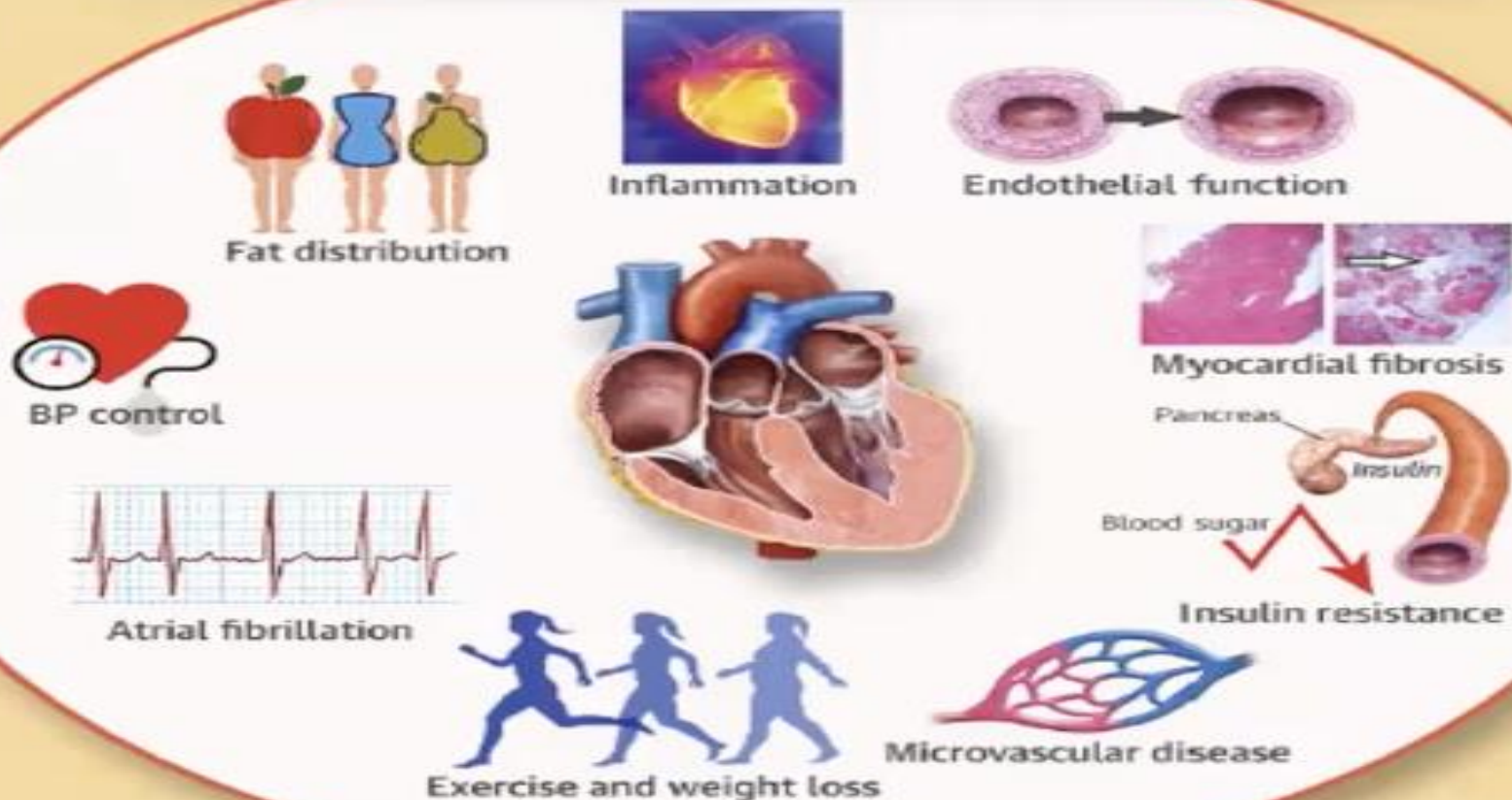
Natural History of HFrEF phenotype



Prevention of HF in Women

Targets and Strategies to Prevent Heart Failure with *Preserved* Ejection Fraction (HFpEF)

B



★ ED'S ★
1 SECOND
ACUPUNCTURE
TREATMENT

Ready?



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Medication Summary

- The goals of pharmacotherapy for heart failure are to:
 - **Reduce morbidity and to**
 - **Prevent complications.**

Treatment

- Early therapeutic considerations in AHF should be based on the status of congestion (“wet” vs. “dry”) and
- Systemic perfusion (“warm” vs. “cold”) based on clinical signs, laboratory tests, and ultrasound.

Indeed, in cases of warm/wet AHF,

- The management is based on **diuretics, vasodilators, and oxygen**, or on NIV in cases of APE.
- The use of **inotropes and vasopressors** should be restricted to maintain perfusion pressure in those AHF **patients with signs of hypoperfusion and/or shock**

Medication.....

- **Diuretics**, reduce edema by reduction of blood volume and venous pressures;
- **Vasodilators**, for preload and afterload reduction;
- **Digoxin**, which can cause a small increase in cardiac output;
- **Inotropic agents**, which help to restore organ perfusion and reduce congestion;
- **Anticoagulants**, to decrease the risk of thromboembolism;

Recommended dosing of intravenous vasodilators to treat acute heart failure

	Dosing	Main side effects	Other
Nitroglycerin	Start with 10–20 µg/min, increase up to 200 µg/min	Hypotension, headache	Tolerance after continuous use
Isosorbide dinitrate	Start with 1 mg/h, increase up to 10 mg/h	Hypotension, headache	Tolerance after continuous use
Nitroprusside	Start with 0.3 µg/kg/min and increase up to 5 µg/kg/min	Hypotension, Methemoglobinemia	Light sensitive
Nesiritide	Bolus 2 µg/kg + infusion 0.01 µg/kg/min	Hypotension	
Clevidipine	2.0 mg/h for 3 min, double every 3 min up to 32.0 mg/h	Hypotension	Made in fat emulsion

Medication.....

- **Beta-blockers**, for neurohormonal modification, left ventricular ejection fraction (LVEF) improvement, arrhythmia prevention, and ventricular rate control;
- **Angiotensin-converting enzyme inhibitors (ACEIs)**, for neurohormonal modification, vasodilatation, and LVEF improvement;
- **Angiotensin II receptor blockers (ARBs)**, also for neurohormonal modification, vasodilatation, and LVEF improvement; and
- **Analgesics**, for pain management.

Medication.....

- **Ivabradine**, inhibitor is available in USA.
- It blocks the hyperpolarization-activated cyclic nucleotide-gated (HCN) channel **responsible for the cardiac pacemaker, which regulates heart rate without any effect on ventricular repolarization or myocardial contractility.**

Sacubitril/valsartan (Entresto)

- Angiotensin receptor-neprilysin inhibitor (ARNI),
- **To reduce the risk of cardiovascular death and hospitalization for heart failure** in patients with congestive heart failure (New York Heart Association [NYHA] class II-IV) and
- **Reduced ejection fraction.**

Drugs that can exacerbate heart failure should be avoided

- **Nonsteroidal anti-inflammatory drugs NSAIDs.**
- **can cause sodium retention and peripheral vasoconstriction,** and they can attenuate the efficacy and enhance the toxicity of diuretics and ACEIs.
- **Calcium channel blockers (CCBs) can worsen heart failure** and may increase the risk of cardiovascular events; only the vasoselective CCBs have been shown not to adversely affect survival.
- **Antiarrhythmic agents (except class III) can have** cardiodepressant effects and may promote arrhythmia;
- ***only amiodarone and dofetilide have been shown not to adversely affect survival.***

Beta-Blockers, Alpha Activity

- Inhibit the sympathomimetic nervous system and block alpha1-adrenergic vasoconstrictor activity.
- **Have moderate afterload reduction** properties and cause slight preload reduction.
- In addition to **decreasing mortality rates**,
- **Reduce** hospitalizations and the risk of sudden death;
- **Improve** LV function and exercise tolerance; and
- **Reduce** heart failure functional class.

Carvedilol (Coreg, Coreg CR)

- Is a nonselective beta- and alpha1-adrenergic blocker. It does not appear to have intrinsic sympathomimetic activity.
- Carvedilol at the target dose of 25 mg twice daily **has been shown to reduce mortality in clinical trials of heart failure patients with reduced ejection fraction.**

Beta-Blockers, Beta-1 Selective

- Certain beta-1 blockers are selective in blocking beta-1 adrenoreceptors.
- **Are used in heart failure to reduce heart rate and blood pressure.**

Beta-Blockers, Beta-1 Selective

- **Metoprolol** (Lopressor, Toprol XL)
- It inhibits beta2-receptors at higher doses. It does not have intrinsic sympathomimetic activity.
- The long-acting formulation (metoprolol succinate) at a target dose of **200 mg daily has been shown to reduce mortality in a clinical trial of patients with heart failure and low ejection fraction.**

Beta-Blockers, Beta-1 Selective

Bisoprolol (Zebeta)

- Bisoprolol is a highly selective beta1 adrenergic receptor blocker that
- **Decreases the automaticity of contractions.**
- **Bisoprolol at the target dose of 10 mg daily has been shown to reduce mortality in a clinical trial of patients with heart failure and reduced ejection fraction,**
- but it is not approved for use in heart failure in the United States.

Emergency Room



Cardiogenic shock

- **Is the most severe manifestation of AHF, accounting for < 5 % of AHF cases in the western world.**
- It is characterized by severe circulatory failure of cardiac cause, with hypotension and signs of organ hypoperfusion.
- The most common etiology of CS is ACS with or without mechanical complication (80 %),
- The other causes of CS include **severe decompensation of chronic heart failure, valvular disease, myocarditis, or even Tako-Tsubo syndrome**

Cardiogenic shock

- Although still **associated with poor prognosis**,
- **Survival has improved markedly** during the last 30–40 years, and
- **short-term mortality is around 40 %** in contemporary cohorts of CS

CARDIOGENIC SHOCK (CS)

Causes of CS

Organ dysfunction

Immediate actions

ECG → ACS?
+/- troponin

Echo: mechanical
complications

Clinical signs
Blood gas
Lung echo
X-ray

Oliguria
GFR↓

Hypoperfusion
high lactate

Respiratory distress

Acute kidney injury

Cath lab

Operating room

Non-invasive or
invasive ventilation

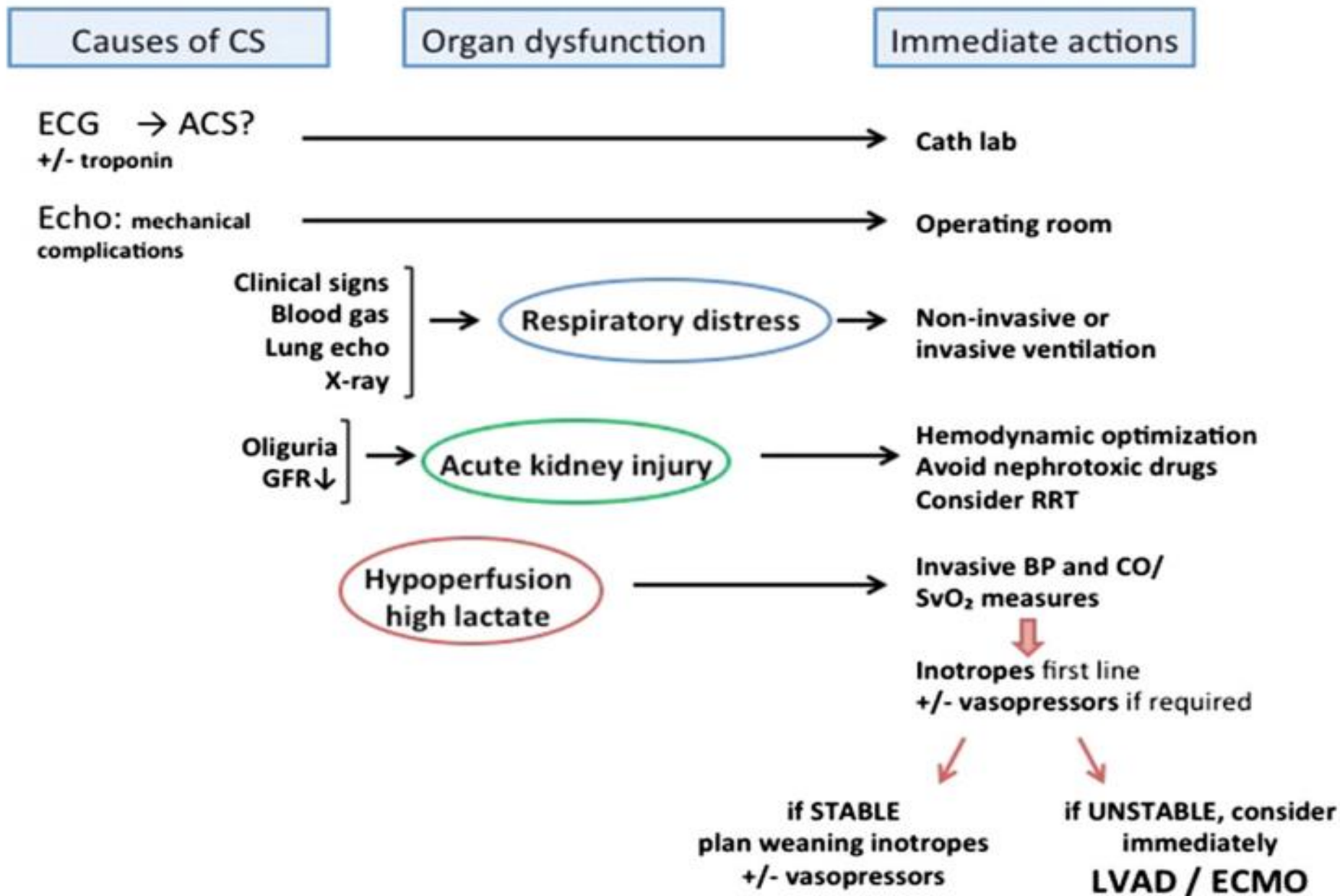
Hemodynamic optimization
Avoid nephrotoxic drugs
Consider RRT

Invasive BP and CO/
SvO₂ measures

Inotropes first line
+/- vasopressors if required

if STABLE
plan weaning inotropes
+/- vasopressors

if UNSTABLE, consider
immediately
LVAD / ECMO



In summary

- – **AHF mostly corresponds to organ congestion.**
- – **Early treatment initiation is associated with superior outcomes in AHF.**
- – **Lung ultrasound is an easy and efficient diagnostic tool to rule out pulmonary congestion.**
- – **The use of vasodilators is strongly recommended in most AHF patients.**
- – **NIV techniques improve respiratory rate faster compared to conventional oxygen therapy in APE patients.**

In summary

- **Inotropes and vasopressors are restricted to patients with cardiogenic shock, and should be used for the shortest possible period and with the lowest possible dose to restore perfusion pressure.**
- Patients with hemodynamic instability or cardiogenic shock should be treated in a specialized center with facilities of assist devices for circulatory support.

In summary

- **Mechanical support with assist devices should be considered early in the treatment of patients with cardiogenic shock, before the development of irreversible end-organ injuries.**
- AHF patients should benefit from a tight multidisciplinary post-discharge program to avoid rehospitalizations and other adverse outcome

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