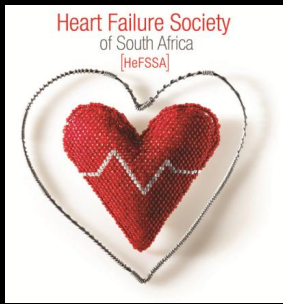


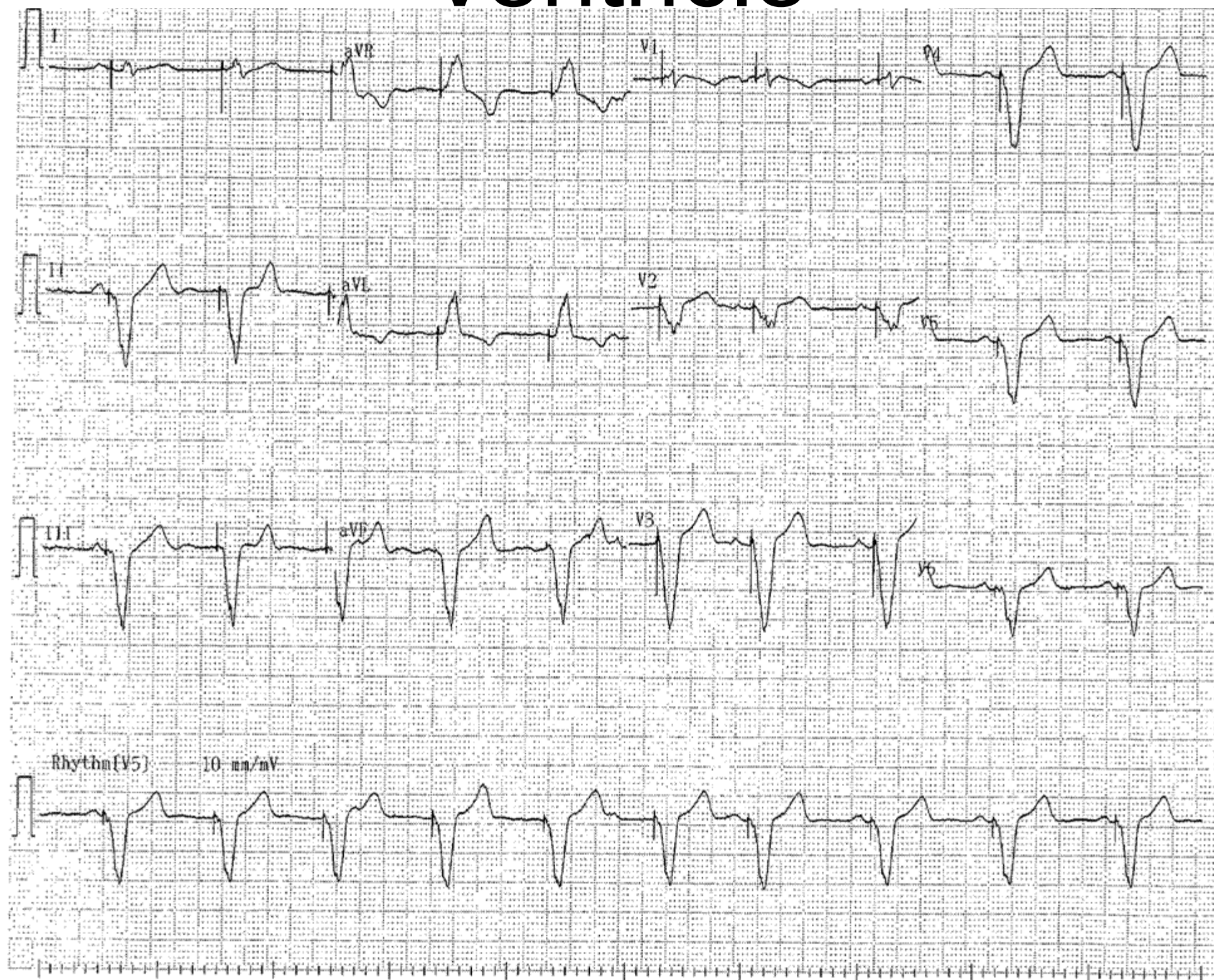
Case Study 2

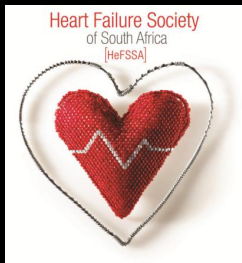


Mrs PM

- Initially seen as 52 year old woman in 1998 with a diagnosis of "burnt out" sarcoid heart disease
- HF-REF with an EF of 12%, NYHA FC IIb
- Had presented with AV Block and had a dual chamber pacemaker previously implanted
- Intermittent ventricular arrhythmias
- Medications included Furosemide 240 mg in divided doses, Carvedilol 25 mg BD, Accupril 10 mg BD, Isosorbide Mononitrate 20 mg BD, Plenish K 2 TDS, Metolazone 2,5 mg twice a week

Sensed atrium, paced ventricle

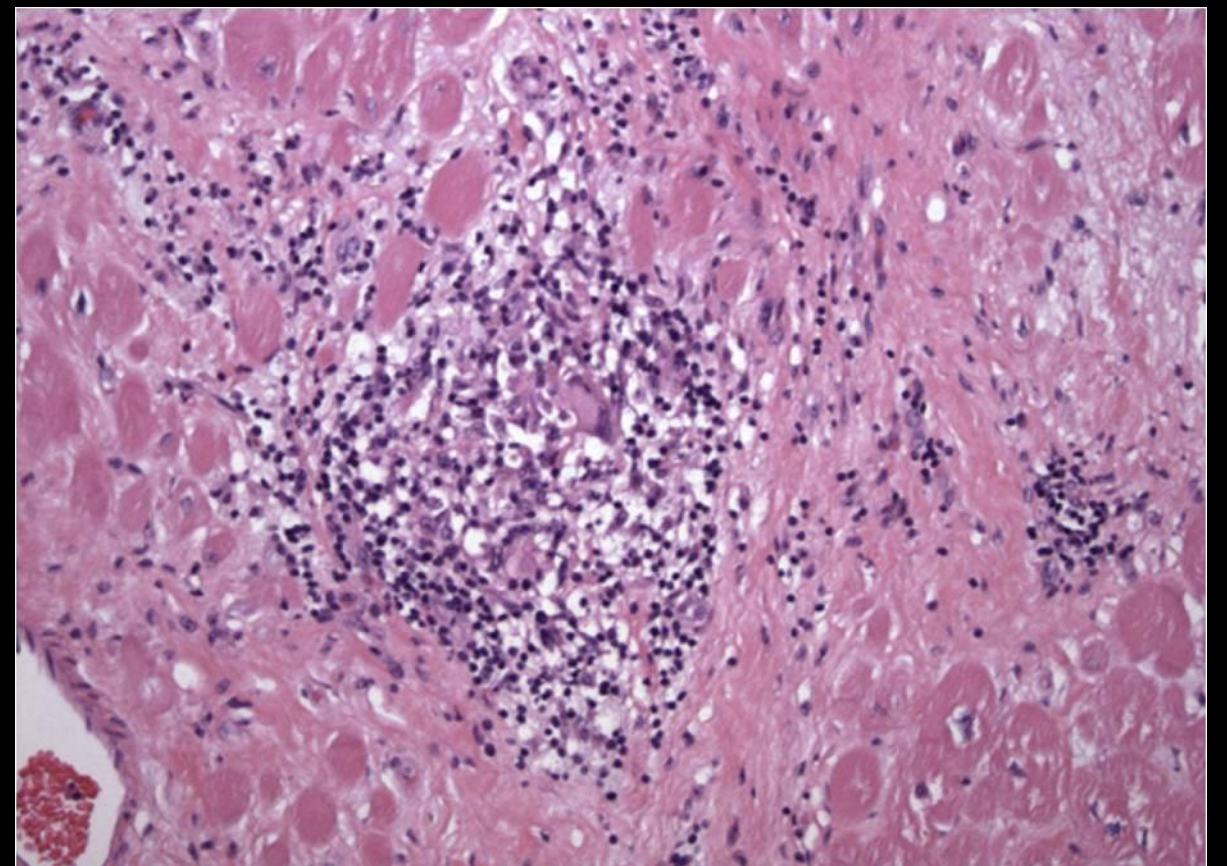


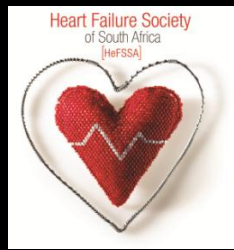


Sarcoid Heart Disease

Granulomatous Disease

- Cardiac sarcoidosis can be a benign, incidentally discovered condition or a life-threatening disorder causing sudden death.
- Predominately affects lungs
- Cardiac involvement rare - 1 in 50 clinical cases
- More cardiac involvement on autopsy - 20-25%
- Less common in Caucasians



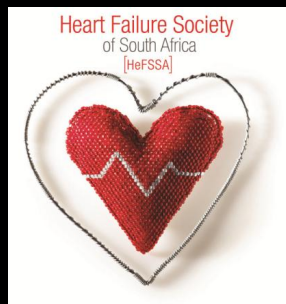


Sarcoid Heart Disease

- All chambers can be involved, ventricles predominately
- Can present as a dilated cardiomyopathy
- Mitral regurgitation can be present related to papillary muscle involvement
- Ventricular and atrial arrhythmias relatively common
- Definitive diagnosis - tissue biopsy
- Role of serum ACE, Calcium levels

Granulomatous Disease





DHF: Deteriorating Heart Failure

- After a period of prior stability, she presents to casualty on a Sunday night with worsening effort tolerance (NYHA FC III), palpitations, cough and body swelling
- She is found to have significant pedal oedema, cool peripheries, a sinus tachycardia of 104 beats per minute and a left sided third heart sound but relatively comfortable at rest, BP 100/85, SCr 140 $\mu\text{mol/l}$



Acute Heart Failure Syndrome

PUFFERS/SSOBAR

Admission usually
short and necessary

- Predominantly
- LV & Mitral Valve Dysfunction
 - Fluid mal-distribution
 - Pulmonary oedema

Orthopnoea
Hypertensive
Paroxysmal nocturnal dyspnoea
Reduced exercise tolerance

Aetiology

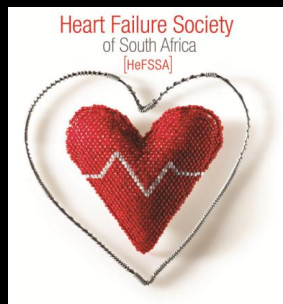
- ACS
- BP/Vascular
- Arrhythmia
- Valves
- Renal

BLOATERS/CARBOSE

Admission often prolonged but
really necessary

- Predominantly
- Right or BiV Dysfunction
 - Leading to fluid retention
 - Peripheral congestion

Ankle swelling
Hypotension
Ascites
Hepatomegaly

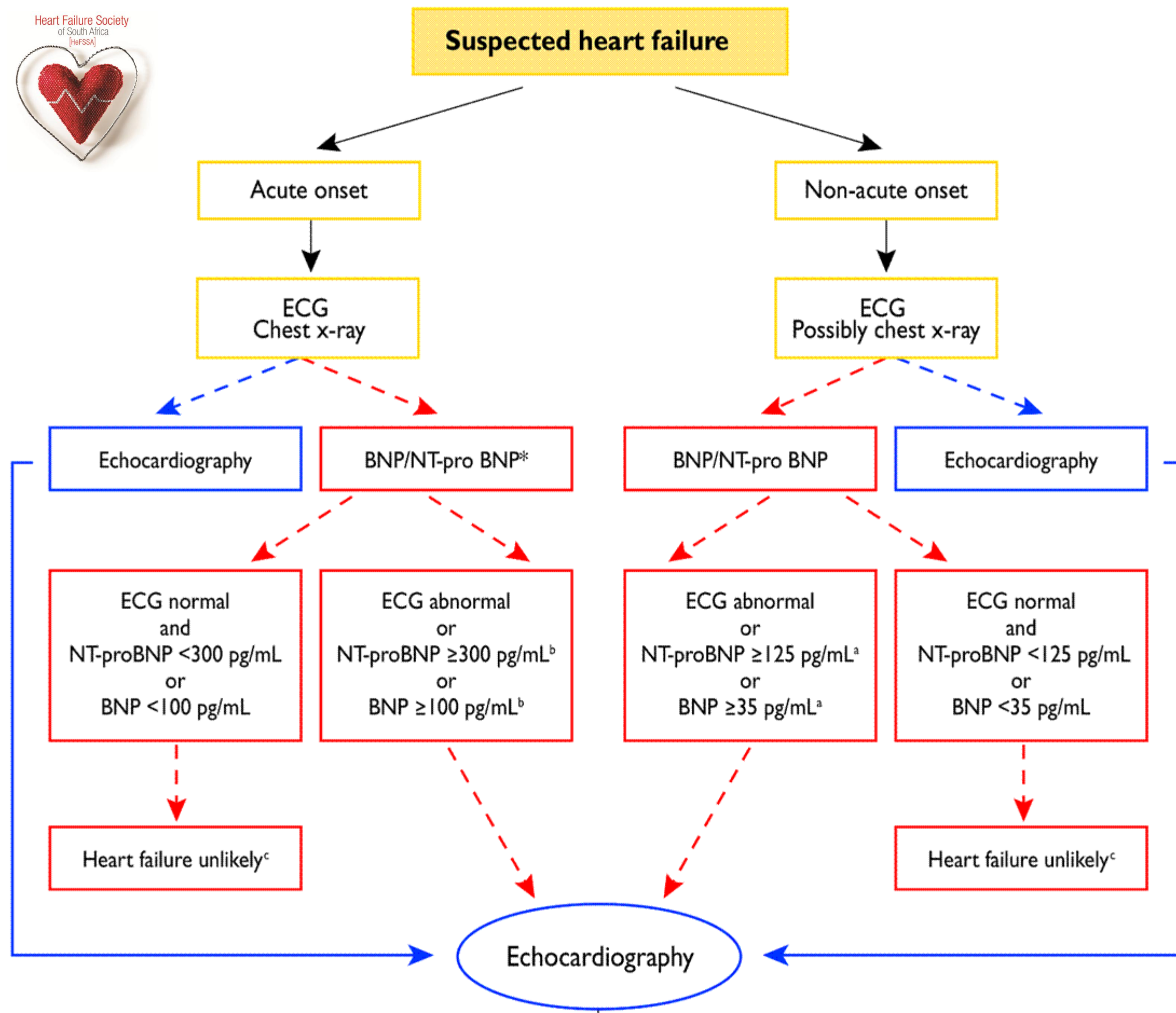


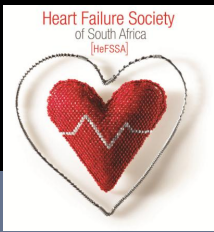
Categories of AHF

**end result of a
relatively slow (days to
weeks) deterioration of
severe chronic HF**



**rapidly progressive
disorder of high blood
pressure (BP)
accompanied by
severe acute dyspnea**





Ischemia as a Precipitant

The exact incidence of frank myocardial ischemia in AHF remains unknown

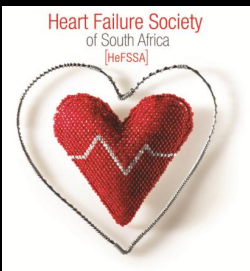
ECG changes and troponin release may occur in patients with HF without CAD

In ACS , AHF a very strong negative prognostic indicator

The EuroHeartFailure survey programme:
32% of patients admitted with AHF had chest pain at admission, and acute myocardial infarction was diagnosed in only 12%

PRESERVED-HF study
detectable troponin T was present in 43.5%, and troponin I was present in 73.9% of patients at baseline

Troponin spillage can occur even when ischemic heart disease is not present or ischemia may occur without significant troponin leakage



Our patient is cold and wet

Subset	Description
I: Warm and dry (normal)	PCWP 15–18 mmHg <i>and</i> CI >2.2 L/min/m ²
II: Warm and wet (congestion)	PCWP >18 mmHg <i>and</i> CI >2.2 L/min/m ²
III: Cold and dry (hypoperfusion)	PCWP 15–18 mmHg <i>and</i> CI <2.2 L/min/m ²
IV: Cold and wet (congestion and hypoperfusion)	PCWP >18 mmHg <i>and</i> CI <2.2 L/min/m ²

CI: cardiac index; PCWP: pulmonary capillary wedge pressure. Source: Reference 11.



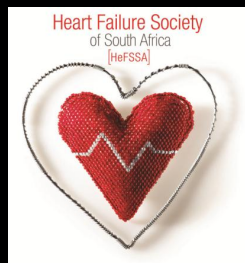
Perfusion	Warm	Cold
Volume status		
Wet	Warm/Wet	Cold/Wet
Dry	Warm/Dry	Cold/Dry

Diagram illustrating the relationship between Perfusion (Warm/Cold) and Volume status (Wet/Dry) in heart failure, with arrows indicating transitions between states.

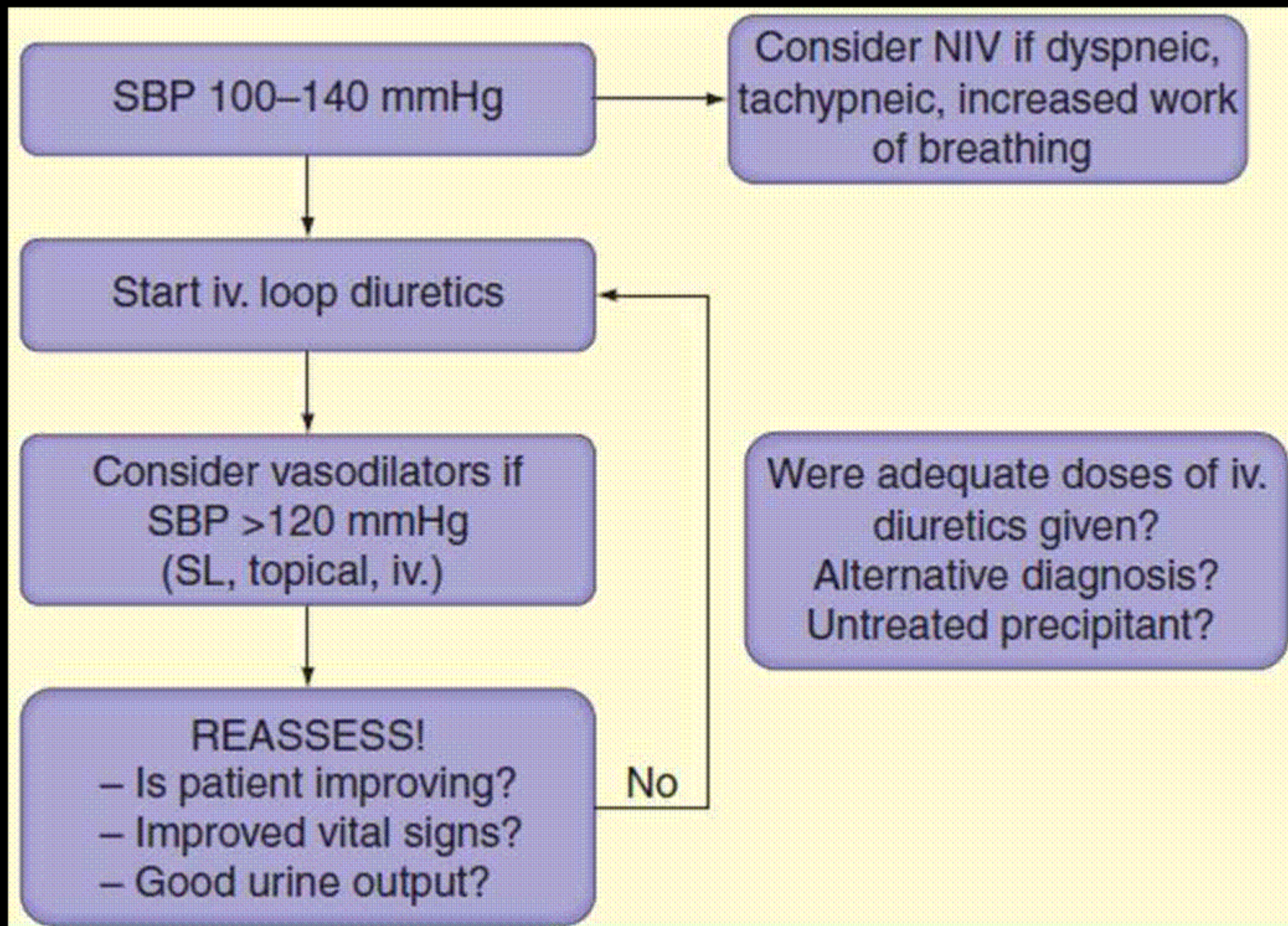
Transitions shown:

- From Cold/Wet to Warm/Wet (Left arrow)
- From Warm/Wet to Warm/Dry (Down arrow)
- From Cold/Dry to Cold/Wet (Up arrow)

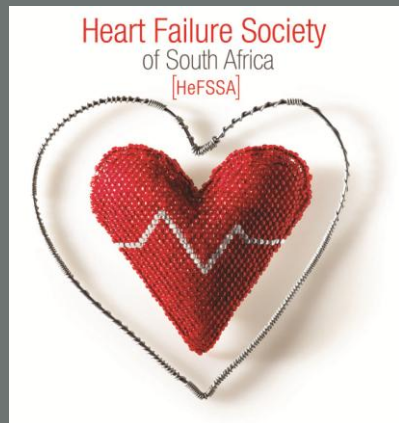
The Cold/Wet state is highlighted with a red dashed box.

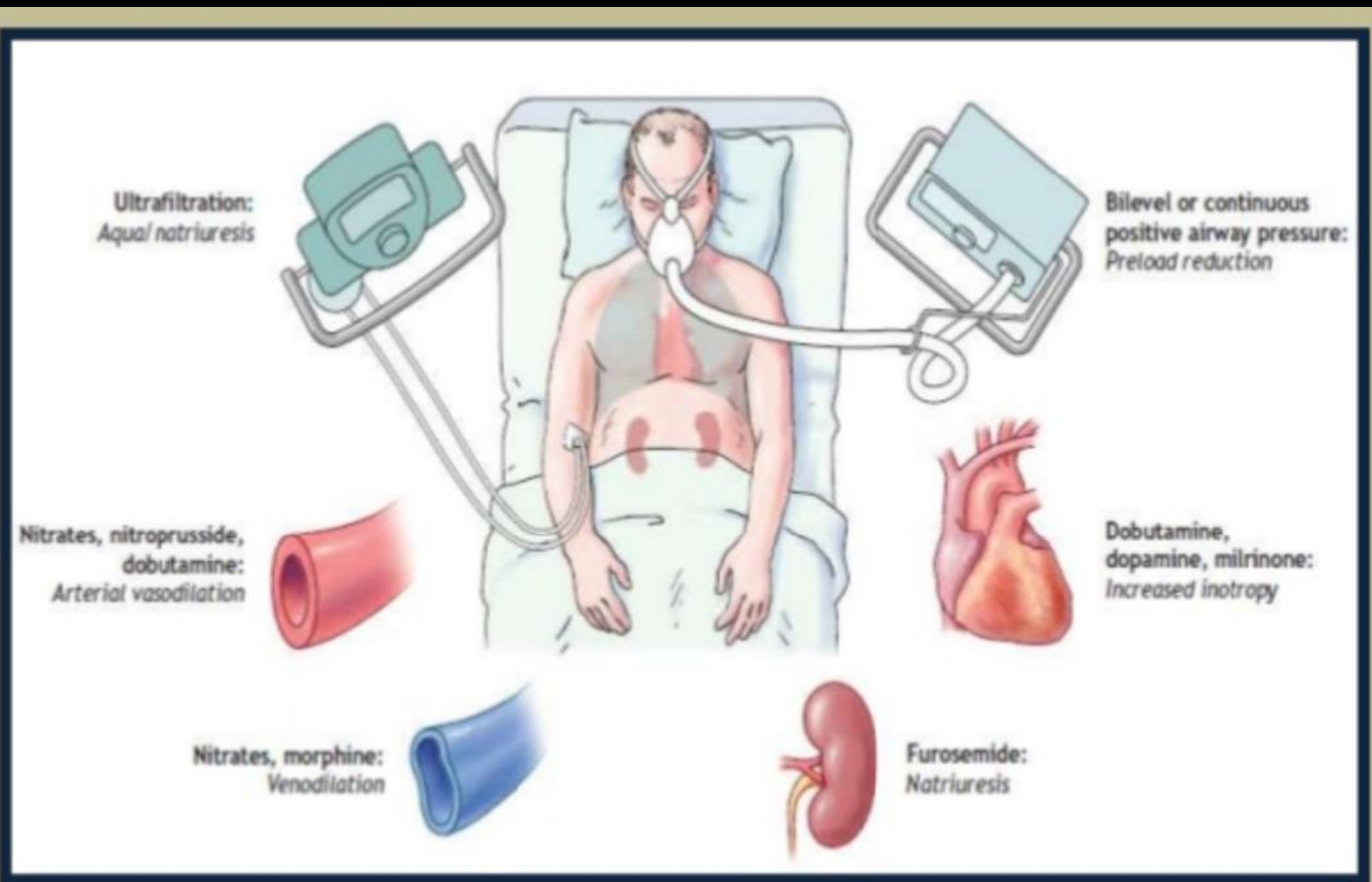


Algorithm for normotensive AHF

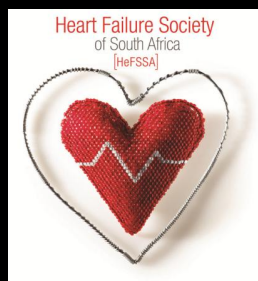


Non-Invasive positive pressure ventilation (NPPV) is a way of assuring positive pressure in the airways throughout the entire respiratory cycle without intubation





Various targets for therapies used in the management of acute heart failure.



Maintenance of GDMT During Hospitalization



In patients with HFrEF experiencing a symptomatic exacerbation of HF requiring hospitalization during chronic maintenance treatment with GDMT, it is recommended that GDMT be continued in the absence of hemodynamic instability or contraindications.

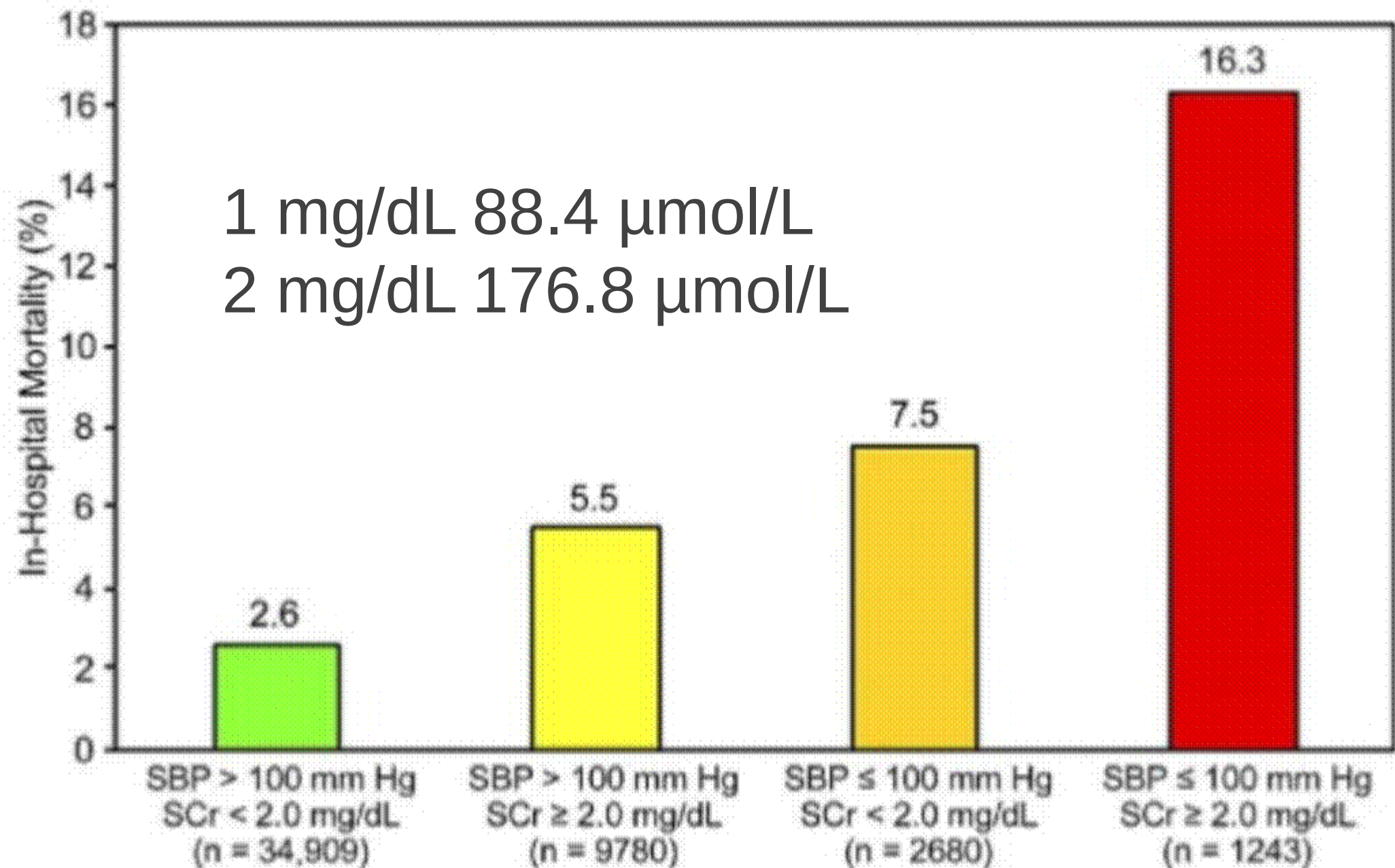


Therapies in the Hospitalized HF Patient

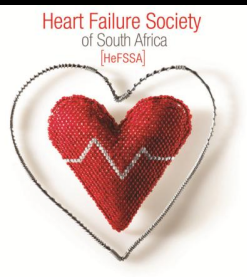
Recommendation	COR	LOE
HF patients hospitalized with fluid overload should be treated with intravenous diuretics	I	B
HF patients receiving loop diuretic therapy, should receive an initial parenteral dose greater than or equal to their chronic oral daily dose, then should be serially adjusted	I	B
HFrEF patients requiring HF hospitalization on GDMT should continue GDMT unless hemodynamic instability or contraindications	I	B
Initiation of beta-blocker therapy at a low dose is recommended after optimization of volume status and discontinuation of intravenous agents	I	B
Thrombosis/thromboembolism prophylaxis is recommended for patients hospitalized with HF	I	B
Serum electrolytes, urea nitrogen, and creatinine should be measured during the titration of HF medications, including diuretics	I	C



In-Hospital Mortality by SCr and SBP From OPTIMIZE HF Registry

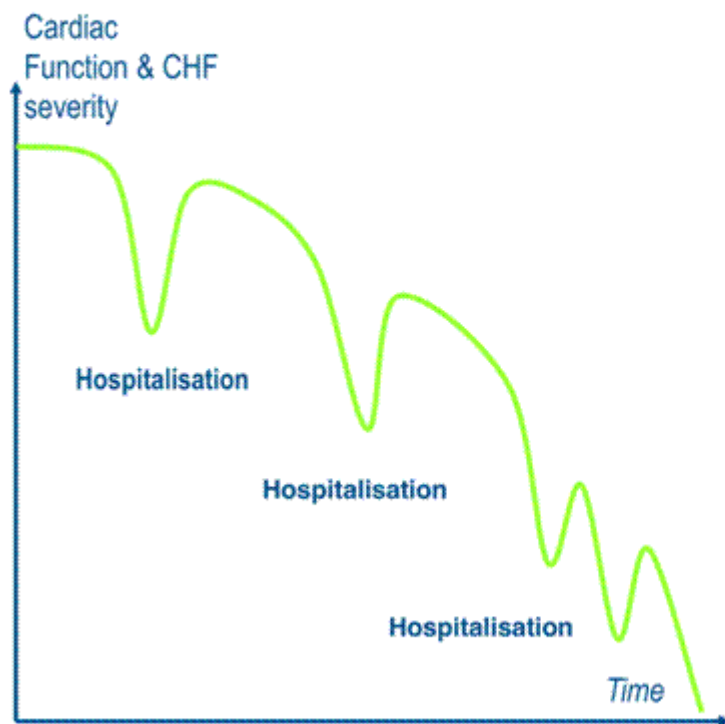


Abraham, W. T. et al. J Am Coll Cardiol 2008;52:347-356

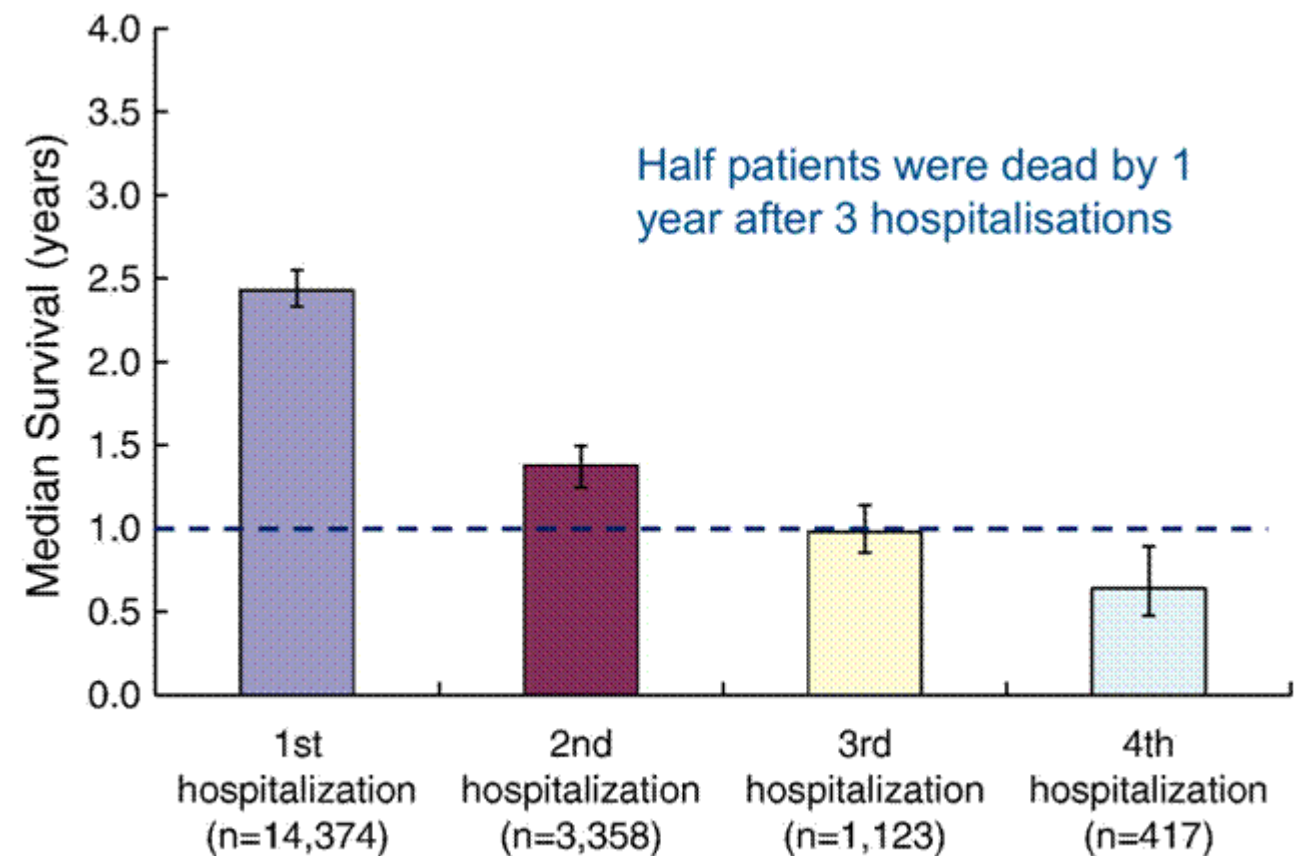


"Retained Cardiac Memory of Hospitalisation"

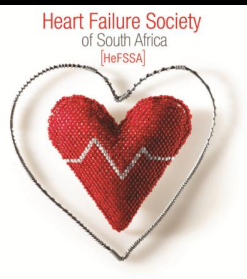
Recurring hospitalisations...



Gheorghiade M, et al. *Am J Cardiol*. 2005; 96:11-17.

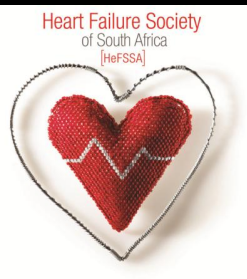


Setoguchi S, et al. *Am Heart J*. 2007;154:260-266.



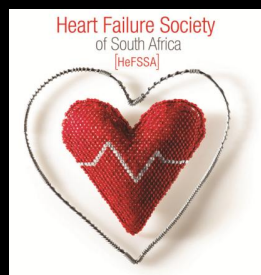
What next after stabilisation in hospital?

- Upgrade the pacemaker?
- Up-titrate current medications?
- Introduce additional therapies?

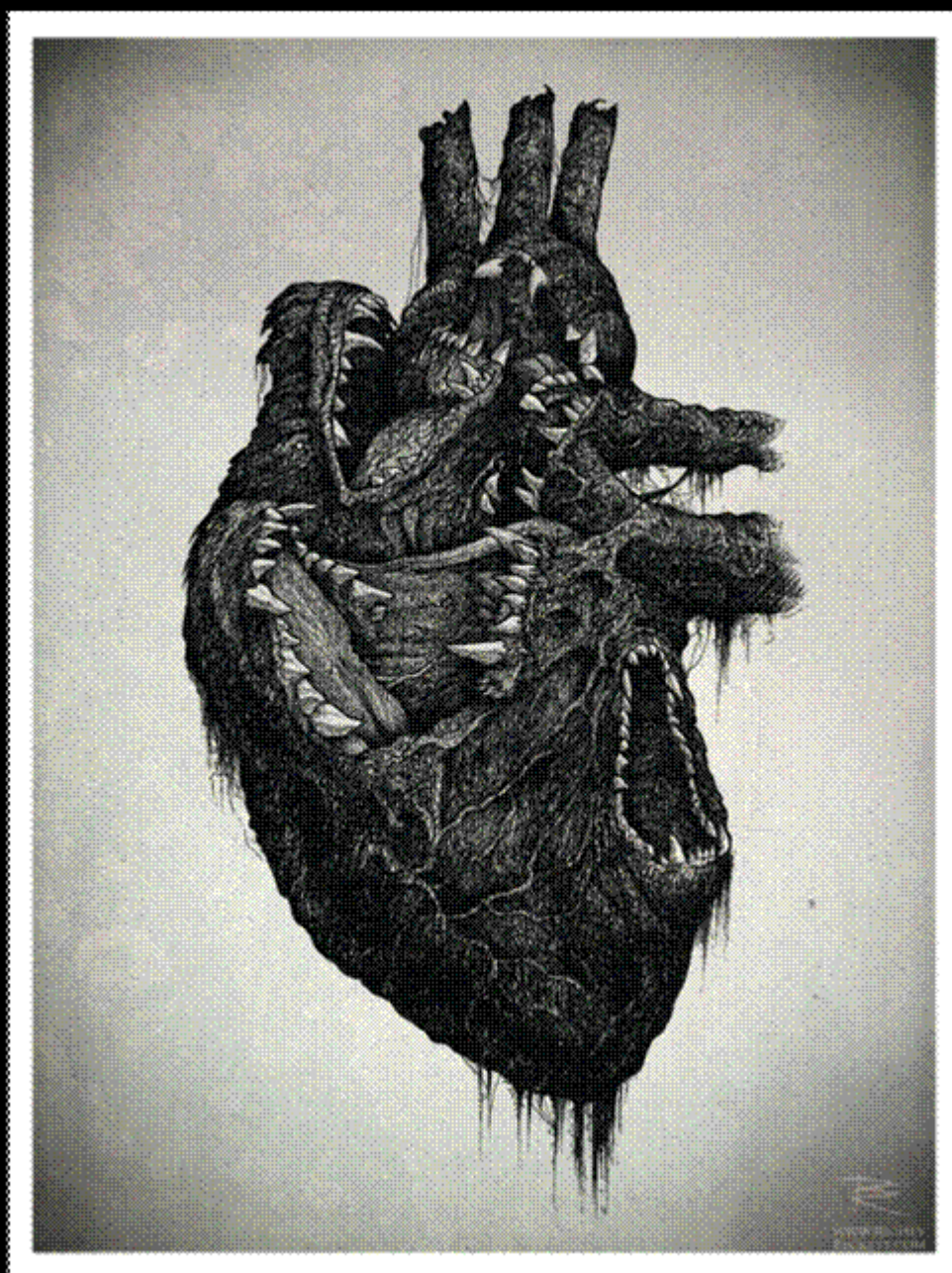


Ultimate treatment plan

- Try uptitrate Carvedilol to 50 mg bd
- Spironolactone should be added but.....
- Upgrade the pacemaker to CRT-D
- If heart rate remains above 70 beats/minute, add Ivabradine
- Manage the discharge, follow up planning



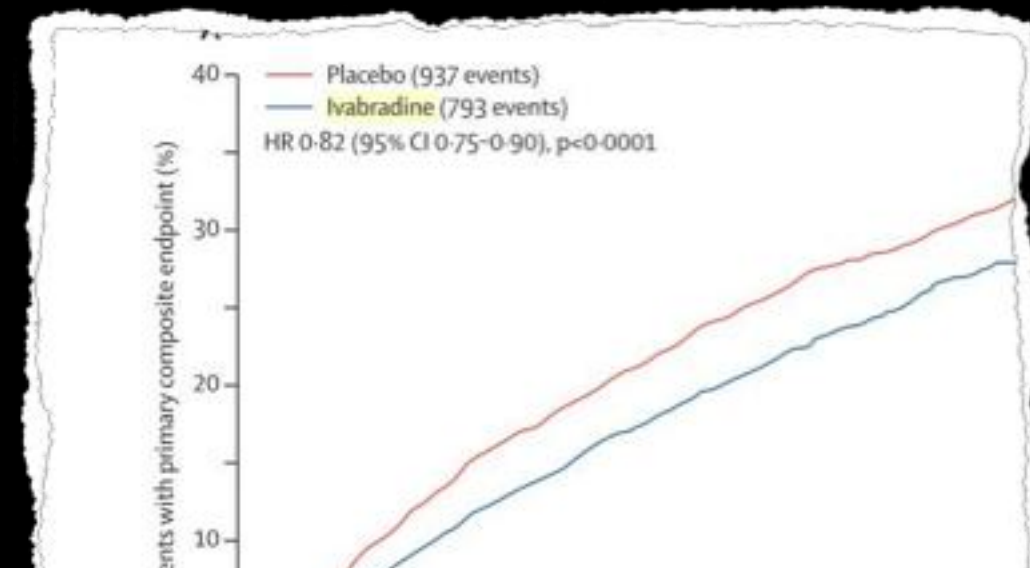
ICD



Recommendations	Class ^a	Level ^b
Secondary prevention An ICD is recommended in a patient with a ventricular arrhythmia causing haemodynamic instability, who is expected to survive for >1 year with good functional status, to reduce the risk of sudden death.	I	A
Primary prevention An ICD is recommended in a patient with symptomatic HF (NYHA class II–III) and an EF ≤35% despite ≥3 months of treatment with optimal pharmacological therapy, who is expected to survive for >1 year with good functional status, to reduce the risk of sudden death		
(i) Ischaemic aetiology and >40 days after acute myocardial infarction	I	A
(ii) Non-ischaemic aetiology	I	B



Ivabradine



Ivabradine

Should be considered to reduce the risk of HF hospitalization in patients in sinus rhythm with an EF $\leq 35\%$, a heart rate remaining ≥ 70 b.p.m., and persisting symptoms (NYHA class II–IV) despite treatment with an evidence-based dose of beta-blocker (or maximum tolerated dose below that), ACE inhibitor (or ARB), and an MRA (or ARB).^a

IIa

B

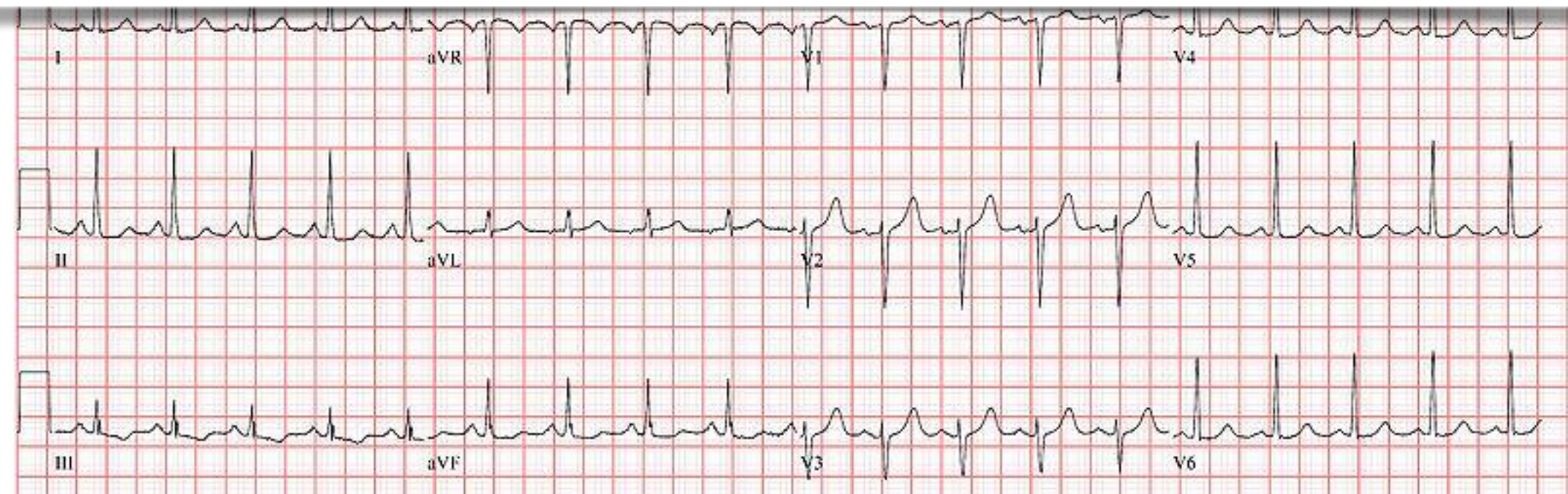
II2

May be considered to reduce the risk of HF hospitalization in patients in sinus rhythm with an EF $\leq 35\%$ and a heart rate ≥ 70 b.p.m. who are unable to tolerate a beta-blocker. Patients should also receive an ACE inhibitor (or ARB) and an MRA (or ARB).^a

IIb

C

–





The patient died in 2010 from intractable heart failure - 12 years after the admission for AHF. She was able to see all 5 grandchildren being born.

HeFSSA Practitioners Program 2014

(Heart Failure Society of South Africa)

08:00 – 08:30 Welcome

08:30 – 09:15 Clinical Case Presentation 1

09:15 – 10:00 Clinical Case Presentation 2

10:00 – 10:30 Tea Break

10:30 – 11:15 Clinical Case Presentation 3

11:15 – 11:45 Clinical Case Presentation 4

11:45 – 12:00 Questionnaire

12:00 – 14:00 Lunch

