ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2008

The Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2008 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association of the ESC (HFA) and endorsed by the European Society of Intensive Care Medicine (ESICM)

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- Patients with acute heart failure frequently develop chronic heart failure
- Patients with chronic heart failure frequently decompensate acutely

Classes of Recommendations	Definition	Suggested wording to use
Class I	Evidence and/or general agreement that a given treatment or procedure is beneficial,useful and effective	Is recommended/is indicated
Class II	Conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of the given treatment or procedure	
Class IIa	Weight of evidence/opinion is in favour of usefulness/efficacy	Should be considered
Class IIb	Usefulness/efficacy is less well established by evidence/opinion	May be considered
Class III	Evidence or general agreement that the given treatment or procedure is not useful/effective, and in some cases may be harmful	Is not recommended

Levels of evidence

Level of Evidence A	Date derived from multiple randomized clinical trials or meta-analyses.
Level of Evidence B	Date derived from a single randomized clinical trial or large non-randomized studies.
Level of Evidence C	Consensus of opinion of the experts and/or small studies, retrospective studies, registries.

”A clinical response to treatment directed at HF alone is not sufficient for the diagnosis, but is helpful when the diagnosis remains unclear after appropriate diagnostic investigations”

Definition of heart failure

HF is a clinical syndrome in which patients have the following features:

- Symptoms typical of HF
  (breathlessness at rest or on exercise, fatigue, tiredness, ankle swelling)

and

- Signs typical of HF
  (tachycardia, tachypnoea, pulmonary rales, pleural effusion, raised jugular venous pressure, peripheral oedema, hepatomegaly)

and

- Objective evidence of a structural or functional abnormality of the heart at rest
  (cardiomegaly, third heart sound, cardiac murmurs, abnormality on the echocardiogram, raised natriuretic peptide concentration)

Common clinical manifestations of heart failure

Dominant clinical feature	Symptoms	Signs
Peripheral oedema/ congestion	Breathlessness Tiredness, fatigue Anorexia	Peripheral oedema Raised jugular venous pressure Pulmonary oedema Hepatomegaly, ascites Fluid overload (congestion) Cachexia
Pulmonary oedema	Severe breathlessness at rest	Crackles or rales over lungs, effusion Tachycardia, tachypnoea
Cardiogenic shock (low output syndromes)	Confusion Weakness Cold periphery	Poor peripheral perfusion Systolic BP < 90 mmHg Anuria or oliguria
High blood pressure (hypertensive HF)	Breathlessness	Usually raised BP, LVH and preserved EF
Right HF	Breathlessness Fatigue	Evidence of RV dysfunction Raised JVP, peripheral oedema, hepatomegaly, gut congestion

”Most patients with HF have evidence of both systolic and diastolic dysfunction at rest or on exercise. Patients with diastolic HF have symptoms and/or signs of HF and a preserved left ventricular ejection fraction above 45-50%. HFPEF is present in half the patients with HF.”

Classification of heart failure

• **New onset**	First presentation Acute or slow onset
• **Transient**	Recurrent or episodic
• **Chronic**	Persistent Stable, worsening, or decompensated

Classification of HF by structural abnormality (ACC/AHA) or by symptoms relating to functional capacity (NYHA)

ACC/AHA Stages of HF Stage of heart failure based on structure and damage to heart muscle		NYHA Functional Classification Severity based on symptoms and physical activity	
Stage A	At high risk for developing HF. No identified structural or functional abnormality; no signs or symptoms.	Class I	No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, or dyspnoea.
Stage B	Developed structural heart disease that is strongly associated with the development of HF, but without signs or symptoms.	Class II	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation, or dyspnoea.
Stage C	Symptomatic HF associated with underlying structural heart disease.	Class III	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity results in fatigue, palpitation, or dyspnoea.
Stage D	Advanced structural heart disease and marked symptoms of HF at rest despite maximal medical therapy.	Class IV	Unable to carry on any physical activity without discomfort. Symptoms at rest. If any physical activity is undertaken, discomfort is increased.
ACC = American College of Cardiology; AHA, American Heart Association. Hunt SA et al. Circulation. 2005;112:1825-1852.		NYHA = New York Heart Association. The Criteria Committee of the New York Heart Association. Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Great Vessels. 9th ed. Boston, Mass: Little, Brown & Co; 1994:253-256	

Common causes of heart failure due to disease of heart muscle (myocardial disease)

Coronary heart disease	Many manifestations
Hypertension	Often associated with left ventricular hypertrophy and preserved ejection fraction
Cardiomyopathies*	Familial/genetic or non-familial/non-genetic (including acquired, e.g. myocarditis)
	Hypertrophic (HCM), dilated (DCM), restrictive (RCM), arrhythmogenic right ventricular (ARVC), Unclassified
Drugs	β-Blockers, calcium antagonists, antiarrhythmics, cytotoxic agents
Toxins	Alcohol, medication, cocaine, trace elements (mercury, cobalt, arsenic)
Endocrine	Diabetes mellitus, hypo/hyperthyroidism, Cushing syndrome, adrenal insufficiency, excessive growth hormone, phaeochromocytoma
Nutritional	Deficiency of thiamine, selenium, carnitine. Obesity, cachexia
Infiltrative	Sarcoidosis, amyloidosis, haemochromatosis, connective tissue disease
Others	Chagas' disease, HIV infection, peripartum cardiomyopathy, end-stage renal failure

*See text for details.

Key features of the clinical history in patients with heart failure

Symptoms	Breathlessness	(orthopnoea, paroxysmal nocturnal dyspnoea)
	Fatigue	(tiredness, exhaustion)
	Angina, palpitations, syncope	
Cardiovascular events	Coronary heart disease	
	Myocardial infarction	Thrombolysis
	Intervention	PCI
	Other surgery	CABG
	Stroke or peripheral vascular disease	
	Valvular disease or dysfunction	
Risk profile	Family history, smoking, hyperlipidaemia, hypertension, diabetes	
Response to current and previous therapy		

Key features of the clinical examination in patients with heart failure

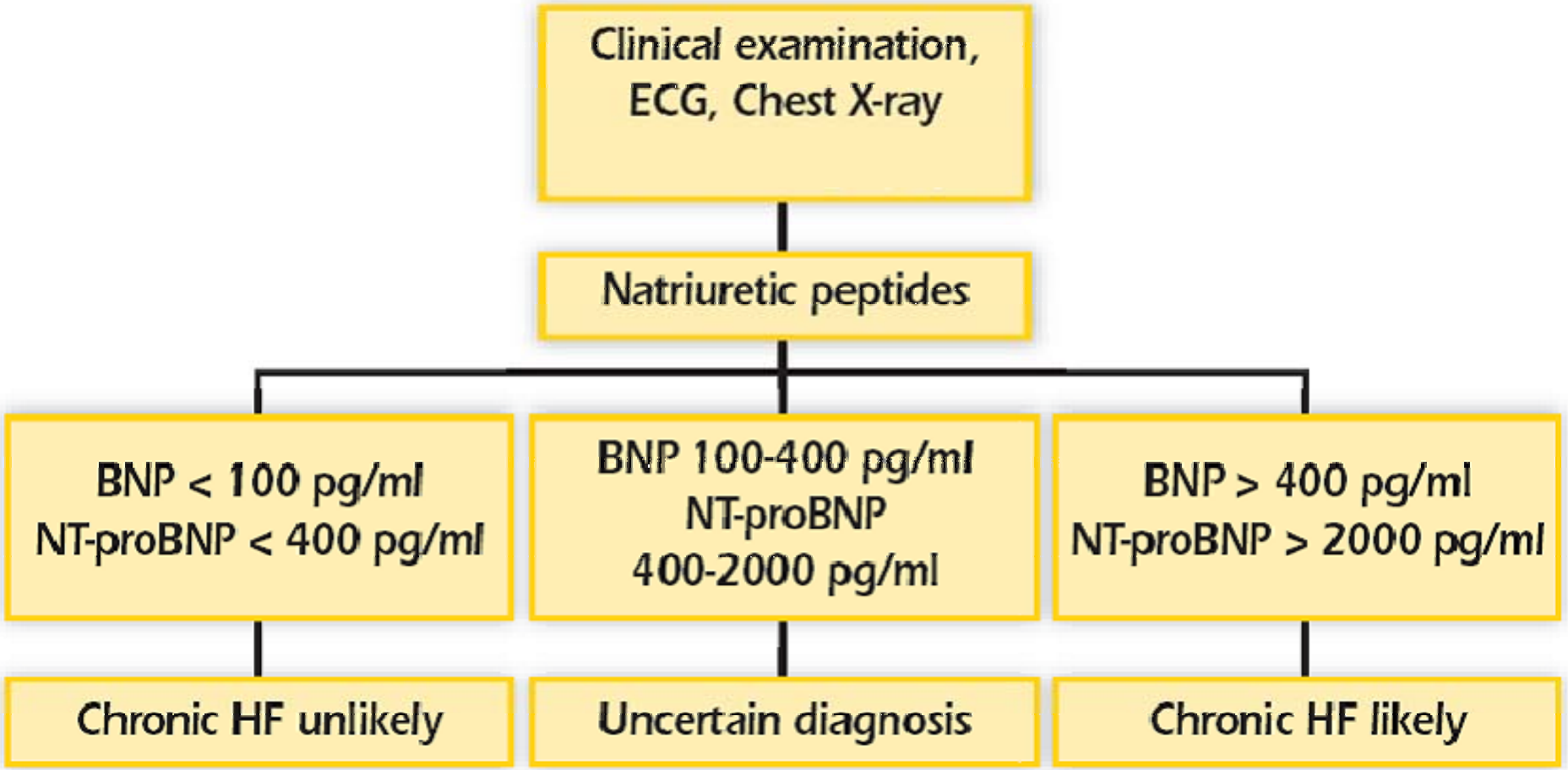
Appearance	Alertness, nutritional status, weight
Pulse	Rate, rhythm, and character
Blood pressure	Systolic, diastolic, pulse pressure
Fluid overload	Jugular venous pressure Peripheral oedema (ankles and sacrum) hepatomegaly, ascites
Lungs	Respiratory rate Rales Pleural effusion
Heart	Apex displacement Gallop rhythm, third heart sound Murmurs suggesting valvular dysfunction

Two classifications of the severity of heart failure in the context of acute myocardial infarction

Killip classification		Forrester classification
Designed to provide a clinical estimate of the severity of circulatory derangement in the treatment of acute myocardial infarction.		Designed to describe clinical and haemodynamic status in acute myocardial infarction.
Stage I	No heart failure. No clinical signs of cardiac decompensation	1. Normal perfusion and pulmonary wedge pressure (PCWP—estimate of left atrial pressure)
Stage II	Heart failure. Diagnostic criteria include rales. S3 gallop. and pulmonary venous hypertension. Pulmonary congestion with wet rales in the lower half of the lung fields.	2. Poor perfusion and low PCWP (hypovolaemic) 3. Near normal perfusion and high PCWP (pulmonary oedema)
Stage III	Severe heart failure. Frank pulmonary oedema with rales throughout the lung fields	4. Poor perfusion and high PCWP (cardiogenic shock)
Stage IV	Cardiogenic shock. Signs include hypotension (SBP <90 mmHg), and evidence of peripheral vasoconstriction such as oliguria, cyanosis and sweating	

Killip T, 3rd, Kimball JT. Treatment of myocardial infarction in a coronary care unit. A two year experience with 250 patients. *Am J Cardiol* 1967;**20**:457–464.
Forrester JS, Diamond GA, Swan HJ. Correlative classification of clinical and hemodynamic function after acute myocardial infarction. *Am J Cardiol* 1977;**39**:137–145.

Flow-chart for the diagnosis of HF in untreated patients with symptoms suggestive of HF using natriuretic peptides



Diagnostic assessments supporting the presence of heart failure

Assessment	Diagnosis of heart failure	
	Supports if present	Opposes if normal or absent
Compatible symptoms	++	++
Compatible signs	++	+
Cardiac dysfunction on echocardiography	+++	+++
Response of symptoms or signs to therapy	+++	++
ECG		
Normal		++
Abnormal	++	+
Dysrhythmia	+++	+
Laboratory		
Elevated BNP/NT-proBNP	+++	+
Low/normal BNP/NT-proBNP	+	+++
Hyponatraemia	+	+
Renal dysfunction	+	+
Mild elevations of troponin	+	+
Chest X-ray		
Pulmonary congestion	+++	+
Reduced exercise capacity	+++	++
Abnormal pulmonary function tests	+	+
Abnormal haemodynamics at rest	+++	++

+ = some importance; ++ = intermediate importance; +++ = great importance.

Common ECG abnormalities in HF

Abnormality	Causes	Clinical Implications
Sinus tachycardia	Decompensated HF, anaemia, fever, hyperthyroidism	Clinical assessment Laboratory investigation
Sinus bradycardia	Beta-blockade, anti-arrhythmics, hypothyroidism, sick sinus syndrome	Evaluate drug therapy Laboratory investigation
Atrial tachycardia/flutter/ fibrillation	Hyperthyroidism, infection, decompensated HF, infarction	Slow AV conduction, medical conversion, electroversion, catheter ablation, anticoagulation
Ventricular arrhythmias	Ischaemia, infarction, cardiomyopathy, myocarditis hypokalaemia, hypomagnesaemia, digitalis overdose	Laboratory investigation, exercise test, perfusion studies, coronary angiography, electrophysiology testing, ICD
Ischaemia/Infarction	Coronary artery disease	Echo, troponins, coronary angiography, revascularization
Q waves	Infarction, hypertrophic cardiomyopathy, LBBB, pre-excitation	Echo, coronary angiography
LV hypertrophy	Hypertension, aortic valve disease, hypertrophic cardiomyopathy	Echo/Doppler
AV block	Infarction, drug toxicity, myocarditis, sarcoidosis, Lyme disease	Evaluate drug therapy, pacemaker, systemic disease
Microvoltage	Obesity, emphysema, pericardial effusion, amyloidosis	Echo, chest X-ray
QRS length >120 msec of LBBB morphology	Electrical dyssynchrony	Echo, CRT-P, CRT-D

Common chest X-ray abnormalities in heart failure

Abnormality	Causes	Clinical Implications
Cardiomegaly	Dilated LV, RV, atria Pericardial effusion	Echo/Doppler
Ventricular hypertrophy	Hypertension, aortic stenosis, hypertrophic cardiomyopathy	Echo/Doppler
Normal pulmonary findings	Pulmonary congestion unlikely	Reconsider diagnosis (if untreated) Serious lung disease unlikely
Pulmonary venous congestion	Elevated LV filling pressure	Left heart failure confirmed
Interstitial oedema	Elevated LV filling pressure	Left heart failure confirmed
Pleural effusions	Elevated filling pressures HF likely if bilateral Pulmonary infection, surgery or malignant effusion	Consider non-cardiac aetiology If abundant, consider diagnostic or therapeutic centesis
Kerley B lines	Increased lymphatic pressures	Mitral stenosis or chronic HF
Hyperlucent lung fields	Emphysema or pulmonary embolism	Spiral CT, spirometry, Echo
Pulmonary infection	Pneumonia may be secondary to pulmonary congestion	Treat both infection and HF
Pulmonary infiltration	Systemic disease	Diagnostic work-up

Common laboratory test abnormalities in heart failure (1)

Abnormality	Cause	Clinical implications
Increased serum creatinine (>150 µmol/L)	Renal disease ACEI/ARB, aldosterone blockade	Calculate GFR, Consider reducing ACEI/ARB, or aldosterone blocker dose Check potassium and BUN
Anaemia (13 g/dL in men, 12 in women)	Chronic HF, haemodilution, iron loss or poor utilization, renal failure, chronic disease	Diagnostic work-up Consider treatment
Hyponatraemia (<135 mmol/L)	Chronic HF, haemodilution. AVP release, diuretics	Consider water restriction reducing diuretic dosage Ultrafiltration, vasopressin antagonist
Hypernatraemia (>150 mmol/L)	Hyperglycaemia Dehydratation	Assess water intake Diagnostic work-up
Hypokalaemia (<3.5 mmol/L)	Diuretics, secondary hyperaldosteronism	Risk of arrhythmia Consider potassium supplements, ACEIs/ARB, aldosterone blockers
Hyperkalaemia (>5.5 mmol/L)	Renal failure, potassium supplement, renin–angiotensin–aldosterone system blockers	Stop potassium-sparing treatment (ACEIs/ARB, aldosterone blockers) Assess renal function and pH Risk of bradycardia
Hyperglycaemia (>6.5 mmol/L)	Diabetes, insulin resistance	Evaluate hydration treat glucose intolerance
Hyperuricaemia (>500 µmol/L)	Diuretic treatment, gout, malignancy	Allopurinol Reduce diuretic dose
BNP >400 pg/mL, NT-proBNP >2000 pg/mL	Increased ventricular wall stress	HF likely Indication for echo Consider treatment

Common laboratory test abnormalities in heart failure (2)

BNP <100 pg/mL, NT-proBNP <400 pg/mL	Normal wall stress	Re-evaluate diagnosis HF unlikely if untreated
Albumin high (>45 g/L)	Dehydratation, myeloma	Rehydrate
Albumin low (<30 g/L)	Poor nutrition, renal loss	Diagnostic work-up
Transaminase increase	Liver dysfunction Right heart failure Drug toxicity	Diagnostic work-up Liver congestion Reconsider therapy
Elevated troponins	Myocyte necrosis Prolonged ischaemia, severe HF, myocarditis, sepsis, renal failure, pulmonary embolism	Evaluate pattern of increase (mild increases common in severe HF) Coronary angiography Evaluation for revascularization
Abnormal thyroid tests	Hyper/hypothyroidism Amiodarone	Treat thyroid abnormality
Urinalysis	Proteinuria, glycosuria, bacteria	Diagnostic work-up Rule out infection
INR >2.5	Anticogulant overdose Liver congestion	Evaluate anticoagulant dosage Assess liver function Assess anticoagulant dose
CRP >10 mg/L, neutrophilic leukocytosis	Infection, inflammation	Diagnostic work-up

Common echocardiographic abnormalities in heart failure

Measurement	Abnormality	Clinical Implications
LV ejection fraction	Reduced (< 45-50%)	Systolic dysfunction
Left ventricular function, global and focal	Akinesis, hypokinesis, dyskinesis	Myocardial infarction/ischaemia Cardiomyopathy, myocarditis
End diastolic diameter	Increased (> 55-60 mm)	Volume overload - HF likely
End systolic diameter	Increased (> 45 mm)	Volume overload Systolic dysfunction likely
Fractional shortening	Reduced (< 25%)	Systolic dysfunction
Left atrial size	Increased (> 40 mm)	Increased filling pressures Mitral valve dysfunction Atrial fibrillation
Left ventricular thickness	Hypertrophy (> 11-12 mm)	Hypertension, aortic stenosis, hypertrophic cardiomyopathy
Valvular structure and function	Valvular stenosis or regurgitation (especially aortic stenosis and mitral insufficiency)	May be primary cause of HF or complicating factor Assess gradients and regurgitant fraction - Assess haemodynamic consequences Consider surgery
Mitral diastolic flow profile	Abnormalities of the early and late diastolic filling patterns	Indicates diastolic dysfunction and suggests mechanism
Tricuspid regurgitation peak velocity	Increased (> 3 m/sec)	Increased right ventricular systolic pressure - suspect pulmonary hypertension
Pericardium	Effusion, haemopericardium, thickening	Consider tamponade, uraemia, malignancy, systemic disease, acute or chronic pericarditis, constrictive pericarditis
Aortic outflow velocity time integral	Reduced (< 15 cm)	Reduced low stroke volume
Inferior vena cava	Dilated Retrograde flow	Increased right atrial pressures Right ventricular dysfunction Hepatic congestion

Doppler-echocardiographic indices and ventricular filling

Doppler indices	Pattern	Consequence
E/A waves ratio	Restrictive (> 2, short deceleration time < 115 to 150 msec)	High filling pressures Volume overload
	Slowed relaxation (< 1)	Normal filling pressures Poor compliance
	Normal (> 1)	Inconclusive as may be pseudo-normal
E/Ea	Increased (> 15)	High filling pressures
	Reduced (< 8)	Low filling pressures
	Intermediate (8 - 15)	Inconclusive
(A mitral - A pulm) duration	> 30 msec	Normal filling pressures
	< 30 msec	High filling pressures
Pulmonary S wave	> D wave	Low filling pressures
Vp	< 45 cm/sec	Slow relaxation
E/Vp	> 2.5	High filling pressures
	< 2	Low filling pressures
Valsalva manoeuver	Change of the pseudonormal to abnormal filling pattern	Unmasks high filling pressure in the setting of systolic and diastolic dysfunction

1. Presence of signs or symptoms of CHF
2. Presence of normal or only mildly abnormal left ventricular systolic function (LVEF > 45-50%)
3. Evidence of diastolic dysfunction (abnormal left ventricular relaxation or diastolic stiffness)

Conditions associated with a poor prognosis in HF

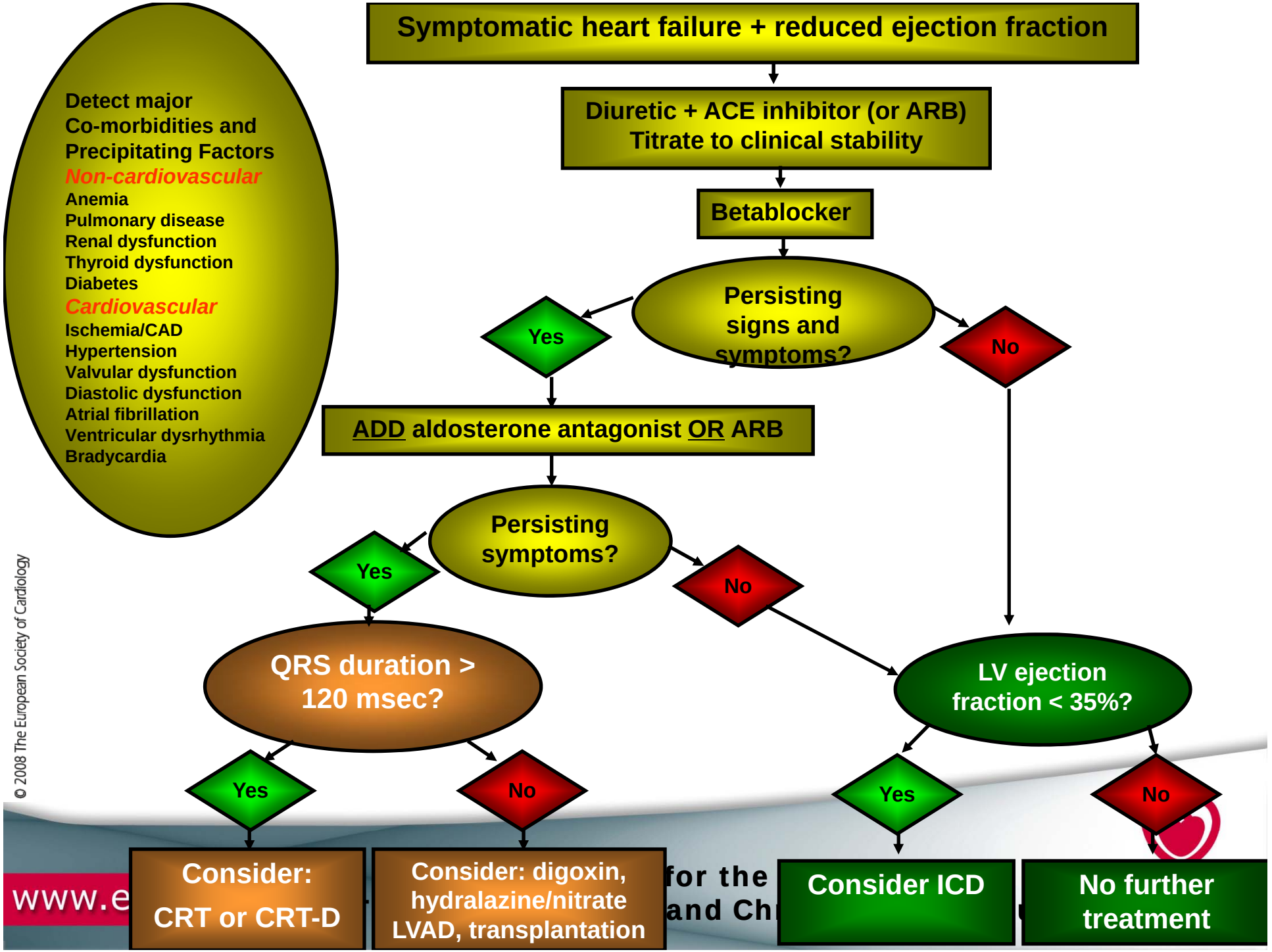
Demographics	Clinical	Electrophysiological	Functional/Exertional	Laboratory	Imaging
Advanced age*	**Hypotension***	**Tachycardia**	**Reduced work,**	**Marked elevation of BNP/NT pro-BNP***	**Low LVEF***
Ischaemic aetiology*	**NYHA Functional Class III-IV***	**Q Waves**	**Low peak VO2***	**Hyponatraemia***	
Resuscitated sudden death*	**Recent HF hospitalization***	**Wide QRS***		**Elevated troponin***	
		LV hypertrophy		**Elevated biomarkers, neurohumoral activation***	
		Complex ventricular arrhythmias*			
Poor compliance	Tachycardia	Low heart rate variability	Poor 6 min walk distance	Elevated creatinine/BUN	Increased LV volumes
Renal dysfunction	Pulmonary rales	T-wave alternans	High VE/VCO2 slope	Elevated bilirubin	Low cardiac index
Diabetes	Aortic stenosis	Atrial fibrillation	Periodic breathing	Anaemia	High left ventricular filling pressure
Anaemia	Low body mass index			Elevated uric acid	Restrictive mitral filling pattern, pulmonary hypertension
COPD	Sleep related breathing disorders				Impaired right ventricular function
Depression					

Essential topics in patient education with associated skills and appropriate self care behaviours

Educational topics	Skills and Self-care Behaviours
Definition and aetiology of heart failure	Understand the cause of heart failure and why symptoms occur
Symptoms and signs of heart failure	Monitor and recognise signs and symptoms Record daily weight and recognise rapid weight gain Know how and when to notify health care provider Use flexible diuretic therapy if appropriate and recommended
Pharmacological treatment	Understand indications, dosing and effects of drugs Recognise the common side-effects of each drug prescribed
Risk factor modification	Understand the importance of smoking cessation Monitor blood pressure if hypertensive Maintain good glucose control if diabetic Avoid obesity
Diet recommendation	Sodium restriction if prescribed - Avoid of excessive fluid intake Modest intake of alcohol - Monitor and prevent malnutrition
Exercise recommendations	Be reassured and comfortable about physical activity Understand the benefits of exercise Perform exercise training regularly
Sexual activity	Be reassured about engaging in sex and discuss problems with health care professionals Understand specific sexual problems and various coping strategies
Immunisation	Receive immunisation against infections such as influenza and pneumococcal disease
Sleep and breathing disorders	Recognise preventive behaviour such as weight loss if obese, smoking cession and abstinence from alcohol Learn about treatment options if appropriate
Adherence	Understand the importance of following treatment recommendations and maintaining motivation to follow treatment plan
Psychosocial aspects	Understand that depressive symptom and cognitive dysfunction are common in patients with heart failure and the importance of social support - Learn about treatment options if appropriate
Prognosis	Understand important prognostic factors and make realistic decisions - Seek psychosocial support if appropriate

Objectives of treatment in chronic heart failure

1. Prognosis	Reduce mortality
2. Morbidity	Relieve symptoms and signs Improve quality of life Eliminate oedema and fluid retention Increase exercise capacity Reduce fatigue and breathlessness Reduce need for hospitalisation Provide for end of life care
3. Prevention	Occurrence of myocardial damage Progression of myocardial damage Remodelling of the myocardium Reoccurrence of symptoms and fluid accumulation Hospitalisation


Symptomatic heart failure + reduced ejection fraction
Diuretic + ACE inhibitor (or ARB)
Titrate to clinical stability
Betablocker
Persisting signs and symptoms?
Yes
No
ADD aldosterone antagonist OR ARB
Persisting symptoms?
Yes
No
QRS duration > 120 msec?
Yes
No
LV ejection fraction < 35%?
Yes
No
Consider: CRT or CRT-D
Consider: digoxin, hydralazine/nitrate LVAD, transplantation
Consider ICD
No further treatment
Detect major Co-morbidities and Precipitating Factors
Non-cardiovascular
Anemia
Pulmonary disease
Renal dysfunction
Thyroid dysfunction
Diabetes
Cardiovascular
Ischemia/CAD
Hypertension
Valvular dysfunction
Diastolic dysfunction
Atrial fibrillation
Ventricular dysrhythmia
Bradycardia
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ACE inhibitors

- An ACE inhibitor is recommended in all patients with symptomatic HF and an EF ≤40%

- Treatment with an ACE inhibitor improves LV function, patient well-being, reduces hospital admission for worsening HF and increases survival

Class of recommendation I, level of evidence A

- In hospitalised patients, treatment should be initiated before discharge

Angiotensin receptor blockers (ARBs)

- An ARB is recommended in all pts. with HF and an EF ≤40% who:
  - remain symptomatic despite optimal Rx with an ACE inhibitor and β-blocker
  - as an alternative in pts. intolerant of an ACE inhibitor
- Unless pts. are treated with an aldosterone antagonist
- Treatment with an ARB improves LV function, patient well-being and reduces hospital admission for worsening HF

Class of recommendation I, level of evidence A

- Treatment reduces the risk of CV death

Class of recommendation IIa, level of evidence B

- In hospitalised pts., treatment with an ARB should be initiated before discharge

β-blockade

- A β-blocker should be used in all patients with symptomatic HF and an EF ≤40%
- β-Blockade improves ventricular function and patient well-being, reduces hospital admission for worsening HF and increases survival

Class of recommendation I, level of evidence A

- In hospitalised patients, treatment with a β-blocker should be initiated cautiously before discharge

Aldosterone antagonists

- The addition of an aldosterone antagonist is recommended in all patients with an EF ≤35%, severe symptomatic HF without hyperkalaemia or significant renal dysfunction

- Aldosterone antagonists reduce hospital admission for worsening HF and increase survival when added to existing therapy, including an ACE inhibitor

Class of recommendation I, level of evidence B

- In such hospitalised patients, treatment with an aldosterone antagonist should be initiated before discharge

Diuretics

- Diuretics are recommended in patients with clinical signs or symptoms of congestion
- Diuretics provide relief from the symptoms and signs of pulmonary and systemic venous congestion
- Diuretics cause activation of the renin-angiotensin-aldosterone system and should be used in combination with an ACE inhibitor/ARB

Class of recommendation I, level of evidence B

Class I recommendations for drugs in patients with symptomatic systolic dysfunction

ACE inhibitor	All patients*	Class I Level A
ARB	ACE intolerant/persisting signs or symptoms on ACEI/B-blokade*	Class I Level A
B-Blocker	All patients*	Class I Level A
Aldosterone antagonist	Severe symptoms on ACEI*	Class I Level A
Diuretic	All patients with signs or symptoms of congestion	Class I Level B

***unless contraindications or not tolerated**

Dosages of commonly used drugs in HF

	Starting dose (mg)		Target dose (mg)	
ACEI				
captopril	6.25	t.i.d.	50 - 100	t.i.d.
enalapril	2.5	b.i.d.	10 - 20	b.i.d.
lisinopril	2.5 - 5.0	o.d.	20 - 35	o.d.
ramipril	2.5	o.d.	5	b.i.d.
trandolapril	0.5	o.d.	4	o.d.
ARB				
candesartan	4 or 8	o.d.	32	o.d.
valsartan	40	b.i.d.	160	b.i.d.
Aldosterone antagonist				
eplerenone	25	o.d.	50	o.d.
spironolactone	25	o.d.	25 - 50	o.d.
Beta-blocker				
bisoprolol	1.25	o.d.	10	o.d.
carvedilol	3.125	b.i.d.	25 - 50	b.i.d.
metoprolol succinate	12.5/25	o.d.	200	o.d.
nebivolol	1.25	o.d.	10	o.d.
Hydralazine-ISDN				
Hydralazine-ISDN	37.5/20	t.i.d.	75/40	t.i.d.

Practical considerations in treatment of heart failure with loop diuretics

Problems	Suggested action
Hypokalaemia/hypomagnesaemia	• Increase ACEI/ARB dosage • Add aldosterone antagonist • Potassium supplements • Magnesium supplements
Hyponatraemia	▪ Fluid restriction • Stop thiazide diuretic or switch to loop diuretic, if possible • Reduce dose/stop loop diuretics if possible • Consider AVP antagonist, e.g. tolvaptan if available • i.v. Inotropic support • Consider ultrafiltration
Hyperuricaemia/gout	▪ Consider allopurinol • For symptomatic gout use colchicine for pain relief • Avoid NSAIDs
Hypovolaemia/dehydration	▪ Assess volume status • Consider diuretic dosage reduction
Insufficient response or diuretic resistance	▪ Check compliance and fluid intake • Increase dose of diuretic • Consider switching from furosemide to bumetanide or torasemide • Add aldosterone antagonist • Combine loop diuretic and thiazide/metolazone • Administer loop diuretic twice daily or on empty stomach • Consider short-term i.v. infusion of loop diuretic
Renal failure (excessive rise in urea/BUN and/or creatinine)	▪ Check for hypovolaemia/dehydration • Exclude use of other nephrotoxic agents, e.g. NSAIDs, trimethoprim • Withhold aldosterone antagonist • If using concomitant loop and thiazide diuretic stop thiazide diuretic • Consider reducing dose of ACEI/ARB • Consider ultrafiltration

Diuretic dosages

Diuretics	Initial dose (mg)		Usual daily dose (mg)	
Loop diuretics*				
▪ furosemide	20 - 40		40 - 240	
▪ bumetanide	0.5 - 1.0		1 - 5	
▪ torasemide	5 - 10		10 - 20	
Thiazides**				
▪ bendroflumethiazide	2.5		2.5 - 10	
▪ hydrochlorothiazide	25		12.5 - 100	
▪ metolazone	2.5		2.5 - 10	
▪ indapamide	2.5		2.5 - 5	
Potassium-sparing diuretics***				
	+ ACEI/ARB	– ACEI/ARB	+ ACEI/ARB	– ACEI/ARB
▪ spironolactone/ eplerenone	12.5 - 25	50	50	100 - 200
▪ amiloride	2.5	5	20	40
▪ triamterene	25	50	100	200

* Dose might need to be adjusted according to volume status/weight; excessive doses may cause renal impairment and ototoxicity.

** Do not use thiazides if eGFR < 30 mL/min, except when prescribed synergistically with loop diuretics.

*** Aldosterone antagonists should always be preferred to other potassium sparing diuretics.

Practical considerations in treatment with loop diuretics:

Problems	Suggested actions
Hypokalaemia/ hypomagnesaemia	▪ increase ACEI/ARB dosage ▪ add aldosterone antagonist ▪ potassium supplements ▪ magnesium supplements
Hyponatraemia	▪ water restriction ▪ stop thiazide diuretic or switch to loop diuretic, if possible ▪ reduce dosage/stop loop diuretics if possible ▪ consider AVP antagonist e.g. tolvaptan if available ▪ i.v. inotropic support ▪ consider ultrafiltration
Hyperuricaemia/gout	▪ consider allopurinol ▪ for symptomatic gout use colchicine for pain relief ▪ avoid NSAIDs
Hypovolaemia/ dehydration	▪ assess volume status ▪ consider diuretic dosage reduction
Insufficient response or diuretic resistance	▪ check compliance and fluid intake ▪ increase dose of diuretic ▪ consider switching from furosemide to bumetanide or torasemide ▪ add aldosterone antagonist ▪ combine loop diuretic and thiazide ▪ administer loop diuretic twice daily or on empty stomach ▪ consider short-term i.v. infusion of loop diuretic
Renal failure (excessive rise in urea/BUN and/or creatinine)	▪ check for hypovolaemia/dehydration ▪ exclude use of other nephrotoxic agents e.g. NSAIDs, trimethoprim ▪ withhold aldosterone antagonist ▪ if using concomitant loop and thiazide diuretic stop thiazide diuretic ▪ consider reducing dose of ACEI/ARB ▪ consider ultrafiltration

Management of arterial hypertension in HF

In hypertensive patients with evidence of LV dysfunction:

- systolic and diastolic blood pressure should be carefully controlled with a therapeutic target of ≤ 140/90 and ≤ 130/80 mmHg in diabetics and high risk patients.
- anti-hypertensive regimens based on renin-angiotensin system antagonists (ACEI or ARBs) are preferable.

In hypertensive patients with HFPEF:

- aggressive treatment (often with several drugs with complementary mechanisms of action) is recommended.
- ACEI and/or ARBs should be considered the first-line agents.

Class I recommendations for devices in patients with LV systolic dysfunction

ICD	
Prior resuscitated cardiac arrest	Class I Level A
Ischaemic aetiology and >40 days of MI	Class I Level A
Non-ischaemic aetiology	Class I Level B
CRT	
NYHA Class III/IV and QRS >120 ms	Class I Level A
To improve symptoms/reduce hospitalization	Class I Level A
To reduce mortality	Class I Level A

Management of patients with heart failure and atrial fibrillation

General recommendations

- Precipitating factors and co-morbidities should be identified
- HF treatment should be optimized

Rhythm control

- Immediate electrical cardioversion is recommended for patients with new-onset AF and myocardial ischaemia, symptomatic hypotension or symptoms of pulmonary congestion or rapid ventricular response not controlled by appropriate pharmacological measures

Rate control

- Digoxin alone or in combination with β-blocker is recommended

Prevention of thromboembolism

- Antithrombotic therapy is recommended, unless contraindicated
- Optimal approach should be based on risk stratification: in patients at highest risk of stroke [prior stroke, transient ischaemic attack (TIA), or systemic embolism] oral anticoagulant therapy with a vitamin K antagonist is recommended

Management of arterial hypertension in patients with heart failure

In hypertensive patients with evidence of LV dysfunction

- Systolic and diastolic blood pressure should be carefully controlled with a therapeutic target of ≤140/90 and ≤130/80 mmHg in diabetics and high risk patients
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In hypertensive patients with HFPEF:

- Aggressive treatment (often with several drugs with complementary mechanisms of action) is recommended
- ACEIs and/or ARBs should be considered the first-line agents

- **ACUTE HF** is defined as a rapid onset or change in the signs and symptoms of HF, resulting in the need of urgent therapy

- It may present as new HF or worsening HF in the presence of chronic HF

- It may be associated with worsening symptoms or signs or as a medical emergency such as acute pulmonary oedema

- Multiple cardiovascular and non-cardiovascular morbidities may precipitate AHF

Causes and precipitating factors of AHF

Ischaemic heart disease

- Acute coronary syndromes
- Mechanical complications of acute MI
- Right ventricular infarction

Valvular

- Valve stenosis
- Valvular regurgitation
- Endocarditis
- Aortic dissection

Myopathies

- Postpartum cardiomyopathy
- Acute myocarditis

Hypertension/arrhythmia

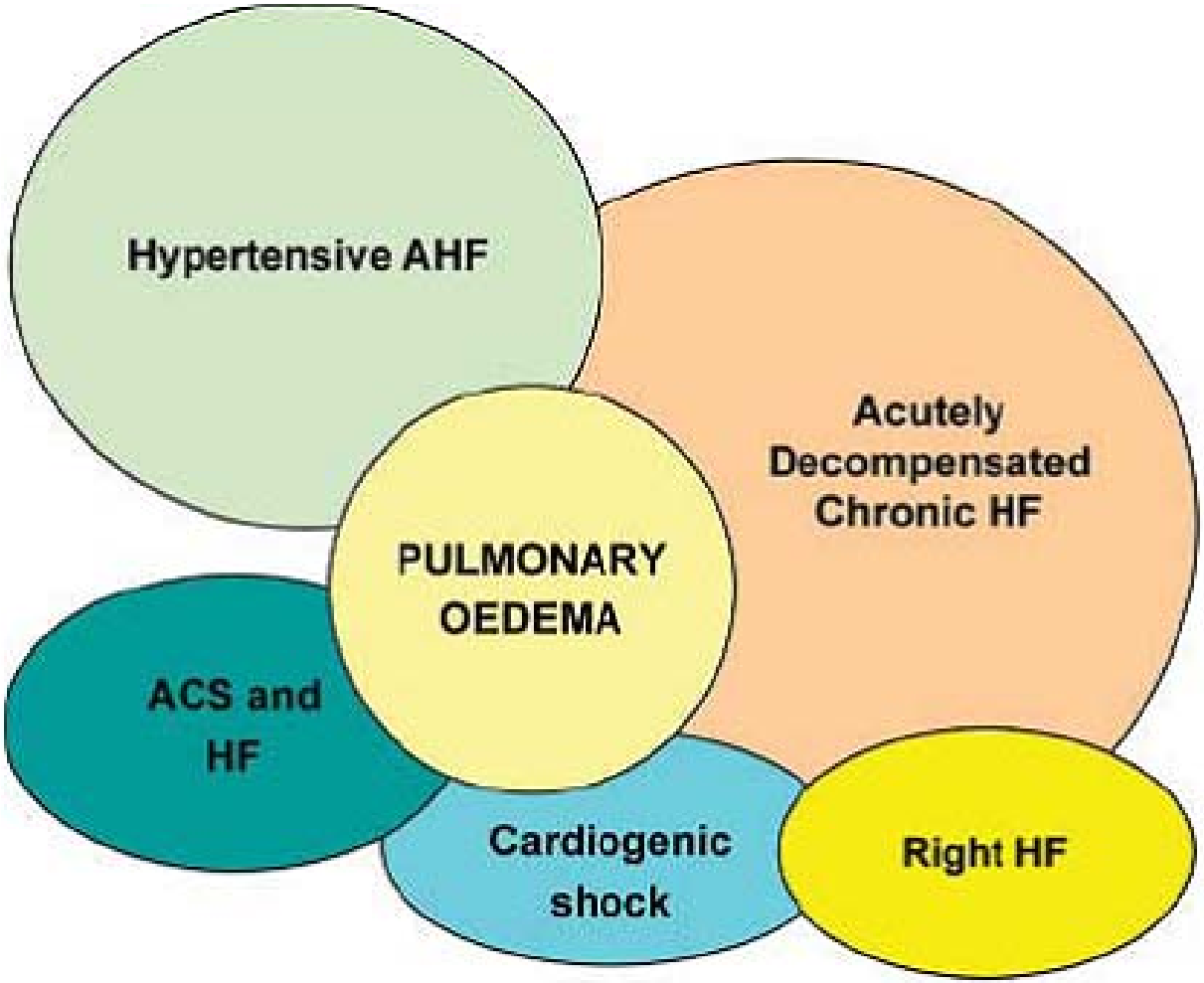
- Hypertension
- Acute arrhythmia

Circulatory failure

- Septicaemia
- Thyrotoxicosis
- Anaemia
- Shunts
- Tamponade
- Pulmonary embolism

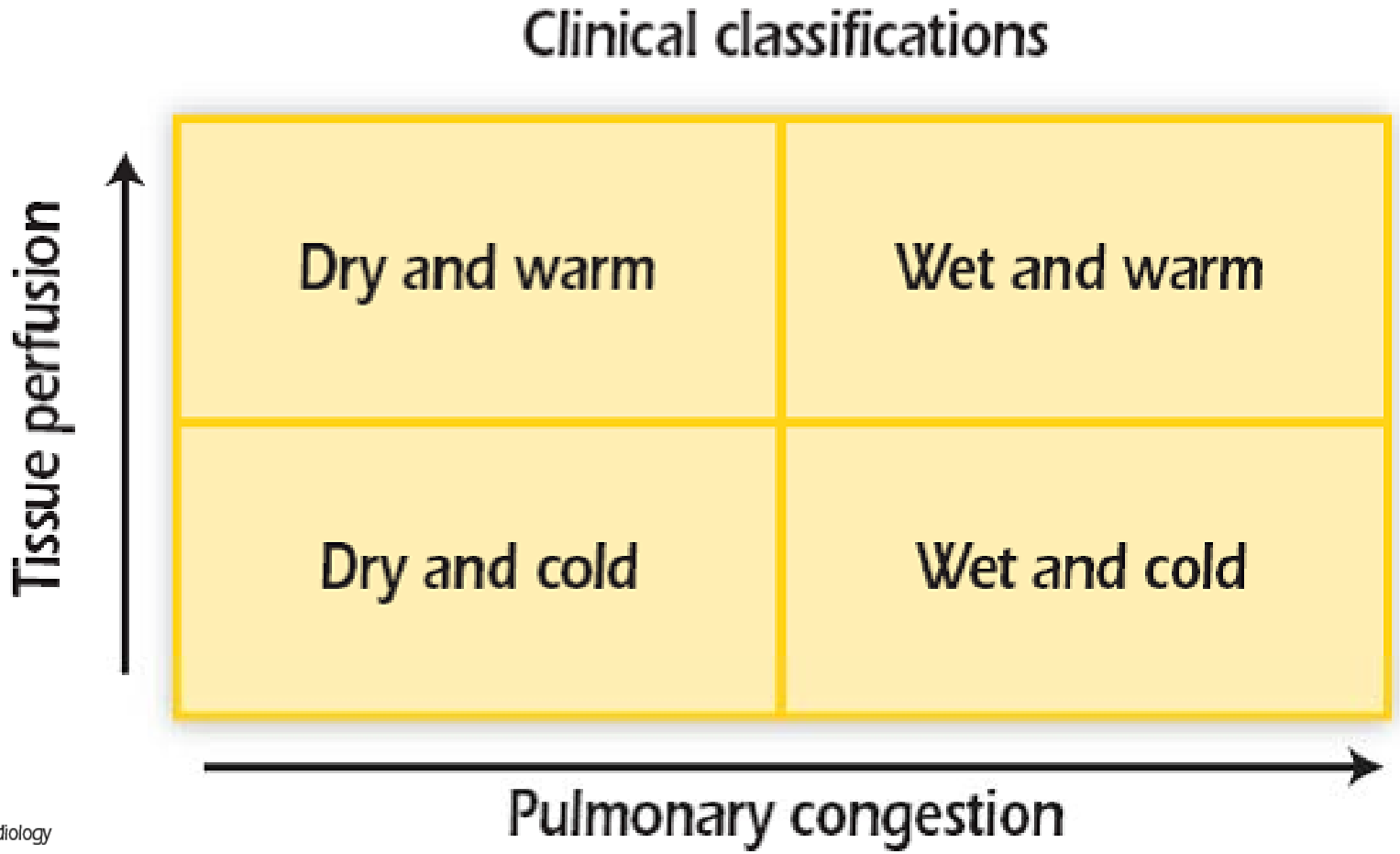
Decompensation of preexisting chronic HF

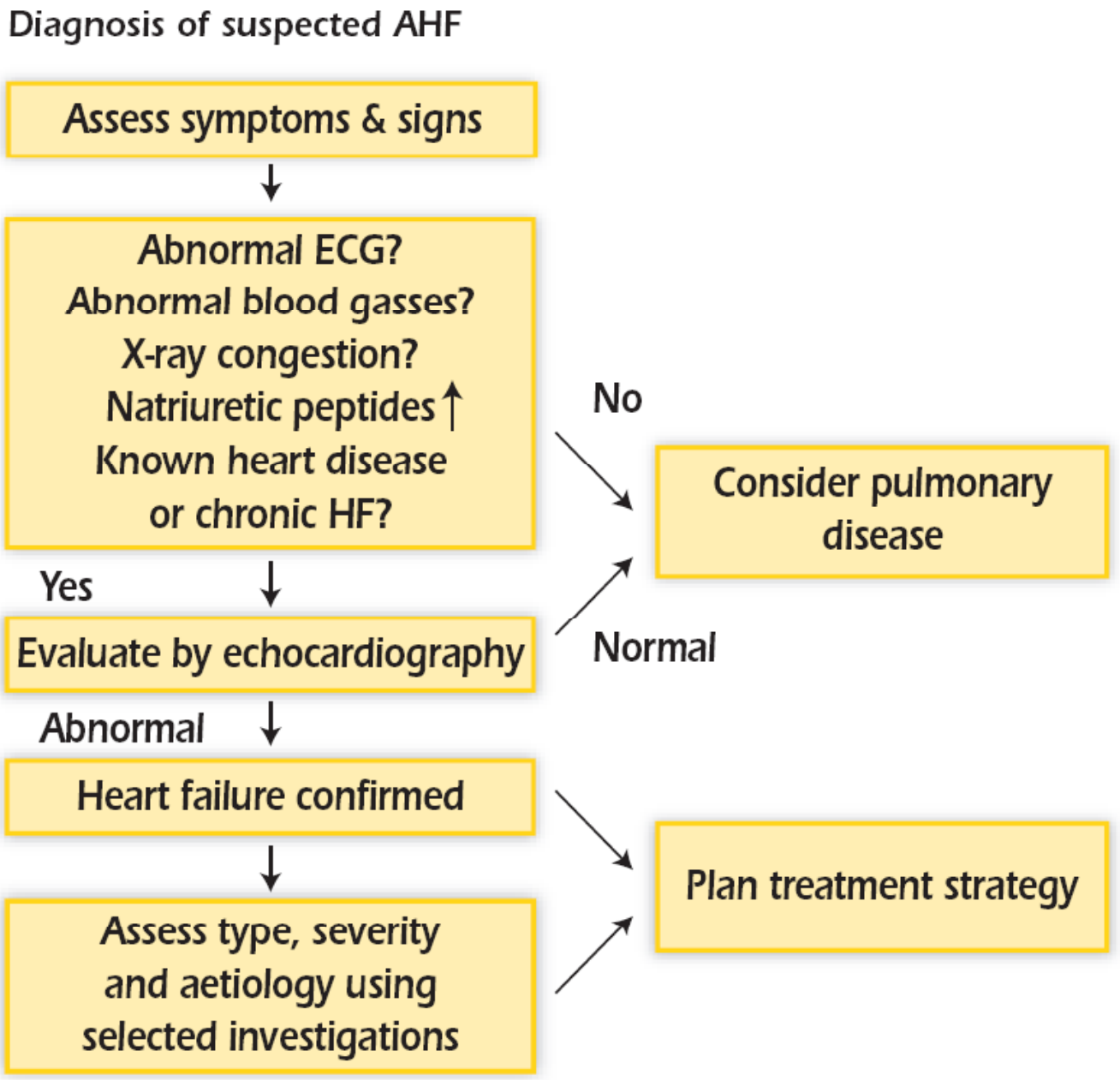
- Lack of adherence
- Volume overload
- Infections, especially pneumonia
- Cerebrovascular insult
- Surgery
- Renal dysfunction
- Asthma, COPD
- Drug abuse
- Alcohol abuse



Clinical classification of acute heart failure. Modified from reference 205.

A clinical assessment of patients with AHF





Goals of treatment in acute heart failure

- **Immediate (ED/ICU/CCU)**

  Improve symptoms

  Restore oxygenation

  Improve organ perfusion and haemodynamics

  Limit cardiac/renal damage

  Minimize ICU length of stay

- **Intermediate (in hospital)**

  Stabilize patient and optimize treatment strategy

  Initiate appropriate (life-saving) pharmacological therapy

  Consider device therapy in appropriate patients

  Minimize hospital length of stay

- **Long-term and pre-discharge management**

  Plan follow-up strategy

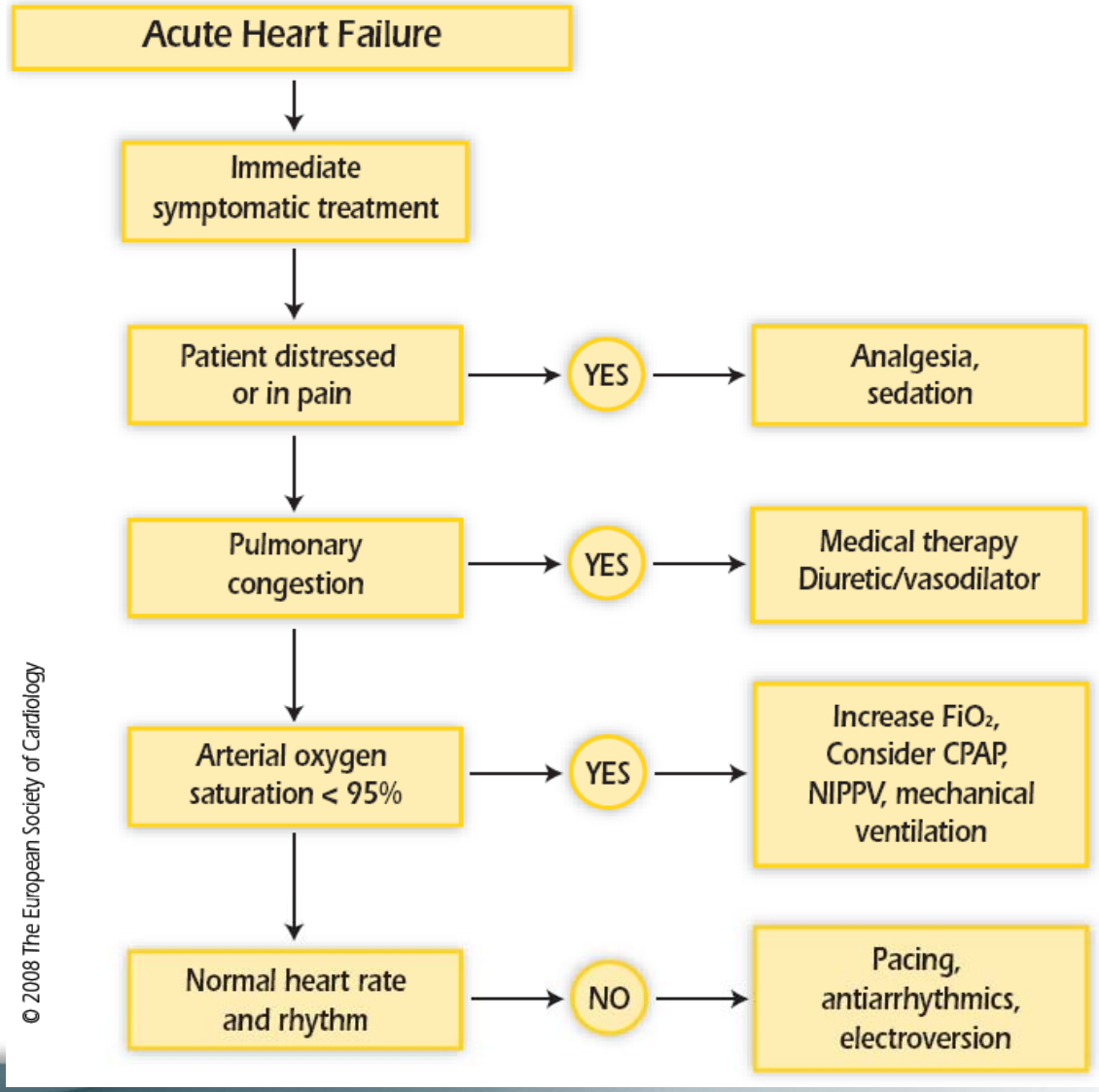
  Educate and initiate appropriate lifestyle adjustments

  Provide adequate secondary prophylaxis

  Prevent early readmission

  Improve quality of life and survival

Initial treatment algorithm in AHF



Goals of treatment in AHF

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- **Long-term and predischarge management**

  Plan follow-up strategy
  Educate and initiate appropriate lifestyle adjustments
  Provide adequate secondary prophylaxis
  Prevent early readmission
  Improve quality of life and survival

Indications and dosing of diuretics in AHF

Fluid retention	Diuretic	Daily Dose (mg)	Comments
Moderate	furosemide or bumetanide or torasemide	20 - 40 0.5 - 1 10 - 20	Oral or i.v. according to clinical symptoms Titrate dose according to clinical response - Monitor K, Na, creatinine, blood pressure
Severe	furosemide furosemide infusion bumetanide torasemide	40 - 100 (5 - 40 mg/h) 1 - 4 20 - 100	i.v. Increase dose. better than very high bolus doses oral or i.v. oral
Refractory to loop diuretic	add hydrochlorothiazide or metolazone or spironolactone	50 - 100 2.5 - 10 25 - 50	Combination better than very high dose of loop diuretics MTZ more potent if creatinine clr < 30ml/min Spironolactone best choice if no renal failure and normal or low serum potassium
With alkalosis	acetazolamide	0.5 mg	i.v.
Refractory to loop diuretics and thiazides	add dopamine (renal vasodilation) or dobutamine		Consider ultrafiltration or haemodialysis if coexisting renal failure Hyponatraemia

Indications and dosing of i.v. vasodilators in acute heart failure

Vasodilator	Indication	Dosing	Main side-effects	Other
Nitroglycerine	Pulmonary congestion/oedema BP >90 mmHg	Start 10–20 μg/min, increase up to 200 μg/min	Hypotension, headache	Tolerance on continuous use
Isosorbide dinitrate	Pulmonary congestion/oedema BP >90 mmHg	Start with 1 mg/h, increase up to 10 μg/h	Hypotension, headache	Tolerance on continuous use
Nitroprusside	Hypertensive HF congestion/ oedema BP >90 mmHg	Start with 0.3 μg/kg/min and increase up to 5 μg/kg/min	Hypotension, isocyanate toxicity	Light sensitive
Nesiritide*	Pulmonary congestion/oedema BP >90 mmHg	Bolus 2 μg/kg + infusion 0.015–0.03 μg/kg/min	Hypotension	

*Not available in many ESC countries.

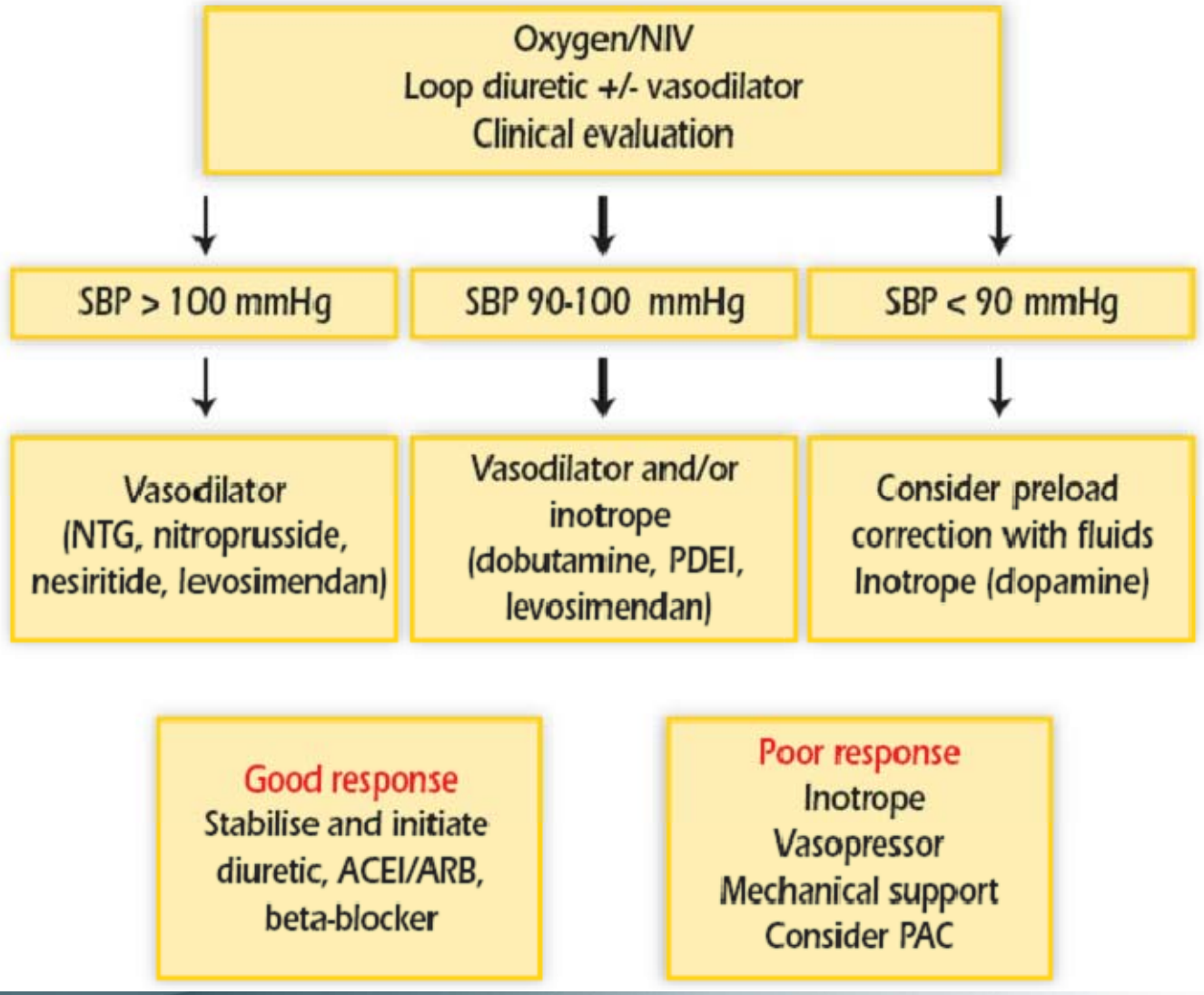
Dosing of positive inotropic agents in AHF

	Bolus	Infusion rate
Dobutamine	No	2 to 20 µg/kg/min (ß+)
Dopamine	No	< 3 µg/kg/min: renal effect (δ+) 3 - 5 µg/kg/min: inotropic (ß+) > 5 µg/kg/min: (ß+), vasopressor (α+)
Milrinone	25 - 75 µg/kg over 10 - 20 min	0.375 - 0.75 µg/kg/min
Enoximone	0.25 - 0.75 mg/kg	1.25 - 7.5 µg/kg/min
Levosimendan*	12 µg/kg over 10 min (optional)**	0.1 µg/kg/min which can be decreased to 0.05 or increased to 0.2 µg/kg/min
Norepinephrine	No	0,2 - 1,0 µg/kg/min
Epinephrine	Bolus: 1 mg can be given i.v. during resuscitation, repeated every 3 - 5 min	0.05 - 0.5 µg/kg/min

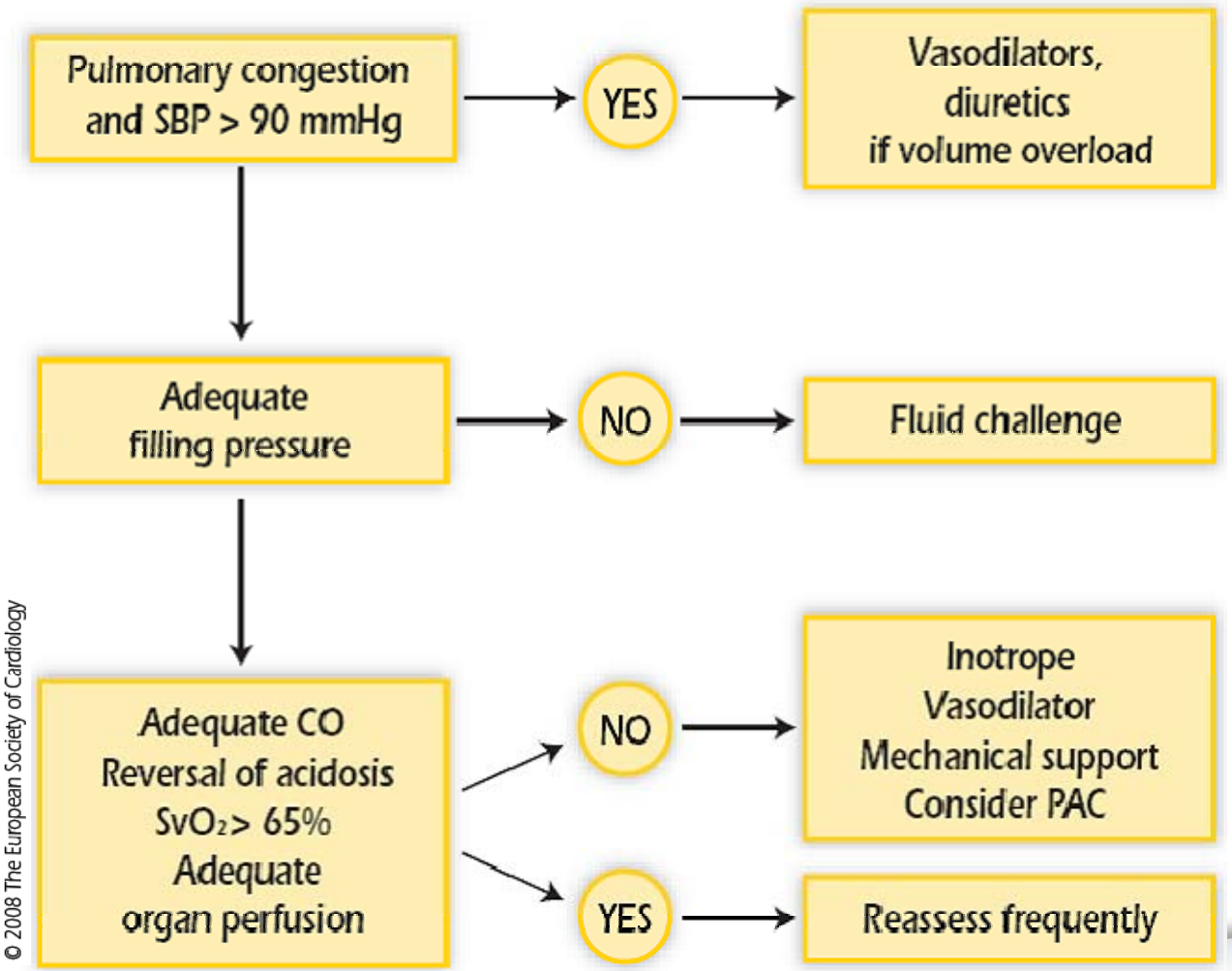
* This agent also has vasodilator properties.

** In hypotensive patients, (SBP < 100 mmHg) initiation of therapy without a bolus is recommended.

Treatment strategy in AHF according to systolic blood pressure



Treatment strategy in AHF according to LV filling pressure



Treatment goals and strategies during the course of the patient's journey

Phase	Diagnostic strategy	Action	Goals	Players
Acute	Assess clinical status Identify cause of symptoms	Treat and stabilize Initiate monitoring Plan required interventions	Stabilize, admit, and triage to appropriate department	Paramedics Primary care/ER physicians Intensivists Nurses Cardiologists
Subacute	Assess cardiac function Identify aetiology and co-morbidities	Initiate chronic medical treatment Perform additional diagnostics Perform indicated procedures	Shorten hospitalization Plan post-discharge follow-up	Hospital physicians Cardiologists CV nurses HF Management team
Chronic	Target symptoms, adherence, and prognosis Identify decompensation early	Optimize pharmacological and device treatment Support self-care behaviour Remote monitoring	Reduce morbidity and mortality	Primary care physicians HF Management team Cardiologists
End of life	Identify patient concerns and symptoms	Symptomatic treatment Plan for long-term care	Palliation Provide support for patients and family	Palliative care team

Recommended Components of HF Management Programmes

- Multidisciplinary approach frequently led by HF nurses in collaboration with physicians and other related services
- First contact during hospitalisation, early follow-up after discharge through clinic and home-based visits, telephone support and remote monitoring
- Target high-risk, symptomatic patients
- Increased access to health care (telephone, remote monitoring and follow-up)
- Facilitate access during episodes of decompensation
- Optimised medical management
- Access to advanced treatment options
- Adequate patient education with special emphasis on adherence and self-care management
- Patient involvement in symptom monitoring and flexible diuretic use
- Psychosocial support to patients and family and/or caregiver

Advantages and disadvantages of different models of heart failure programmes

	Advantages	Disadvantages
Clinic visits	• Convenient with medical expertise, facilities and equipment available. • Facilitates diagnostic investigation and adjustments of treatment strategy	• Frail, non-ambulatory patients not suitable for out-patient follow-up
Home care	• Access to immobile patients • More reliable assessment of the patient's needs, capabilities and adherence to treatment in their own home environment • Convenient for a follow-up visit shortly after hospitalization	• Time consuming travel for the HF team • Transportation and mobile equipment required • Nurses face medical responsibilities alone and may have difficulty contacting the responsible physician
Telephone support	• Low cost, time saving and convenient both for the team and the patient	• Difficult to assess symptoms and signs of heart failure and no tests can be performed • Difficult to provide psychosocial support, adjust treatment and educate patients
Remote monitoring	• Provides permitting informed clinical decisions • Need is increasing as care shifts into patients' homes • New equipment and technology becoming rapidly available	• Requires education on the use of the equipment • Time-consuming for HF team • Difficult for patients with cognitive disability • Most helpful measurements not known

Steps in the process of providing palliative care

Patient features	> 1 episode of decompensation/6 months Need for frequent or continual i.v. support Chronic poor quality of life with NYHA IV symptoms Signs of cardiac cachexia Clinically judged to be close to the end of life
Confirm diagnosis	Essential to ensure optimal treatment.
Patient education	Principles of self-care maintenance and management of HF
Establish an advanced care plan	Designed with the patient and a family member. Reviewed regularly and includes the patients' preferences for future treatment options.
Services should be organised	The patients' care within the multidisciplinary team, to ensure optimal pharmacological treatment, self-care management and to facilitate access to supportive services.
Symptom management	Requires frequent assessment of patients' physical, psychological, social and spiritual needs. Patients frequently have multiple co-morbidities that need to be identified.
Identifying end-stage HF	Confirmation of end-stage HF is advisable to ensure that all appropriate treatment options have been explored a plan for the terminal stage of illness should be agreed upon.
Breaking bad news to the patient and family	Explaining disease progression and a change in treatment emphasis is a sensitive issue and must be approached with care.
Establishing new goals of care	End of life care should include avoidance of circumstances which may detract from a peaceful death. All current pharmacological treatment and device programmes should be considered. Resuscitation orders should be clear.

- Clinicians responsible for managing patients with HF must frequently make treatment decisions without adequate evidence or consensus expert opinion

Some examples

- Does any specific treatment of these co-morbidities in HF reduce morbidity and mortality?
  - renal dysfunction
  - anaemia
  - diabetes
  - depression
  - disordered breathing during sleep

- Should ACE inhibitors always be prescribed before beta-blockers?

- Aldosterone antagonist or ARB next in symptomatic patients on ACE inhibitor and beta-blocker?

- Tailoring therapy by natriuretic peptide levels?
- Aldosterone antagonist in mild symptoms?
- Is quadruple therapy (ACE inhibitor, ARB, aldosterone antagonist and beta-blocker) better than use of three agents?

- Does revascularisation in hibernating myocardium improve clinical outcomes?
- What criteria with aortic stenosis/regurgitation or mitral regurgitation support valvular surgery?
- Restoring sinus rhythm in patients with atrial fibrillation and either low EF or HFPEF?

- CRT-D or CRT-P in symptomatic HF and a wide QRS complex?

- Role for echo assessment of dyssynchrony in CRT selection?

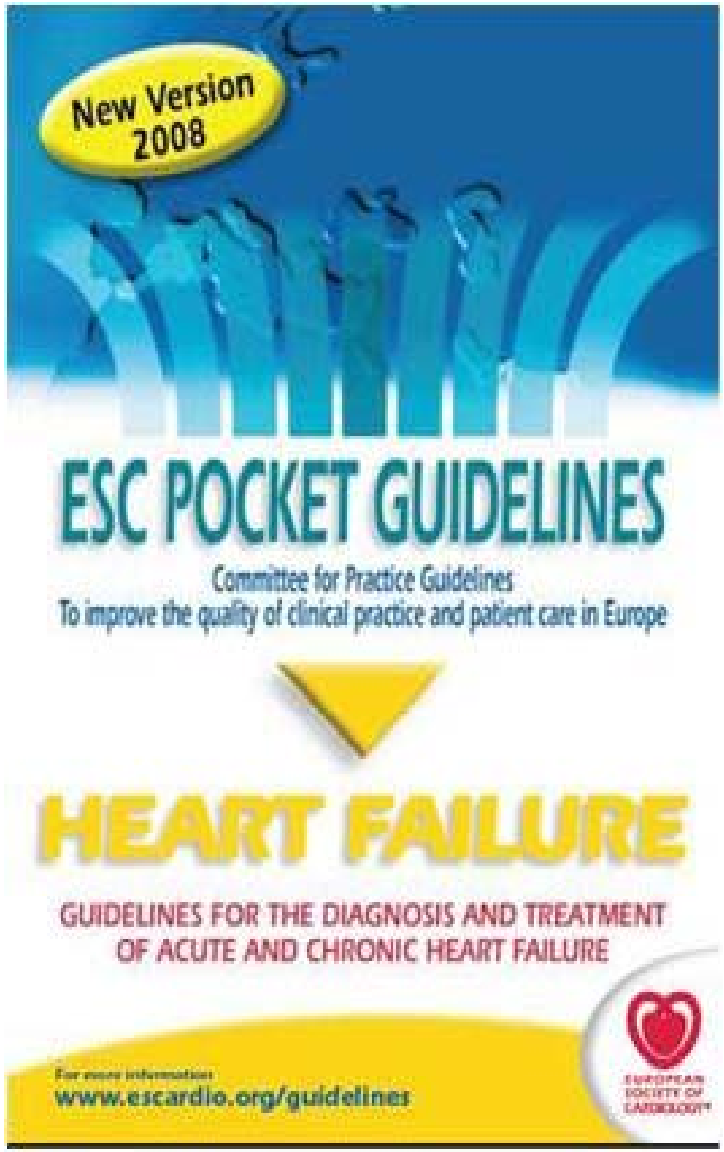
- CRT in symptomatic patients with a low EF, and a QRS width <120 msec?
- ICD in HF and an EF>35%?
- How should patients be selected for bridge to recovery with an LVAD?

- Which components of HF management programmes are most important?

- What aspects of remote monitoring best detect early decompensation?

Acute HF

- Which vasodilator is most efficacious?
- Which inotrope is most efficacious?
- The role of NIV in AHF?
- Management of beta-blockers in acute decompensation?



ESC Pocket Guidelines

Diagnosis and Treatment of Acute and Chronic Heart Failure*

The Task Force on Heart Failure of the European Society of Cardiology (ESC)
Developed in collaboration with the Heart Failure Association of the ESC (HFA)
and endorsed by the European Society of Intensive Care Medicine (ESICM)

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Special thanks to Tessa Baak for her contribution.

*Adapted from the ESC Guidelines on Diagnosis and Treatment of Acute and Chronic Heart Failure 2008 (European Heart Journal 2008;doi:10.1093/eurheartj/ehm309

”The very essence of cardiovascular medicine is the recognition of early heart failure”

Sir Thomas Lewis 1933

	CONSENSUS	SOLVD-T	RALES	CHARM-Alternative	CHARM-Added	Val-HeFT
Intervention	enalapril	enalapril	spironolactone	candesartan	candesartan	valsartan
n =	253	2569	1663	2028	2548	5010
Mean age (yr)	71	61	65	67	64	63
Female (%)	30	20	27	32	21	20
NYHA class (%) I II III IV	0 0 0 100	11 57 30 2	0 0.5 71 29	0 48 49 4	0 24 73 3	0 62 36 2
Mean LVEF (%)	NR	25	25	30	28	31
History (%) • CHD • Hypertension • Diabetes mellitus • AF	73 22 23 50	71 42 26 10	55 NR NR NR	62 50 27 25	56 48 30 26	57 NR 25 12
Treatment (%) • Diuretic • Digitalis • ACE inhibitor • Beta-blocker • Aldosterone antagonist	98* 93 N/A 3 53 0	86 67 N/A 8 9** 0	100* 74 95 11 N/A 0	86 46 N/A 55 24 N/A	90 58 100 55 17 N/A	86 88 93 35 2 N/A

	CONSENSUS	SOLVD-T	RALES	CHARM-Alternative	CHARM-Added	Val-HeFT
Intervention	enalapril	enalapril	spironolactone	candesartan	candesartan	valsartan
Mean follow-up (months)	6.5	41.4	24	33.7	41	23
Rate of death in placebo group during follow up (%)	53.9%	39.7	45.9	29.2	32.4	19.4
RRR (%)	27%	16 (5,26)	30 (18,40)	13 (-3, 26)	11 (-2,23)	+2 (-12, +18)
ARR (%)	14.6%	4.5	11.4	3.0	2.9	+0.3
NNT	7	22	9	33	35	N/A
Rate of death or HF hospitalisation in placebo group during follow-up (%)	_	57.3	NR	42.7	46.2	32.1**
RRR	_	26 (18,34)	-	20 (8,30)	13 (2,22)	13 (3,23)
ARR	_	9.6	-	6.1	4.0	3.3
NNT	_	10	-	16	25	30