Scompenso cardiaco

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Definizione di Braunwald

- Anomalia della funzione cardiaca tale che il cuore non è in grado di pompare sangue in maniera adeguata alle richieste metaboliche o di farlo solo a spese di un aumento della P di riempimento (EDP) e/o di un'attivazione dei sistemi neuroendocrini

2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

HF is a clinical syndrome characterized by typical symptoms that may be accompanied by signs caused by a structural and/or functional cardiac abnormality, resulting in a reduced cardiac output and/or elevated intracardiac pressures at rest or during stress.

The current definition of HF restricts itself to stages at which clinical symptoms are apparent. Before clinical symptoms become apparent, patients can present with asymptomatic structural or functional cardiac abnormalities [systolic or diastolic left ventricular (LV) dysfunction], which are precursors of HF. Recognition of these precursors is important because they are related to poor outcomes, and starting treatment at the precursor stage may reduce mortality in patients with asymptomatic systolic LV dysfunction

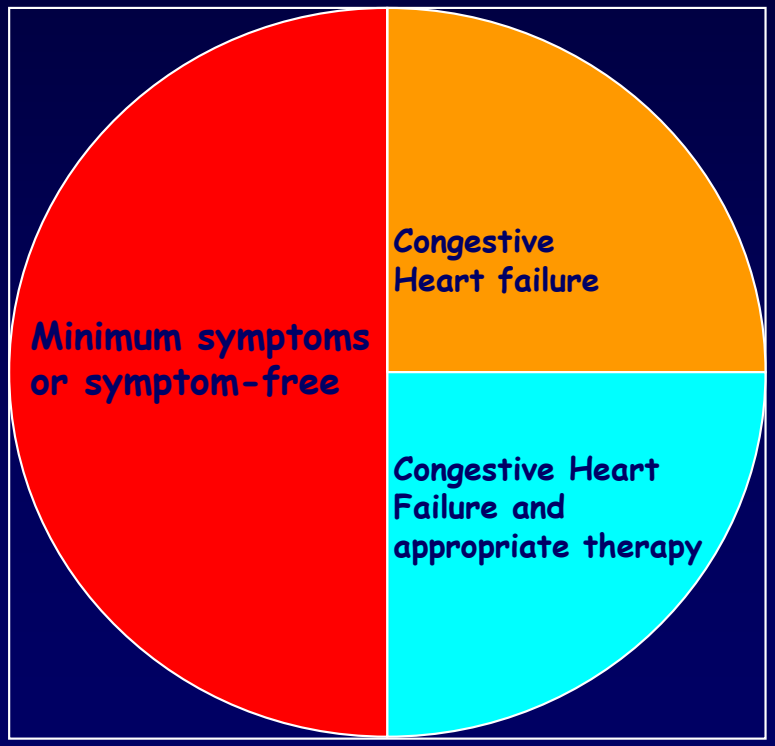
Epidemiologia e storia naturale dello scompenso cardiaco in Italia

In Europa ci sono circa 700 milioni di persone di cui almeno 10 milioni sono affetti da scompenso cardiaco. Circa la metà di loro moriranno entro i prossimi 4 anni.

In Italia: 1 milione di persone affette da scompenso. Nel 30% dei casi sono persone oltre i 65 anni e la maggior causa è la cardiopatia ischemica. Ci sono circa 170 mila ospedalizzazioni ogni anno. Incidenza: 1 nuovo caso ogni 1000 persone. In Italia il costo totale a causa dello scompenso è stimato essere circa 1,4% della spesa nazionale per la sanità

La disfunzione asintomatica

Una nuova sfida epidemiologica



	Sample Size (Age Range)	EF Method	Definition of Abnormal	Prevalence (%) VE	M mode	2D
Cardiovascular Health Study	5,201 (65-100)	VE*	"Abnormal EF" by VE	3.7	—	—
Glasgow	1,467 (25-75)	2D†	2 SD < "nl" mean; <35%	—	—	7.7
Regensburg	1,566 (25-75)	M mode	2 SD < "nl" mean; <48%	—	2.8	—
Rotterdam	2,267 (55-94)	M mode	EF <42.5%	—	3.7	—
Olmsted PAVD	2,042 (45-96)	M mode, 2D, and VE	2 SD < "nl" mean: ≤50% for 2D, ≤57% for M mode, ≤53% for VE	6.2	4.8	5.1
Strong Heart Study	1,066 (49-78)	M mode	2 S.D. < "nl" mean; <54%	—	16.1	—

Abbreviations: 2D, 2-dimensional; SD, standard deviation.
*Visual estimate.
†2-dimensional echocardiogram.

Rodeheffer RJ, Journal of Cardiac Failure Vol 8. 6 Suppl. 2002

Eziologia

CAUSE PIU' FREQUENTI

CARDIOPATIA ISCHEMICA

IPERTENSIONE ARTERIOSA

DISEASED MYOCARDIUM		
Ischaemic heart disease	Myocardial scar	
	Myocardial stunning/hibernation	
	Epicardial coronary artery disease	
	Abnormal coronary microcirculation	
	Endothelial dysfunction	
Toxic damage	Recreational substance abuse	Alcohol, cocaine, amphetamine, anabolic steroids.
	Heavy metals	Copper, iron, lead, cobalt.
	Medications	Cytostatic drugs (e.g. anthracyclines), immunomodulating drugs (e.g. interferons monoclonal antibodies such as trastuzumab, cetuximab), antidepressant drugs, antiarrhythmics, non-steroidal anti-Inflammatory drugs, anaesthetics.
	Radiation	
Immune-mediated and inflammatory damage	Related to infection	Bacteria, spirochaetes, fungi, protozoa, parasites (Chagas disease), rickettsiae, viruses (HIV/AIDS).
	Not related to infection	Lymphocytic/giant cell myocarditis, autoimmune diseases (e.g. Graves' disease, rheumatoid arthritis, connective tissue disorders, mainly systemic lupus erythematosus), hypersensitivity and eosinophilic myocarditis (Churg–Strauss).
Infiltration	Related to malignancy	Direct infiltrations and metastases.
	Not related to malignancy	Amyloidosis, sarcoidosis, haemochromatosis (iron), glycogen storage diseases (e.g. Pompe disease), lysosomal storage diseases (e.g. Fabry disease).
Metabolic derangements	Hormonal	Thyroid diseases, parathyroid diseases, acromegaly, GH deficiency, hypercortisolaemia, Conn's disease, Addison disease, diabetes, metabolic syndrome, phaeochromocytoma, pathologies related to pregnancy and peripartum.
	Nutritional	Deficiencies in thiamine, L-carnitine, selenium, iron, phosphates, calcium, complex malnutrition (e.g. malignancy, AIDS, anorexia nervosa), obesity.
Genetic abnormalities	Diverse forms	HCM, DCM, LV non-compaction, ARVC, restrictive cardiomyopathy, muscular dystrophies and laminopathies ⇨ counselling genetico

ABNORMAL LOADING CONDITIONS		
Hypertension		
Valve and myocardium structural defects	Acquired	Mitral, aortic, tricuspid and pulmonary valve diseases.
	Congenital	Atrial and ventricular septum defects and others (for details see a respective expert document).
Pericardial and endomyocardial pathologies	Pericardial	Constrictive pericarditis Pericardial effusion
	Endomyocardial	HES, EMF, endocardial fibroelastosis.
High output states		Severe anaemia, sepsis, thyrotoxicosis, Paget's disease, arteriovenous fistula, pregnancy.
Volume overload		Renal failure, iatrogenic fluid overload.
ARRHYTHMIAS		
Tachyarrhythmias		Atrial, ventricular arrhythmias.
Bradyarrhythmias		Sinus node dysfunctions, conduction disorders.

Table 3.1 Definition of heart failure with preserved (HFpEF), mid-range (HFmrEF) and reduced ejection fraction (HFrEF)

Type of HF		HFrEF	HFmrEF	HFpEF
CRITERIA	1	Symptoms ± Signs[a]	Symptoms ± Signs[a]	Symptoms ± Signs[a]
	2	LVEF <40%	LVEF 40–49%	LVEF ≥50%
	3	–	1. Elevated levels of natriuretic peptides[b]; 2. At least one additional criterion: a. relevant structural heart disease (LVH and/or LAE), b. diastolic dysfunction	1. Elevated levels of natriuretic peptides[b]; 2. At least one additional criterion: a. relevant structural heart disease (LVH and/or LAE), b. diastolic dysfunction

[a]Signs may not be present in the early stages of HF (especially in HFpEF) and in patients treated with diuretics.
[b]BNP>35 pg/ml and/or NT-proBNP>125 pg/mL. in the acute setting, higher values should be used (*BNP* >100 pg/mL, *NT-proBNP* >300 pg/mL)

Patients with HFpEF generally do not have a dilated LV, but instead often have an increase in LV wall thickness and/or increased left atrial (LA) size as a sign of increased filling pressures. Most have additional 'evidence' of impaired LV filling or suction capacity, also classified as diastolic dysfunction, which is generally accepted as the likely cause of HF in these patients (hence the term 'diastolic HF'). However, most patients with HFrEF (previously referred to as 'systolic HF') also have diastolic dysfunction, and subtle abnormalities of systolic function have been shown in patients with HFpEF. ~~Hence the preference for stating preserved or reduced LVEF over~~ preserved or reduced 'systolic function'.

Compared with HFrEF, patients with HFpEF are older, more often women and more commonly have a history of hypertension and atrial fibrillation (AF), while a history of myocardial infarction is less common.

Patients with HFpEF and HFmrEF have impaired exercise tolerance, commonly accompanied by an augmented blood pressure response to exercise and chronotropic incompetence.

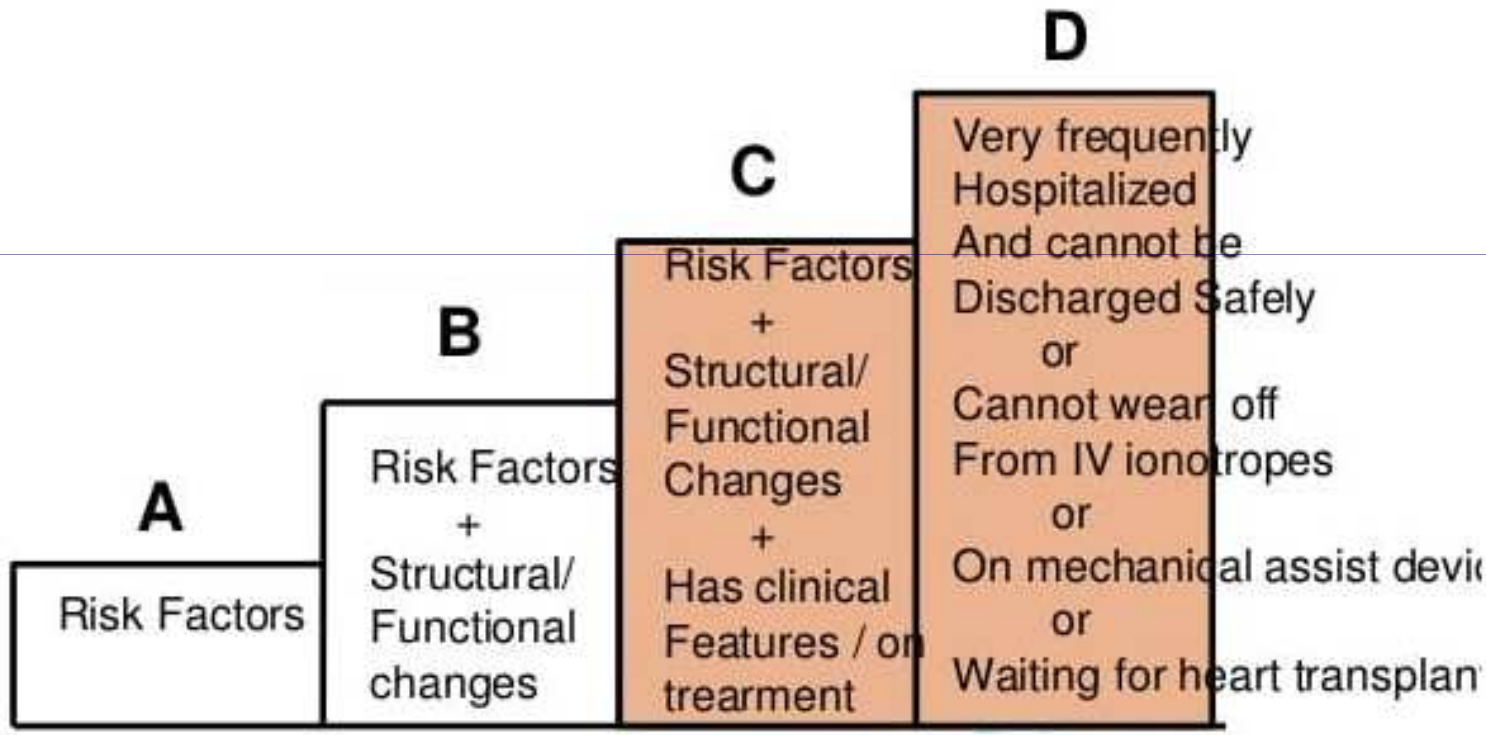
Symptoms and signs

Typical	More specific
Breathlessness Orthopnoea Paroxysmal nocturnal dyspnoea Reduced exercise tolerance Fatigue, tiredness, increased time to recover after exercise Ankle swelling	Elevated jugular venous pressure Hepatojugular reflux Third heart sound (gallop rhythm) Laterally displaced apical impulse
Less typical	**Less specific**
Nocturnal cough Wheezing Bloated feeling	Weight gain (>2 kg/week) Weight loss (in advanced HF) Tissue wasting (cachexia)
Loss of appetite Confusion (especially in the elderly) Depression Palpitations Dizziness Syncope Bendopnea	Cardiac murmur Peripheral oedema (ankle, sacral, scrotal) Pulmonary crepitations Reduced air entry and dullness to percussion at lung bases (pleural effusion) Tachycardia Irregular pulse Tachypnoea Cheyne Stokes respiration Hepatomegaly Ascites Cold extremities Oliguria Narrow pulse pressure

Delaying or preventing the development of overt heart failure or preventing death before the onset of symptoms

Treatment of hypertension is recommended to prevent or delay the onset of HF and prolong life.	I	A
Treatment with statins is recommended in patients with or at high-risk of CAD whether or not they have LV systolic dysfunction, in order to prevent or delay the onset of HF and prolong life.	I	A
Counselling and treatment for smoking cessation and alcohol intake reduction is recommended for people who smoke or who consume excess alcohol in order to prevent or delay the onset of HF.	I	C
Treating other risk factors of HF (e.g. obesity, dysglycaemia) should be considered in order to prevent or delay the onset of HF.	IIa	C
Empagliflozin should be considered in patients with type 2 diabetes in order to prevent or delay the onset of HF and prolong life.	IIa	B
ACE-I is recommended in patients with asymptomatic LV systolic dysfunction and a history of myocardial infarction in order to prevent or delay the onset of HF and prolong life.	I	A
ACE-I is recommended in patients with asymptomatic LV systolic dysfunction without a history of myocardial infarction, in order to prevent or delay the onset of HF.	I	B
ACE-I should be considered in patients with stable CAD even if they do not have LV systolic dysfunction, in order to prevent or delay the onset of HF.	IIa	A
Beta-blocker is recommended in patients with asymptomatic LV systolic dysfunction and a history of myocardial infarction, in order to prevent or delay the onset of HF or prolong life.	I	B
ICD is recommended in patients: a) with asymptomatic LV systolic dysfunction (LVEF ≤30%) of ischaemic origin, who are at least 40 days after acute myocardial infarction, b) with asymptomatic non-ischaemic dilated cardiomyopathy (LVEF ≤30%), who receive OMT therapy, in order to prevent sudden death and prolong life.	I	B

ACC/AHA stages of Heart Failure

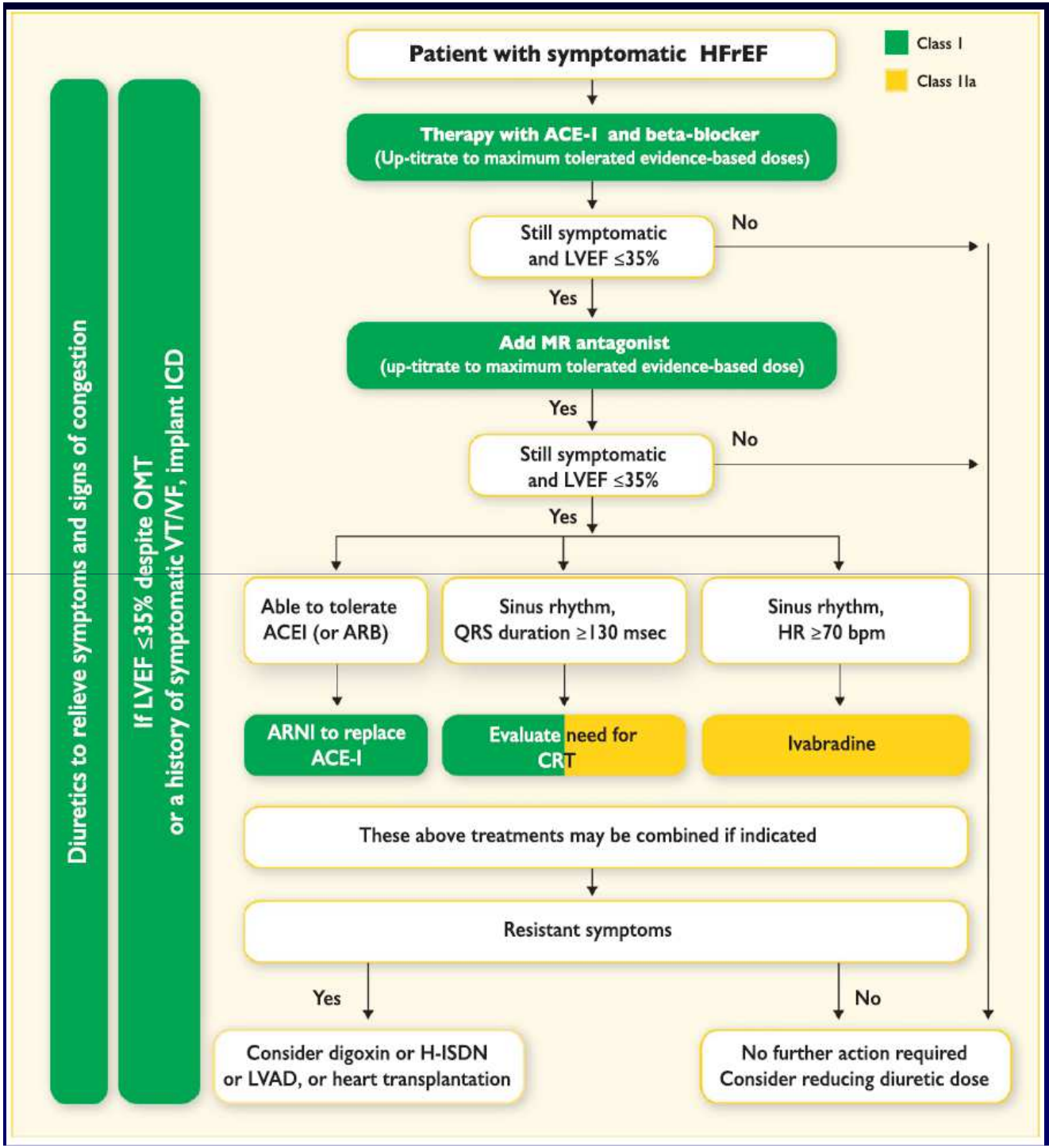


NYHA class II-IV

Treatments recommended in all symptomatic patients with heart failure with reduced ejection fraction

An ACE-I is recommended, in addition to a beta-blocker, for symptomatic patients with HFrEF to reduce the risk of HF hospitalization and death.	I	A
A beta-blocker is recommended, in addition an ACE-I, for patients with stable, symptomatic HFrEF to reduce the risk of HF hospitalization and death.	I	A
An MRA is recommended for patients with HFrEF, who remain symptomatic despite treatment with an ACE-I and a beta-blocker, to reduce the risk of HF hospitalization and death.	I	A

or ARB if ACEI is not tolerated/vsindicated



Symptomatic = NYHA Class II-IV

If ACE-I not tolerated/vsindicated, use ARB

If MRA not tolerated/vsindicated, use ARB

Diuretics		
Diuretics are recommended in order to improve symptoms and exercise capacity in patients with signs and/or symptoms of congestion.	I	B
Diuretics should be considered to reduce the risk of HF hospitalization in patients with signs and/or symptoms of congestion.	IIa	B
Angiotensin receptor neprilysin inhibitor		
Sacubitril/valsartan is recommended as a replacement for an ACE-I to further reduce the risk of HF hospitalization and death in ambulatory patients with HFrEF who remain symptomatic despite optimal treatment with an ACE-I, a beta-blocker and an MRA[d]	I	B
I*f*-channel inhibitor		
Ivabradine should be considered to reduce the risk of HF hospitalization or cardiovascular death in symptomatic patients with LVEF ≤35%, in sinus rhythm and a resting heart rate ≥70 bpm despite treatment with an evidence-based dose of beta-blocker (or maximum tolerated dose below that), ACE-I (or ARB), and an MRA (or ARB).	IIa	B
Ivabradine should be considered to reduce the risk of HF hospitalization and cardiovascular death in symptomatic patients with LVEF ≤35%, in sinus rhythm and a resting heart rate ≥70 bpm who are unable to tolerate or have contra-indications for a beta-blocker. Patients should also receive an ACE-I (or ARB) and an MRA (or ARB).	IIa	C
ARB		
An ARB is recommended to reduce the risk of HF hospitalization and cardiovascular death in symptomatic patients unable to tolerate an ACE-I (patients should also receive a beta-blocker and an MRA).	I	B
An ARB may be considered to reduce the risk of HF hospitalization and death in patients who are symptomatic despite treatment with a beta-blocker who are unable to tolerate an MRA.	IIb	C
Hydralazine and isosorbide dinitrate		
Hydralazine and isosorbide dinitrate should be considered in self-identified black patients with LVEF ≤35% or with an LVEF <45% combined with a dilated LV in NYHA Class III–IV despite treatment with an ACE-I a beta-blocker and an MRA to reduce the risk of HF hospitalization and death.	IIa	B
Hydralazine and isosorbide dinitrate may be considered in symptomatic patients with HFrEF who can tolerate neither an ACE-I nor an ARB (or they are contra-indicated) to reduce the risk of death.	IIb	B

[d]Patient should have elevated natriuretic peptides (plasma BNP ≥150 pg/mL or plasma NT-proBNP ≥600 pg/mL, or if HF hospitalization within the last 12 months, plasma BNP ≥100 pg/mL or plasma NT-proBNP ≥400 pg/mL) and able to tolerate enalapril 10 mg *b.i.d.*

Other treatments with less-certain benefits		
Digoxin		
Digoxin may be considered in symptomatic patients in sinus rhythm despite treatment with an ACE-I (or ARB), a beta-blocker and an MRA, to reduce the risk of hospitalization (both all-cause and HF-hospitalizations).	IIb	B
N-3 PUFA		
An n-3 PUFA preparation may be considered in symptomatic HF patients to reduce the risk of cardiovascular hospitalization and cardiovascular death.	IIb	B

n-3 polyunsaturated fatty acids

Only preparations with eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) as ethyl esters of at least 85% (850 mg/g) have shown an effect on the cumulative endpoint of cardiovascular death and hospitalization. No effect of n-3 PUFA preparations containing <850 mg/g has been shown in either HFrEF or post-myocardial infarction. n-3 PUFA preparations containing 850–882 mg of EPA and DHA as ethyl esters in the average ratio of 1 : 1.2 may be considered as an adjunctive therapy in patients with symptomatic HFrEF who are already receiving optimized recommended therapy with an ACEI (or ARB), a beta-blocker and an MRA.

	Starting dose (mg)	Target dose (mg)
ACE-I		
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.	10 *o.d.*
Beta-blockers		
Bisoprolol	1.25 *o.d.*	10 *o.d.*
Carvedilol	3.125 *b.i.d.*	25 *b.i.d.*[d]
Metoprolol succinate	12.5–25 *o.d.*	200 o.d.
Nebivolol	1.25 *o.d.*	10 *o.d.*
ARBs		
Candesartan	4–8 *o.d.*	32 *o.d.*
Valsartan	40 *b.i.d.*	160 *b.i.d.*
Losartan	50 *o.d.*	150 *o.d.*
MRAs		
Eplerenone	25 *o.d.*	50 *o.d.*
Spironolactone	25 *o.d.*	50 *o.d.*
ARNI		
Sacubitril/valsartan	49/51 *b.i.d.*	97/103 *b.i.d.*
If-channel blocker		
Ivabradine	5 *b.i.d.*	7.5 *b.i.d.*

Evidence-based doses of disease-modifying drugs in key randomized trials in HFrEF (or after MI)

Enapren, Converten, Vasoretic

Zestril

Triatec

Cardicor, Concor, Congescor

Dilatrend

Lopresor, Seloken

Lobivon

Blopress

Tareg

Inspra

Aldactone

[d]A maximum dose of 50 mg twice daily can be administered to patients weighing over 85 kg.

Doses of diuretics commonly used in patients with HF

Diuretics	Initial dose (mg)		Usual daily dose (mg)	
Loop diuretics[a]				
Furosemide	20–40		40–240	
Torasemide	5–10		10–20	
Thiazides[b]				
Hydrochlorothiazide	25		12.5–100	
Metolazone	2.5		2.5–10	
Indapamide[c]	2.5		2.5–5	
Potassium-sparing diuretics[d]				
	+ACE-I/ ARB	-ACE-I/ ARB	+ACE-I/ ARB	-ACE-I/ ARB
Spironolactone/ eplerenone	12.5–25	50	50	100–200
Amiloride	2.5	5	5–10	10–20
Triamterene	25	50	100	200

Diuresix

Zaroxolyn

Natrilix

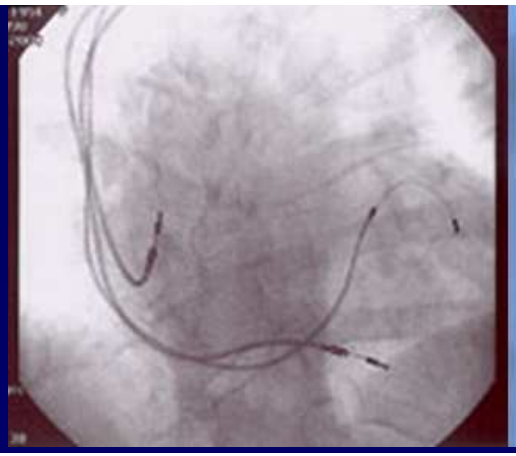
[a]Oral or intravenous; dose might need to be adjusted according to volume status/ weight; excessive doses may cause renal impairment and ototoxicity.
[b]Do not use thiazides if estimated glomerular filtration rate <30 mL/min/1.73 m^2, except when prescribed synergistically with loop diuretics.
[c]Indapamide is a non-thiazide sulfonamide.
[d]A mineralocorticoid antagonist (MRA) i.e. spironolactone/eplerenone is always preferred. Amiloride and triamterene should not be combined with an MRA.

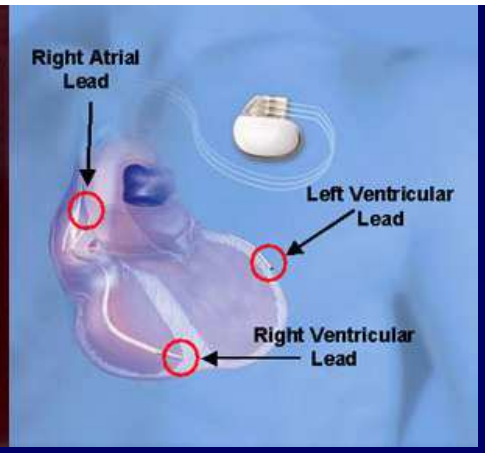
Implantable cardioverter-defibrillator

Secondary prevention An ICD is recommended to reduce the risk of sudden death and all-cause mortality in patients who have recovered from a ventricular arrhythmia causing haemodynamic instability, and who are expected to survive for >1 year with good functional status.	I	A
Primary prevention An ICD is recommended to reduce the risk of sudden death and all-cause mortality in patients with symptomatic HF (NYHA Class II–III), and an LVEF ≤35% despite ≥3 months of OMT, provided they are expected to survive substantially longer than one year with good functional status, and they have:		
• IHD (unless they have had an MI in the prior 40 days – see below).	I	A
• DCM.	I	B
ICD implantation is not recommended within 40 days of an MI as implantation at this time does not improve prognosis.	III	A
ICD therapy is not recommended in patients in NYHA Class IV with severe symptoms refractory to pharmacological therapy unless they are candidates for CRT, a ventricular assist device, or cardiac transplantation.	III	C

Cardiac resynchronization therapy

CRT is recommended for symptomatic patients with HF in sinus rhythm with a QRS duration ≥150 msec and LBBB QRS morphology and with LVEF ≤35% despite OMT in order to improve symptoms and reduce morbidity and mortality.	I	A
CRT should be considered for symptomatic patients with HF in sinus rhythm with a QRS duration ≥150 msec and non-LBBB QRS morphology and with LVEF ≤35% despite OMT in order to improve symptoms and reduce morbidity and mortality.	IIa	B
CRT is recommended for symptomatic patients with HF in sinus rhythm with a QRS duration of 130–149 msec and LBBB QRS morphology and with LVEF ≤35% despite OMT in order to improve symptoms and reduce morbidity and mortality.	I	B
CRT may be considered for symptomatic patients with HF in sinus rhythm with a QRS duration of 130–149 msec and non-LBBB QRS morphology and with LVEF ≤35% despite OMT in order to improve symptoms and reduce morbidity and mortality.	IIb	B
CRT rather than RV pacing is recommended for patients with HFrEF regardless of NYHA class who have an indication for ventricular pacing and high degree AV block in order to reduce morbidity. This includes patients with AF	I	A
CRT should be considered for patients with LVEF ≤35% in NYHA Class III–IV despite OMT in order to improve symptoms and reduce morbidity and mortality, if they are in AF and have a QRS duration ≥130 msec provided a strategy to ensure bi-ventricular capture is in place or the patient is expected to return to sinus rhythm.	IIa	B
Patients with HFrEF who have received a conventional pacemaker or an ICD and subsequently develop worsening HF despite OMT and who have a high proportion of RV pacing may be considered for upgrade to CRT. This does not apply to patients with stable HF.	IIb	B
CRT is contra-indicated in patients with a QRS duration < 130 msec.	III	A





HFpEF: patient characteristics

Compared with HFrEF, patients with HFpEF are:

Older	More often women
More commonly have hypertension	More commonly have AF
A history of myocardial infarction is less common	Frequent finding of CKD, obesity, OSAS, COPD, anemia and iron deficiency

Systemic chronic inflammatory state

indotto dalle co-patologie che evolve in un'**alterazione endoteliale del μcircolo sistemico**

it is recommended to screen patients with HFpEF or HFmrEF for both cardiovascular and non-cardiovascular comorbidities, which, if present, should be treated provided safe and effective interventions exist to improve symptoms, well-being and/or prognosis.	I	C
Diuretics are recommended in congested patients with HFpEF or HFmrEF in order to alleviate symptoms and signs.	I	B

GOALS

- Relief of volume overload
- Reduce symptoms
- Treatment of coexisting conditions
- Increase exercise tolerance

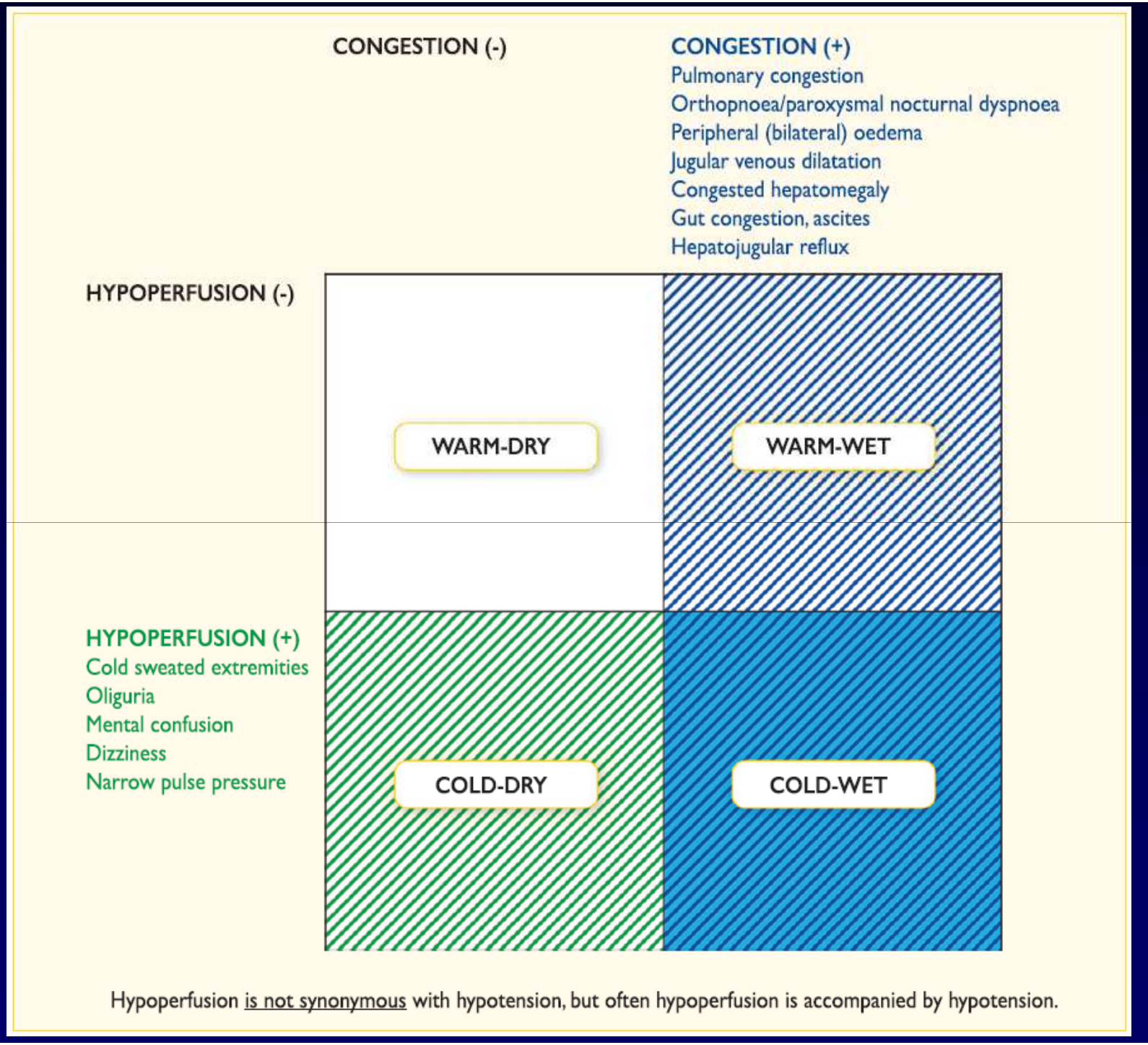
No treatment has yet been shown, convincingly, to reduce morbidity or mortality in patients with HFpEF or HFmrEF. However, since these patients are often elderly and highly symptomatic, and often have a poor quality of life, an important aim of therapy may be to alleviate symptoms and improve well-being.

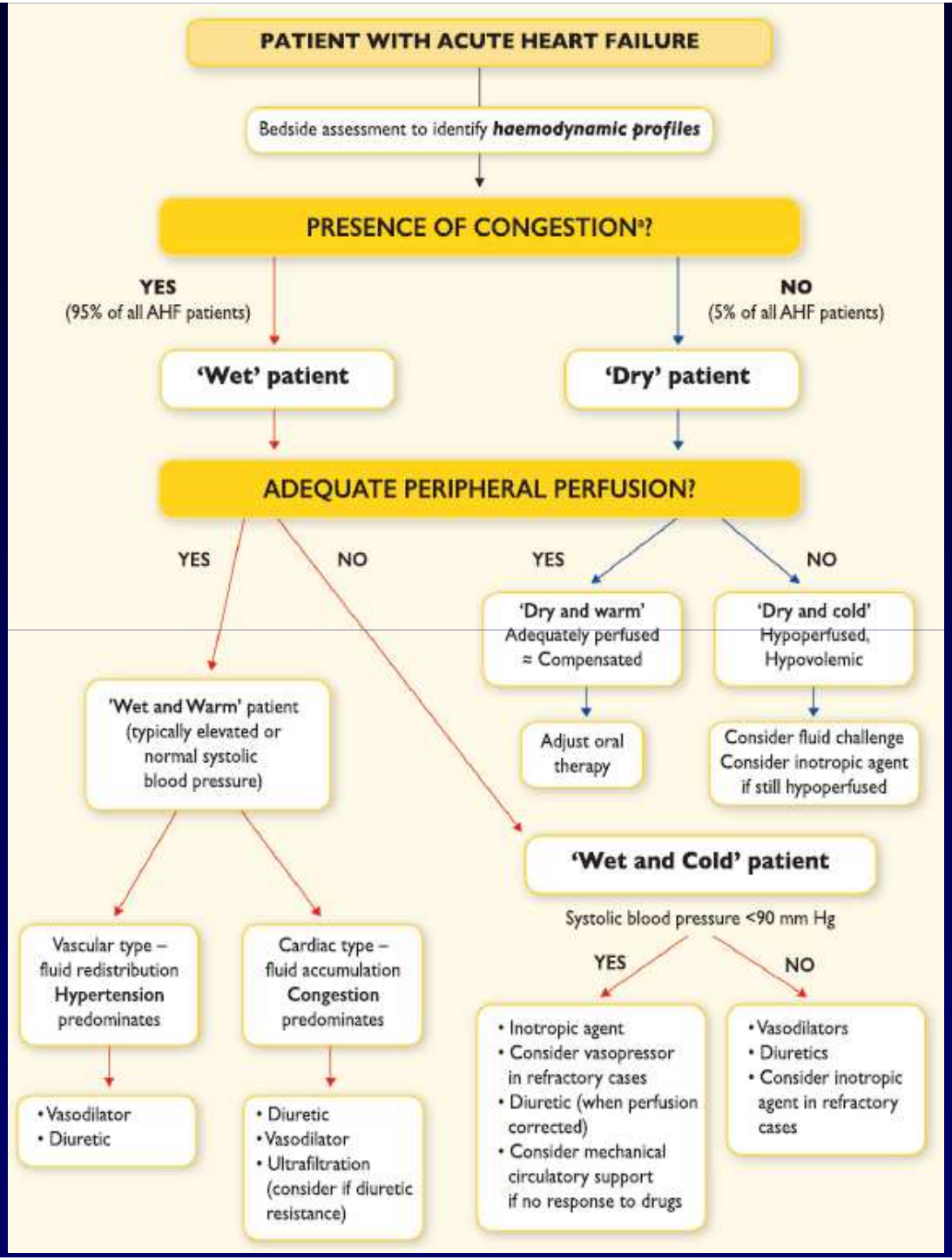
L'aumentata **rigidità** del **miocardio** e della **vascolatura** induce i pz ad ex molto sensibili alle **variazioni di carico** e dello **stato volemico**.

Incremento della volemia e del post carico può indurre quadri di

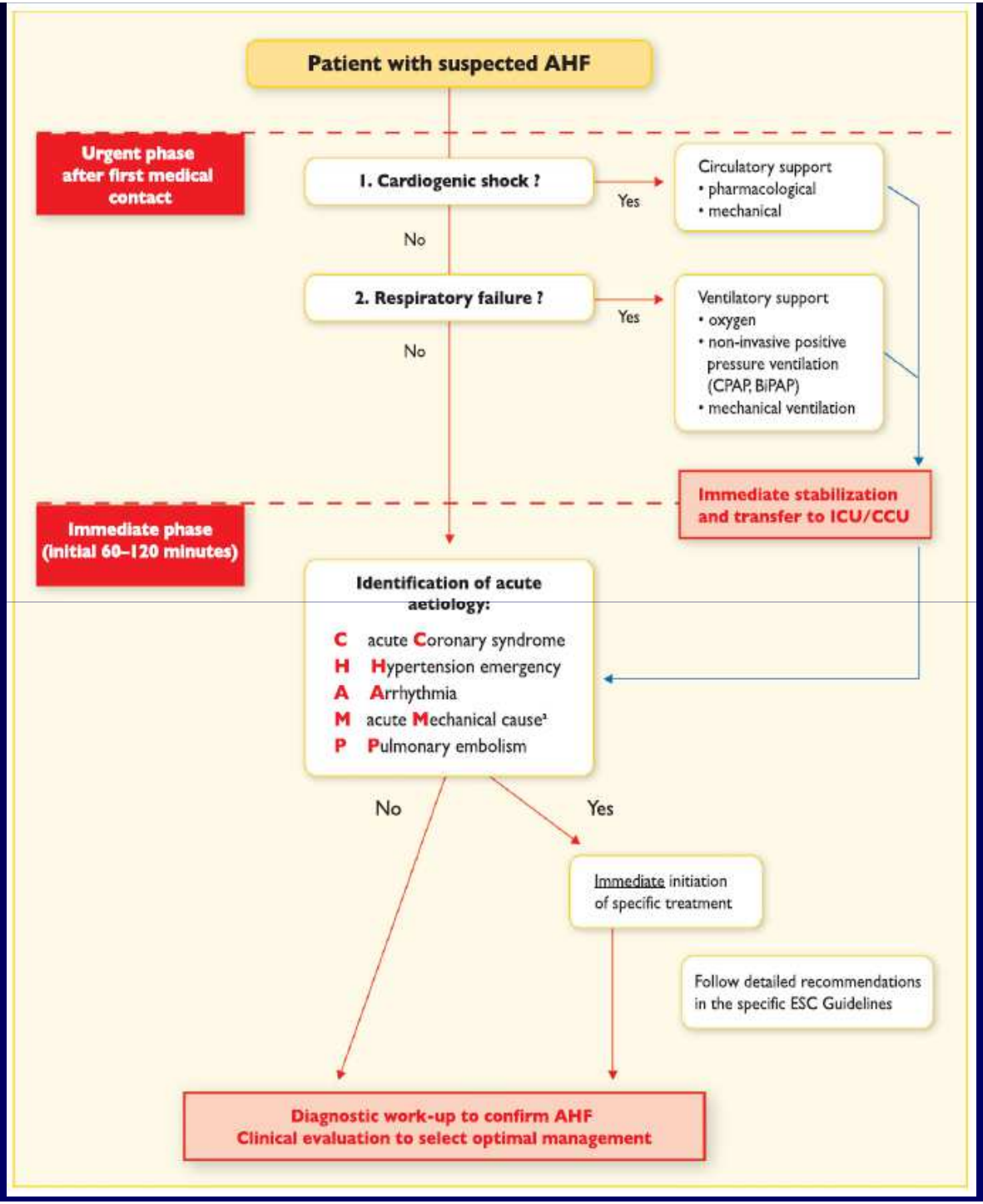
Acute heart failure

Acute coronary syndrome.
Tachyarrhythmia (e.g. atrial fibrillation, ventricular tachycardia).
Excessive rise in blood pressure.
Infection (e.g. pneumonia, infective endocarditis, sepsis).
Non-adherence with salt/fluid intake or medications.
Bradyarrhythmia.
Toxic substances (alcohol, recreational drugs).
Drugs (e.g. NSAIDs, corticosteroids, negative inotropic substances, cardiotoxic chemotherapeutics).
Exacerbation of chronic obstructive pulmonary disease.
Pulmonary embolism.
Surgery and perioperative complications.
Increased sympathetic drive, stress-related cardiomyopathy.
Metabolic/hormonal derangements (e.g. thyroid dysfunction, diabetic ketosis, adrenal dysfunction, pregnancy and peripartum related abnormalities).
Cerebrovascular insult.
Acute mechanical cause: myocardial rupture complicating ACS (free wall rupture, ventricular septal defect, acute mitral regurgitation), chest trauma or cardiac intervention, acute native or prosthetic valve incompetence secondary to endocarditis, aortic dissection or thrombosis.


CONGESTION (-)
CONGESTION (+)
Pulmonary congestion
Orthopnoea/paroxysmal nocturnal dyspnoea
Peripheral (bilateral) oedema
Jugular venous dilatation
Congested hepatomegaly
Gut congestion, ascites
Hepatojugular reflux
HYPOPERFUSION (-)
WARM-DRY
WARM-WET
HYPOPERFUSION (+)
Cold sweated extremities
Oliguria
Mental confusion
Dizziness
Narrow pulse pressure
COLD-DRY
COLD-WET
Hypoperfusion is not synonymous with hypotension, but often hypoperfusion is accompanied by hypotension.


PATIENT WITH ACUTE HEART FAILURE
Bedside assessment to identify haemodynamic profiles
PRESENCE OF CONGESTION?
YES
(95% of all AHF patients)
NO
(5% of all AHF patients)
'Wet' patient
'Dry' patient
ADEQUATE PERIPHERAL PERFUSION?
YES
NO
YES
NO
'Dry and warm'
Adequately perfused
≈ Compensated
'Dry and cold'
Hypoperfused,
Hypovolemic
Adjust oral therapy
Consider fluid challenge
Consider inotropic agent if still hypoperfused
'Wet and Warm' patient (typically elevated or normal systolic blood pressure)
'Wet and Cold' patient
Systolic blood pressure <90 mm Hg
YES
NO
Vascular type – fluid redistribution
Hypertension predominates
Cardiac type – fluid accumulation
Congestion predominates
• Vasodilator
• Diuretic
• Diuretic
• Vasodilator
• Ultrafiltration (consider if diuretic resistance)
• Inotropic agent
• Consider vasopressor in refractory cases
• Diuretic (when perfusion corrected)
• Consider mechanical circulatory support if no response to drugs
• Vasodilators
• Diuretics
• Consider inotropic agent in refractory cases

Term	Definition
Symptoms/signs of congestion (left-sided)	Orthopnoea, paroxysmal nocturnal dyspnoea, pulmonary rales (bilateral), peripheral oedema (bilateral).
Symptoms/signs of congestion (right-sided)	Jugular venous dilatation, peripheral oedema (bilateral), congested hepatomegaly, hepatojugular reflux, ascites, symptoms of gut congestion.
Symptoms/signs of hypoperfusion	Clinical: cold sweated extremities, oliguria, mental confusion, dizziness, narrow pulse pressure. Laboratory measures: metabolic acidosis, elevated serum lactate, elevated serum creatinine. Hypoperfusion is not synonymous with hypotension, but often hypoperfusion is accompanied by hypotension.
Hypotension	Systolic BP <90 mmHg
Bradycardia	Heart rate <40 bpm
Tachycardia	Heart rate >120 bpm
Abnormal respiratory effort	Respiratory rate >25 breaths/min with use of accessory muscles for breathing, or respiratory rate <8 breaths/min despite dyspnoea.
Low O_2 saturation	O_2 saturation (SaO_2) <90% in pulse oximetry Normal SaO_2 neither excludes hypoxaemia (low PaO_2) nor tissue hypoxia.
Hypoxaemia	O_2 partial pressure (PaO_2) in arterial blood <80 mmHg
Hypoxaemic respiratory failure (type I)	PaO_2 <60 mmHg (<8 kPa)
Hypercapnia	CO_2 partial pressure ($PaCO_2$) in arterial blood >45 mmHg
Hypercapnic respiratory failure (type II)	$PaCO_2$ >50 mmHg
Acidosis	pH <7.35
Elevated blood lactate	>2 mmol/L
Oliguria	Urine output <0.5 mL/kg/h


Patient with suspected AHF
Urgent phase after first medical contact
1. Cardiogenic shock ?
Yes
Circulatory support
• pharmacological
• mechanical
No
2. Respiratory failure ?
Yes
Ventilatory support
• oxygen
• non-invasive positive pressure ventilation (CPAP, BiPAP)
• mechanical ventilation
No
Immediate stabilization and transfer to ICU/CCU
Immediate phase (initial 60–120 minutes)
Identification of acute aetiology:
C acute Coronary syndrome
H Hypertension emergency
A Arrhythmia
M acute Mechanical cause²
P Pulmonary embolism
No
Yes
Immediate initiation of specific treatment
Follow detailed recommendations in the specific ESC Guidelines
Diagnostic work-up to confirm AHF
Clinical evaluation to select optimal management

Upon presentation a measurement of plasma natriuretic peptide level (BNP, NT-proBNP or MR-proANP) is recommended in all patients with acute dyspnoea and suspected AHF to help in the differentiation of AHF from non-cardiac causes of acute dyspnoea.	I	A
At admission in all patients presenting with suspected AHF, the following diagnostic tests are recommended:		
a. 12-lead ECG;	I	C
b. chest X-ray to assess signs of pulmonary congestion and detect other cardiac or non-cardiac diseases that may cause or contribute to the patient's symptoms;	I	C
c. the following laboratory assessments in the blood: cardiac troponins, BUN (or urea), creatinine, electrolytes (sodium, potassium), glucose, complete blood count, liver function tests and TSH.	I	C
Echocardiography is recommended immediately in haemodynamically unstable AHF patients and within 48 hours when cardiac structure and function are either not known or may have changed since previous studies.	I	C

Monitoring of transcutaneous arterial oxygen saturation (SpO_2) is recommended.	I	C
Measurement of blood pH and carbon dioxide tension (possibly including lactate) should be considered, especially in patients with acute pulmonary oedema or previous history of COPD using venous blood. In patients with cardiogenic shock arterial blood is preferable.	IIa	C
Oxygen therapy is recommended in patients with AHF and SpO_2 <90% or PaO_2 <60 mmHg to correct hypoxaemia.	I	C
Non-invasive positive pressure ventilation (CPAP, BiPAP) should be considered in patients with respiratory distress (respiratory rate >25 breaths/min, SpO_2 <90%) and started as soon as possible in order to decrease respiratory distress and reduce the rate of mechanical endotracheal intubation. Non-invasive positive pressure ventilation can reduce blood pressure and should be used with caution in hypotensive patients. Blood pressure should be monitored regularly when this treatment is used.	IIa	B
Intubation is recommended, if respiratory failure, leading to hypoxaemia (PaO_2 <60 mmHg), hypercapnia ($PaCO_2$ >50 mmHg) and acidosis (pH <7.35), cannot be managed non-invasively.	I	C

In AHF, oxygen should not be used routinely in non-hypoxaemic patients, as it causes vasoconstriction and a reduction in cardiac output. In COPD, hyperoxygenation may increase ventilation–perfusion mismatch, suppressing ventilation and leading to hypercapnia. The fraction of inspired oxygen (FiO_2) should be increased up to 100% if necessary, according to SpO_2, unless contraindicated. Hyperoxia, however, should be avoided.

Diuretics		
Intravenous loop diuretics are recommended for all patients with AHF admitted with signs/symptoms of fluid overload to improve symptoms. It is recommended to regularly monitor symptoms, urine output, renal function and electrolytes during use of i.v. diuretics.	I	C
In patients with new-onset AHF or those with chronic, decompensated HF not receiving oral diuretics the initial recommended dose should be 20–40 mg i.v. furosemide (or equivalent); for those on chronic diuretic therapy, initial i.v. dose should be at least equivalent to oral dose.	I	B
It is recommended to give diuretics either as intermittent boluses or as a continuous infusion, and the dose and duration should be adjusted according to patients' symptoms and clinical status.	I	B
Combination of loop diuretic with either thiazide-type diuretic or spironolactone may be considered in patients with resistant oedema or insufficient symptomatic response.	IIb	C

Vasodilators		
i.v. vasodilators should be considered for symptomatic relief in AHF with SBP >90 mmHg (and without symptomatic hypotension). Symptoms and blood pressure should be monitored frequently during administration of i.v. vasodilators.	IIa	B
In patients with hypertensive AHF, i.v. vasodilators should be considered as initial therapy to improve symptoms and reduce congestion.	IIa	B

Vasodilator	Dosing	Main side effects	Other
Nitroglycerine	Start with10–20 µg/min, increase up to 200 µg/min	Hypotension, headache	Tolerance on continuous use
Isosorbide dinitrate	Start with 1 mg/h, increase up to 10 mg/h	Hypotension, headache	Tolerance on continuous use
Nitroprusside	Start with 0.3 µg/kg/min and increase up to 5 µg/kg/min	Hypotension, isocyanate toxicity	Light sensitive

They have dual benefit by decreasing venous tone (to optimize preload) and arterial tone (decrease afterload). Consequently, they may also increase stroke volume.

Dosing should be carefully controlled to avoid excessive decreases in blood pressure, which is related to poor outcome. Vasodilators should be used with caution in patients with significant mitral or aortic stenosis.

Inotropic agents – dobutamine, dopamine, levosimendan, phosphodiesterase III (PDE III) inhibitors		
Short-term, i.v. infusion of inotropic agents may be considered in patients with hypotension (SBP <90 mmHg) and/or signs/symptoms of hypoperfusion despite adequate filling status, to increase cardiac output, increase blood pressure, improve peripheral perfusion and maintain end-organ function.	IIb	C
An intravenous infusion of levosimendan or a PDE III inhibitor may be considered to reverse the effect of beta-blockade if beta-blockade is thought to be contributing to hypotension with subsequent hypoperfusion.	IIb	C
Inotropic agents are not recommended unless the patient is symptomatically hypotensive or hypoperfused because of safety concern.	III	A

Use of an inotrope should be reserved for patients with a severe reduction in cardiac output resulting in compromised vital organ perfusion, which occurs most often in hypotensive AHF. Inotropic agents are not recommended in cases of hypotensive AHF where the underlying cause is hypovolaemia or other potentially correctable factors before elimination of these causes.

Inotropes, especially those with adrenergic mechanisms, can cause sinus tachycardia and may induce myocardial ischaemia and arrhythmias, thus ECG monitoring is required.

beta-blockers can be safely continued during AHF presentations except in cardiogenic shock. A recent meta-analysis demonstrated that discontinuation of beta-blockers in patients hospitalized with AHF was associated with significantly increased in-hospital mortality, short-term mortality and the combined endpoint of short-term rehospitalization or mortality.

Vasopressors		
A vasopressor (norepinephrine preferably) may be considered in patients who have cardiogenic shock, despite treatment with another inotrope, to increase blood pressure and vital organ perfusion.	IIb	B
It is recommended to monitor ECG and blood pressure when using inotropic agents and vasopressors, as they can cause arrhythmia, myocardial ischaemia, and in the case of levosimendan and PDE III inhibitors also hypotension.	I	C
In such cases intra-arterial blood pressure measurement may be considered.	IIb	C

Drugs with prominent peripheral arterial vasoconstrictor action such as norepinephrine or dopamine in higher doses (>5 μg/kg/min) are given to patients with marked hypotension. These agents are given to raise blood pressure and redistribute blood to the vital organs. However, this is at the expense of an increase in LV afterload.

Epinephrine (adrenaline) should be restricted to patients with persistent hypotension despite adequate cardiac filling pressures and the use of other vasoactive agents, as well as for resuscitation protocols.

Positive inotropes and/or vasopressors used to treat acute HF

Vasodilator	Bolus	Infusion rate
Dobutamine[a]	No	2–20 µg/kg/min (beta+)
Dopamine	No	3–5 µg/kg/min; inotropic (beta+)
		>5 µg/kg/min: (beta+), vasopressor (alpha+)
Milrinone[a,b]	25–75 µg/kg over 10–20 min	0.375–0.75 µg/kg/min
Enoximone[a]	0.5–1.0 mg/kg over 5–10 min	5–20 µg/kg/min
Levosimendan[a]	12 µg/kg over 10 min (optional) [c]	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

[a]Also a vasodilator.
[b]Not recommended in acutely worsened ischaemic heart failure.
[c]Bolus not recommended in hypotensive patients.

- Microgrammo (µg o mcg) o gamma (γ): millesima parte del milligrammo

  Esempi:

  400 mcg = 0,4 mg; 2 mg = 2000 mcg

Thrombo-embolism prophylaxis		
Thrombo-embolism prophylaxis (e.g. with LMWH) is recommended in patients not already anticoagulated and with no contra-indication to anticoagulation, to reduce the risk of deep venous thrombosis and pulmonary embolism.	I	B

Other drugs		
For acute control of the ventricular rate In patients with atrial fibrillation:		
a. digoxin and/or beta-blockers should be considered as the first-line therapy.[d]	IIa	C
b. amiodarone may be considered. [d]Beta-blockers should be used cautiously, if the patient is hypotensive.	IIb	B
Opiates may be considered for cautious use to relieve dyspnoea and anxiety in patients with severe dyspnoea but nausea and hypopnea may occur.	IIb	B

Digoxin

Digoxin is mostly indicated in patients with AF and rapid ventricular rate (>110 bpm) and given in boluses of 0.25–0.5 mg i.v. if not used previously (0.0625–0.125 mg may be an adequate dose in patients with moderate to severe renal dysfunction).

Goals of treatment during the different stages of management of acute heart failure

Immediate (ED/ICU/CCU)
Improve haemodynamics and organ perfusion.
Restore oxygenation.
Alleviate symptoms.
Limit cardiac and renal damage.
Prevent thrombo-embolism.
Minimize ICU length of stay.
Intermediate (in hospital)
Identify aetiology and relevant co-morbidities.
Titrate therapy to control symptoms and congestion and optimize blood pressure.
Initiate and up-titrate disease-modifying pharmacological therapy.
Consider device therapy in appropriate patients.
Pre-discharge and long-term management
Develop a careplan that provides: o A schedule for up-titration and monitoring of pharmacological therapy. o Need and timing for review for device therapy. o Who will see the patient for follow-up and when.
Enrol in disease management programme, educate, and initiate appropriate lifestyle adjustments.
Prevent early readmission.
Improve symptoms, quality of life, and survival.

Paziente di 88 anni sottoposta ad impianto di pace-maker Medtronic Sensia bicamerale per malattia del nodo del seno ed episodi sincopali.

Ipertensione arteriosa. Dall'impianto del PM non recidive sincopali, nell’ultimo mese riferisce graduale peggioramento della dispena per sforzi lievi ed a riposo con edemi declivi. Incremento ponderale di 6 kg.

All'ultimo controllo del PM programmato in VVIR per FA sottostante con frequenza 50/90

Terapia in atto: coumadin sec. INR, lasitone 1 cp a giorni alterni attualmente 1 cp/die cardicor 2,5 mg. 1cp. x 2, cardura 2 mg, halcion.

Agli esami ematici del 2017 Hb 12,5 g/dl eGFR 40 K sierico 4,7 mmol/l

ECG: fibrillazione atriale a media frequenza cardiaca con sovraccarico ventricolare sinistro.

Esame Obiettivo: toni validi aritmici soffio mitralico 3/6 edemi declivi crepitazioni bibasali e turgore giugulare. PA110/65

Ecocardiografia: ventricolo sinistro di normali dimensioni con cinesi globale buona, ipertrofia ventricolare sinistro di grado moderato. IM moderata severa cava inferiore di 26 mm.

CONCLUSIONI:

Iniziali segni di scompenso cardiaco. Consiglierei di sospendere cardura 2 mg. ½ cp, ridurre cardicor 2,5 mg. ½ cp. X 2 continuare con coumadin sec. INR. Per quanto riguarda la terapia diuretica consiglierei lasix 25 1 cp. ore 8 e lasitone 1 cp. ore 16 da integrare con zaroxolin 5 mg. 1/2 cp. ore 8 il lunedì ed il giovedì. Da risentire tra 7 giorni cell 3493541890 email st.simonini@ausl.mo.it. Controllerei esami ematici tra 7-10 giorni.

16/3 Le invio un breve resoconto del decorso della terapia .per quanto riguarda il calo di peso è stato il seguente 13/3 59kg ,14/3 57,59kg 15/3 56,4 kg 16/3 55,6kg mi pare quindi che la terapia stia funzionando .la pressione si mantiene tra 105/110 e la mamma non mostra segni di indebolimento. La ringrazio fin da ora in attesa di sapere se continuare la terapia come programmato.

19/3 Le invio il resoconto del calo ponderale :17/3 54,100 18/3 53,400 19/3 53,300 cosa devo fare con il zaroxolin che dovrebbevassumere domani? Per quanto riguarda gli esami del sangue da lei richiesti quando ritiene sia meglio farli?attendo sua risposta grazie

24/3 Questa mattina abbiamo fatto gli esami del sangue che saranno pronti martedì! Se fosse possibile per lei vedere la mamma prima di Pasqua sarebbe perfetto!per quanto riguarda il peso siamo sui 52 kg i gonfiori mi sembrano completamente spariti così anche l affanno. La pressione è un Po bassa oggi 97/65 penso di non darle la compressa per la pressione questa sera. Aspetto sue notizie grazie

DANNO ANATOMICO (1)

DANNO MIOCARDICO PRIMITIVO:

- **CARDIOMIOPATIA DILATATIVA**
- **CARDIOMIOPATIA IPERTROFICA**
- **CARDIOMIOPATIA RESTRITTIVA**
- **CARDIOMIOPATIA ARITMOGENA DEL VENTRICOLO DX**

DANNO ANATOMICO (2)

- DANNO ANATOMICO SECONDARIO:
  - DA SOVRACCARICO DI VOLUME: IPERTROFIA ECCENTRICA
  - DA SOVRACCARICO DI PRESSIONE: IPERTROFIA CONCENTRICA
  - DA ISCHEMIA, CAUSE METABOLICHE E NUTRIZIONALI, MALATTIE SISTEMICHE

DA INSUFFICIENZA FUNZIONALE

- ALTERAZIONI DEL RITMO
- ALCUNE CAUSE DI INSUFFICIENZA DIASTOLICA (PERICARDITE, STENOSI MITRALICA)

LE PATOLOGIE SOPRA INDICATE POSSONO ESSERE CAUSA DI:

- **INSUFFICIENZA DIASTOLICA**
- **INSUFFICIENZA SISTOLICA**

DISFUNZIONE SISTOLICA

- **FRAZIONE DI EIEZIONE DIMINUITA**

DISFUNZIONE DIASTOLICA

- FRAZIONE DI EIEZIONE NORMALE
- CAUSE:
  - **SOVRACCARICO DI PRESSIONE**
  - **MIOCARDIOPATIA IPERTROFICA**
  - **STENOSI MITRALICA O TRICUSPIDALICA**
  - **PERICARDITE COSTRITTIVA**
  - **MIOCARDIOPATIA RESTRITTIVA**
  - **SOVRACCARICHE ACUTI DI VOLUME**
  - **ISCHEMIA MIOCARDICA**

CLASSE I NYHA

- L'ATTIVITA' FISICA ABITUALE NON PROVOCA SINTOMI
- IL SOGGETTO:
  - **E' CARDIOPATICO CON SEGNI OBIETTIVI DI SCOMPENSO**
  - **E/O DIVENTA SINTOMATICO SE NON CURATO**
  - **E/O IN PASSATO E' STATO IN UNA CLASSE NYHA SUPERIORE**

CLASSE II NYHA

- L’ATTIVITA’ FISICA ABITUALE PROVOCA AFFATICAMENTO O DISPNEA

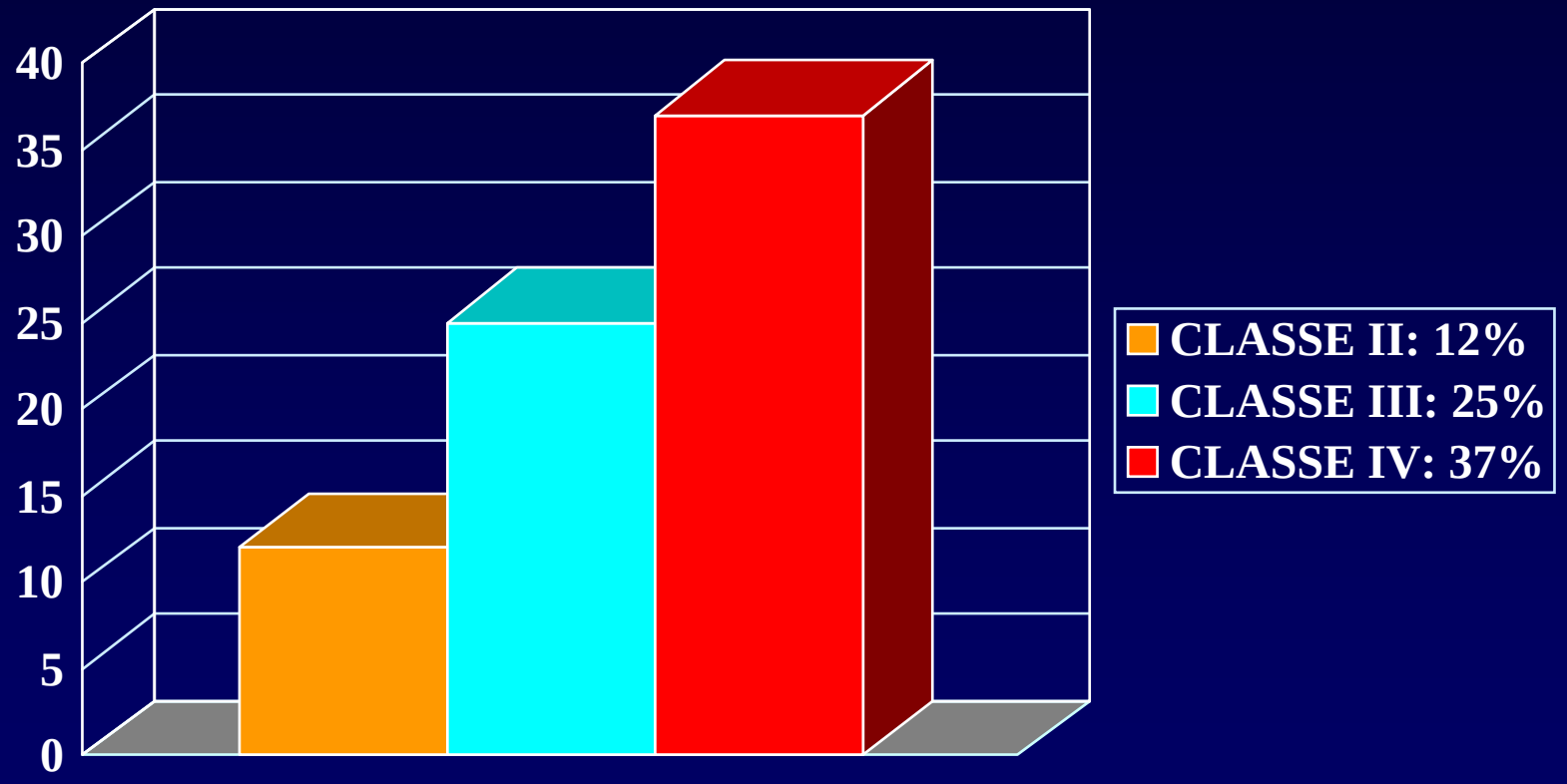
CLASSE III NYHA

- BENESSERE A RIPOSO, MA ATTIVITA' FISICHE INFERIORI ALLA NORMA PROVOCANO SINTOMI

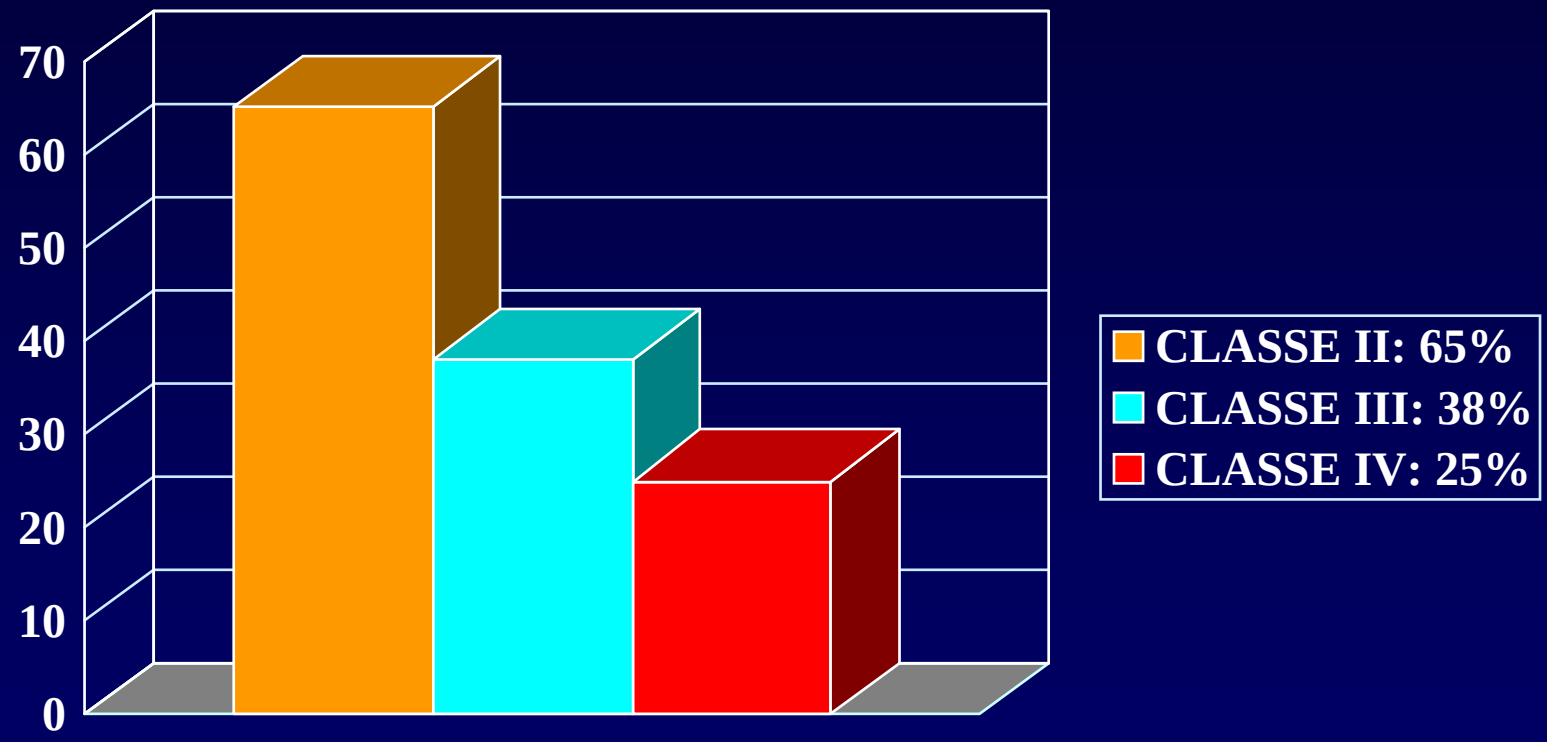
CLASSE IV NYHA

- SINTOMI A RIPOSO, CON AUMENTO DEI DISTURBI AD OGNI MINIMA ATTIVITA’

MORTALITA' AD UN ANNO: 15,5% (DATO COMPLESSIVO)



SOPRAVVIVENZA A 5 ANNI



SEGNI E SINTOMI NELLO SCOMPENSO CARDIACO

- **DA SOVRACCARICO DI VOLUME INTRAVASCOLARE E INTERSTIZIALE**
- **DA BASSA PORTATA**

SINTOMI E SEGNI DI CONGESTIONE

- DISPNEA (da sforzo, ortopnea, dispnea parossistica notturna, asma cardiaco, edema polmonare acuto)
- RUMORI DA STASI
- NICTURIA, OLIGURIA
- DISTURBI ADDOMINALI
- TOSSE
- EDEMI DECLIVI, TURGORE GIUGULARE
- FEGATO DA STASI, ASCITE
- VERSAMENTO PLEURICO

SINTOMI E SEGNI DI BASSA PORTATA

- STANCHEZZA
- CONFUSIONE MENTALE
- DIMAGRIMENTO (FINO ALLA CACHESSIA)
- PALLORE

IL PROBLEMA DEI SINTOMI

- IL 20% DEI PAZIENTI CON FE < 40% NON RIFERISCE SINTOMI
- SOLO IL 42% DEI PAZIENTI CON FE < 30% LAMENTA DISPNEA DA SFORZO

VALUTAZIONE DEI SINTOMI

- SIX MINUTES WALK TEST:
  - PER OBIETTIVARE VARIAZIONE DELLA PERFORMANCE FISICA
  - PER VALUTARE LA TERAPIA

IL PROBLEMA DEI SEGNI

- IL 44% DEI PAZIENTI CON SCOMPENSO MODERATO -SEVERO NON PRESENTA SEGNI CARATTERISTICI
- ALCUNI SEGNI SONO ASPECIFICI
- TERZO TONO, POLSO GIUGULARE: SONO VALUTATI IN MANIERA DIVERSA DA DIVERSI OSSERVATORI

SEGNI CLINICI (1)

- POLSO PICCOLO, ALTERNANTE
- TURGORE GIUGULARE
- RIFLUSSO ADDOMINO GIUGULARE
- TERZO TONO: PRESENTE NEL 68% DEI PAZIENTI CON FE < 30%

SEGNI CLINICI (2)

- RITENZIONE IDRICA:
  - **AUMENTO PONDERALE**
  - **EDEMI PERIFERICI**
  - **ASCITE, IDROTORACE**
  - **RANTOLI (PRESENTI NEL 37% DEI PAZIENTI CON FE < 30%)**
  - **EDEMA POLMONARE**
  - **EPATOMEGALIA CONGESTIZIA**

CONCLUSIONE

SEGNI E SINTOMI POSSONO SUGGERIRE L'ESISTENZA DI UN POSSIBILE SCOMPENSO, MA IL SOSPETTO CLINICO DEVE ESSERE SUFFRAGATO DA ULTERIORI DATI CLINICO STRUMENTALI (ECOCARDIO)

MECCANISMI DI COMPENSO EMODINAMICO

- **MODIFICAZIONI STRUTTURALI**
  - **IPERTROFIA CONCENTRICA**
  - **IPERTROFIA ECCENTRICA**
- **MODIFICAZIONI NEURORMONALI**

IPERTROFIA CONCENTRICA

- **CAUSATA DA SOVRACCARICHI DI PRESSIONE**
- **IPERPLASIA E IPERTROFIA DELLE FIBRE MIOCARDICHE**
- **AUMENTO DI SPESSORE DELLE PARETI**
- **CAVITA' VENTRICOLARI UGUALI O RIDOTTE**

IPERTROFIA ECCENTRICA

- CAUSATA DA SOVRACCARICHI DI VOLUME (es.: insufficienza mitralica o aortica, DIA, DIV)
- DILATAZIONE DELLE CAMERE VENTRICOLARI CON MODERATA IPERTROFIA
- IPOCINESIA DELLE PARETI
- F.E. DIMINUITA

MODIFICAZIONI NEURORMONALI

INTERVENGONO QUANDO

LE MODIFICAZIONI STRUTTURALI SONO INSUFFICIENTI

- **SISTEMA NERVOSO SIMPATICO**
- **FATTORE NATRIURETICO**
- **VASOPRESSINA**
- **ALTERAZIONE DEI FLUSSI REGIONALI**
- **SISTEMA RENINA ANGIOTENSINA ALDOSTERONE**

SISTEMA NERVOSO SIMPATICO

- **RILASCIO DI NORADRENALINA**
- **VASOCOSTRIZIONE , TACHICARDIA**
- **ATTIVAZIONE MAGGIORE NEI PAZIENTI A PROGNOSI SFAVOREVOLE**

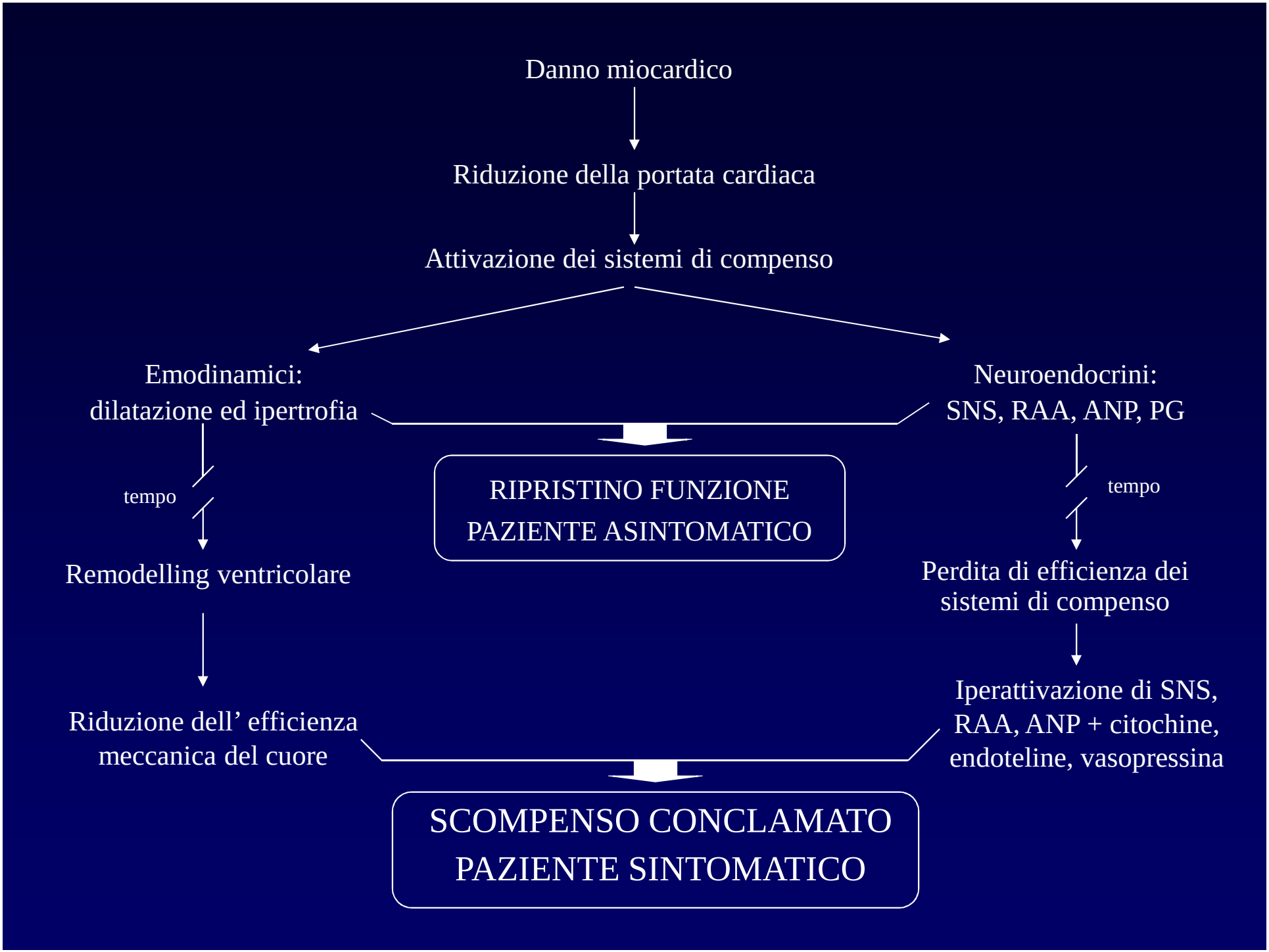
ALTERAZIONI DEI FLUSSI REGIONALI

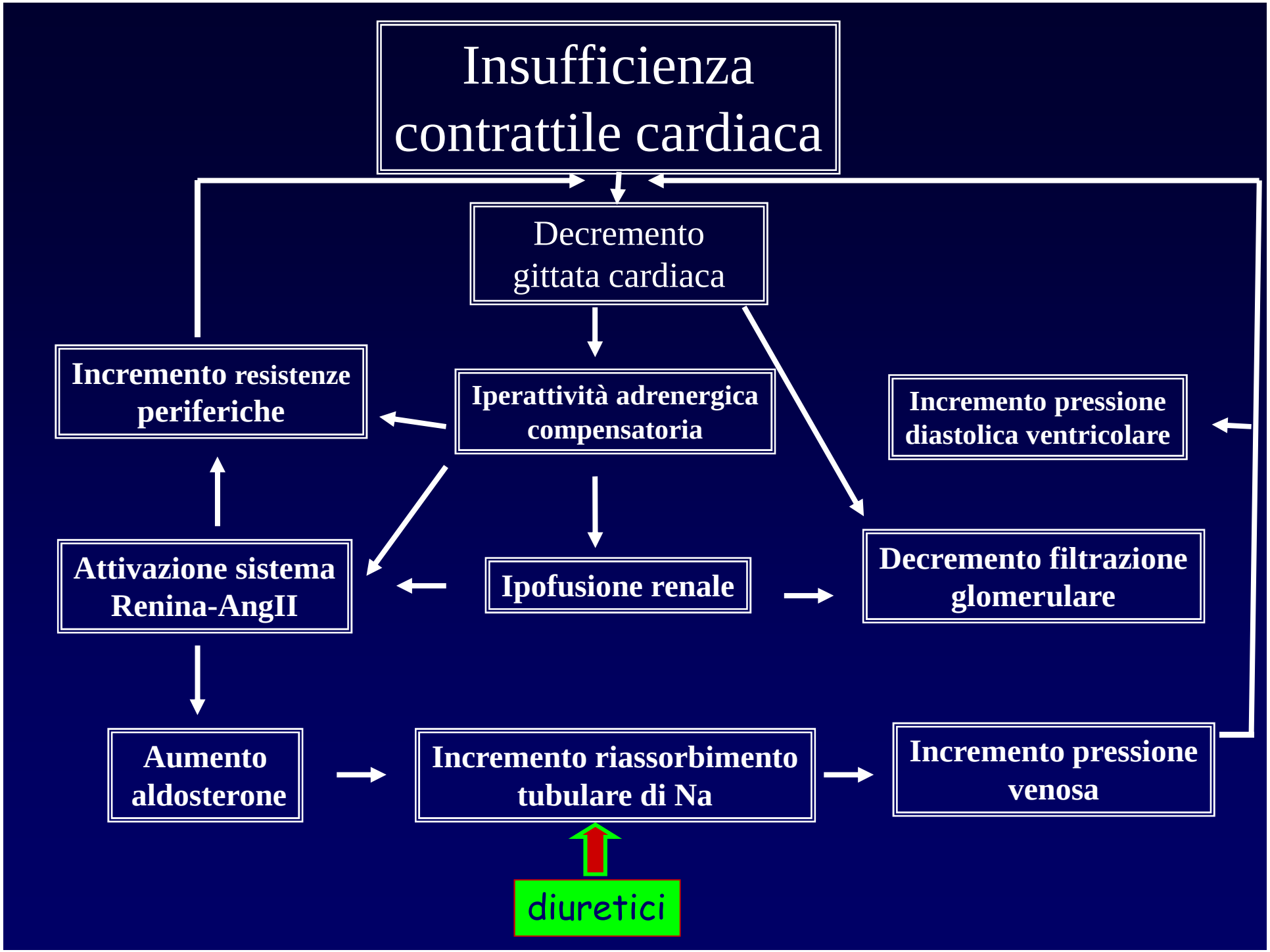
IN PRESENZA DI RIDOTTA PORTATA:

- I FLUSSI MIOCARDICO E CEREBRALE VENGONO MANTENUTI
- I FLUSSI RENALE, EPATOSPLANCNICO E DEGLI ARTI SI RIDUCONO

SISTEMA
RENINA ANGIOTENSINA ALDOSTERONE

- **ATTIVITA' ELEVATA NEGLI STADI INIZIALI E FINALI DELLO SCOMPENSO**
- **TIENE ELEVATA LA PRESSIONE**
- **SAREBBE ATTIVATO DALLA NORADRENALINA**


Danno miocardico
Riduzione della portata cardiaca
Attivazione dei sistemi di compenso
Emodinamici:
dilatazione ed ipertrofia
Neuroendocrini:
SNS, RAA, ANP, PG
tempo
tempo
RIPRISTINO FUNZIONE
PAZIENTE ASINTOMATICO
Remodelling ventricolare
Perdita di efficienza dei
sistemi di compenso
Riduzione dell' efficienza
meccanica del cuore
Iperattivazione di SNS,
RAA, ANP + citochine,
endoteline, vasopressina
SCOMPENSO CONCLAMATO
PAZIENTE SINTOMATICO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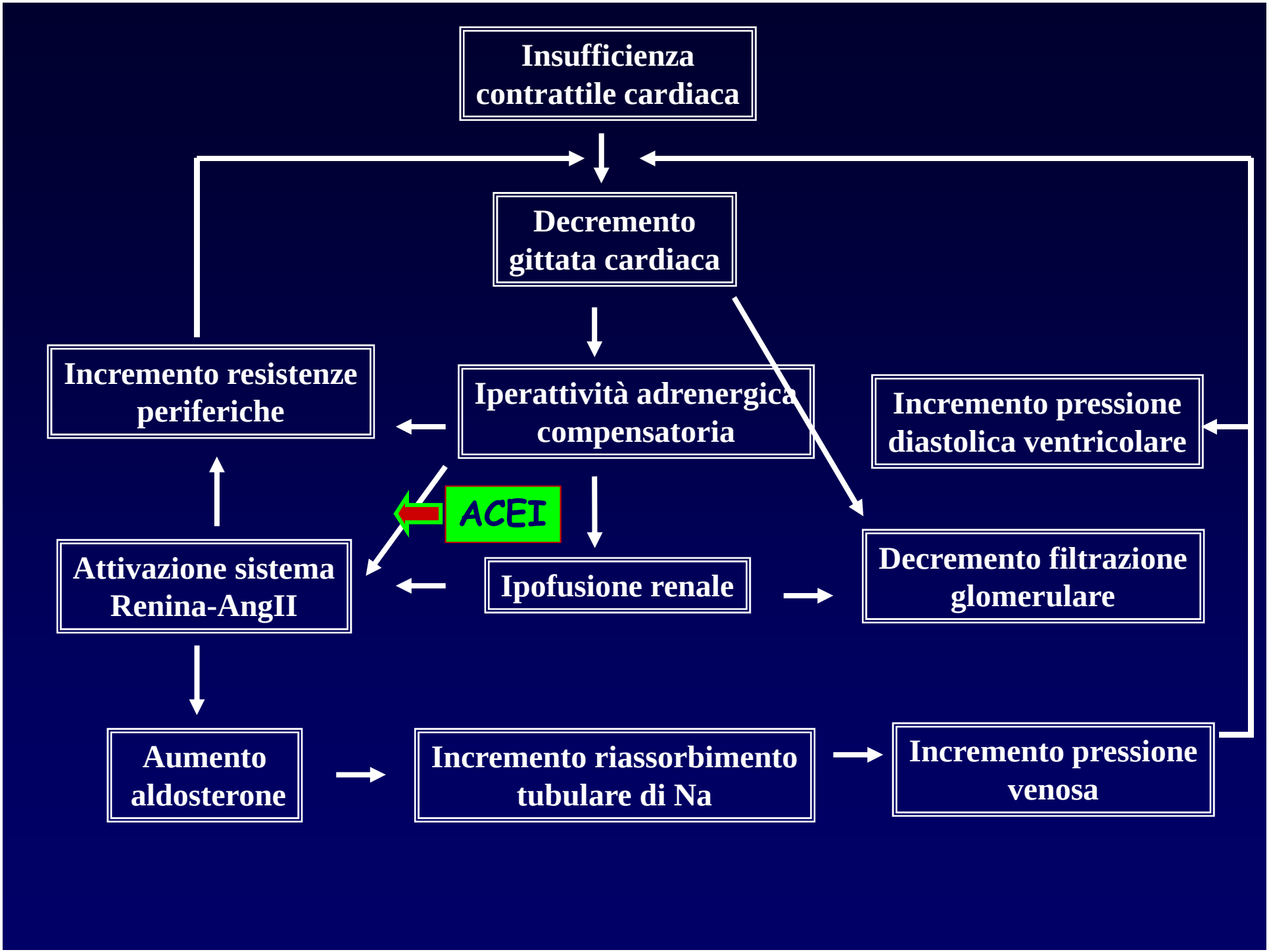
Decremento gittata cardiaca
Incremento resistenze periferiche
Iperattività adrenergica compensatoria
Incremento pressione diastolica ventricolare
Attivazione sistema Renina-AngII
Ipofusione renale
Decremento filtrazione glomerulare
Aumento aldosterone
Incremento riassorbimento tubulare di Na
Incremento pressione venosa
diuretici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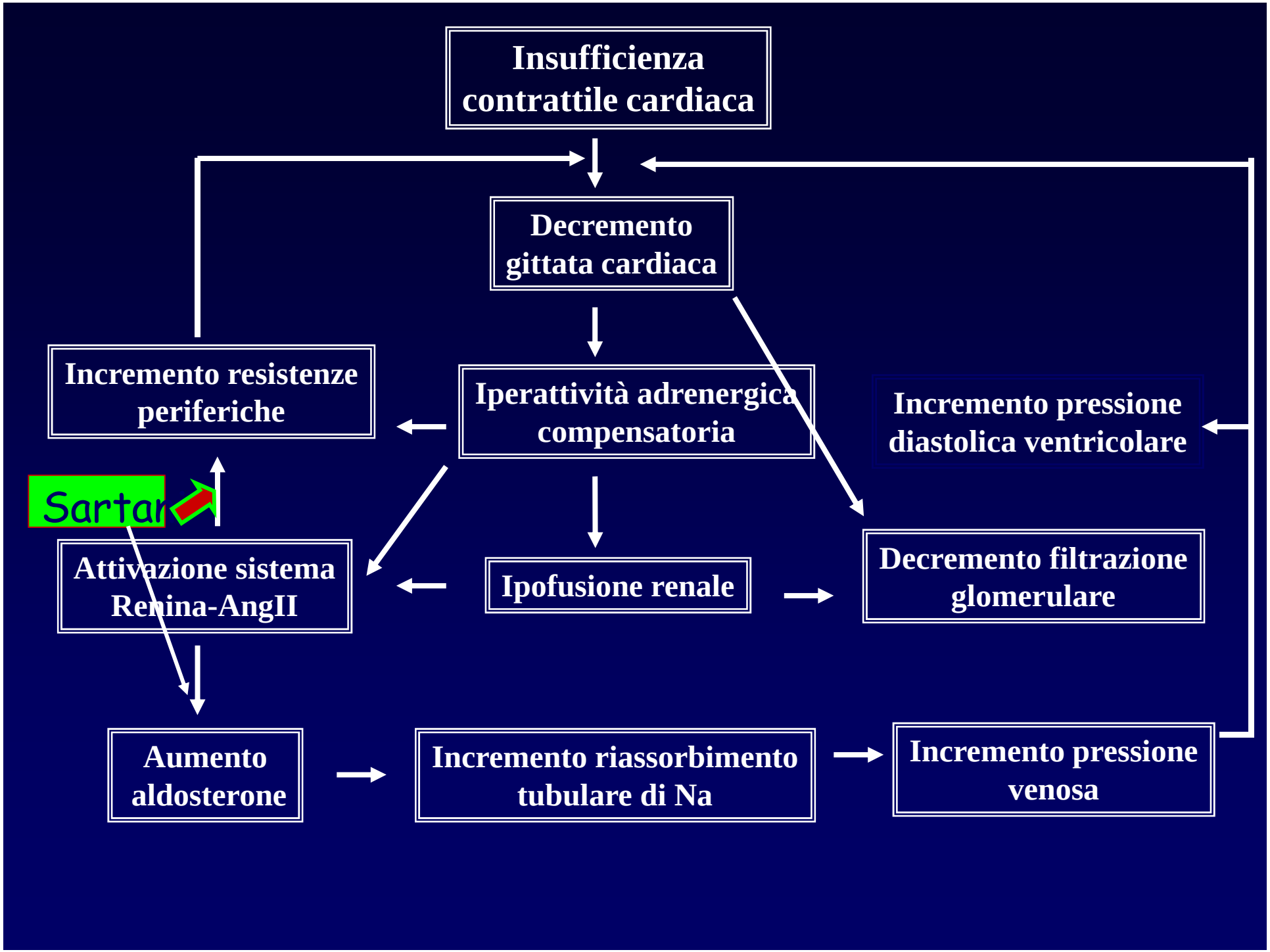
Decremento gittata cardiaca
Incremento resistenze periferiche
Iperattività adrenergica compensatoria
Incremento pressione diastolica ventricolare
ACEI
Attivazione sistema Renina-AngII
Ipofusione renale
Decremento filtrazione glomerulare
Aumento aldosterone
Incremento riassorbimento tubulare di Na
Incremento pressione venosa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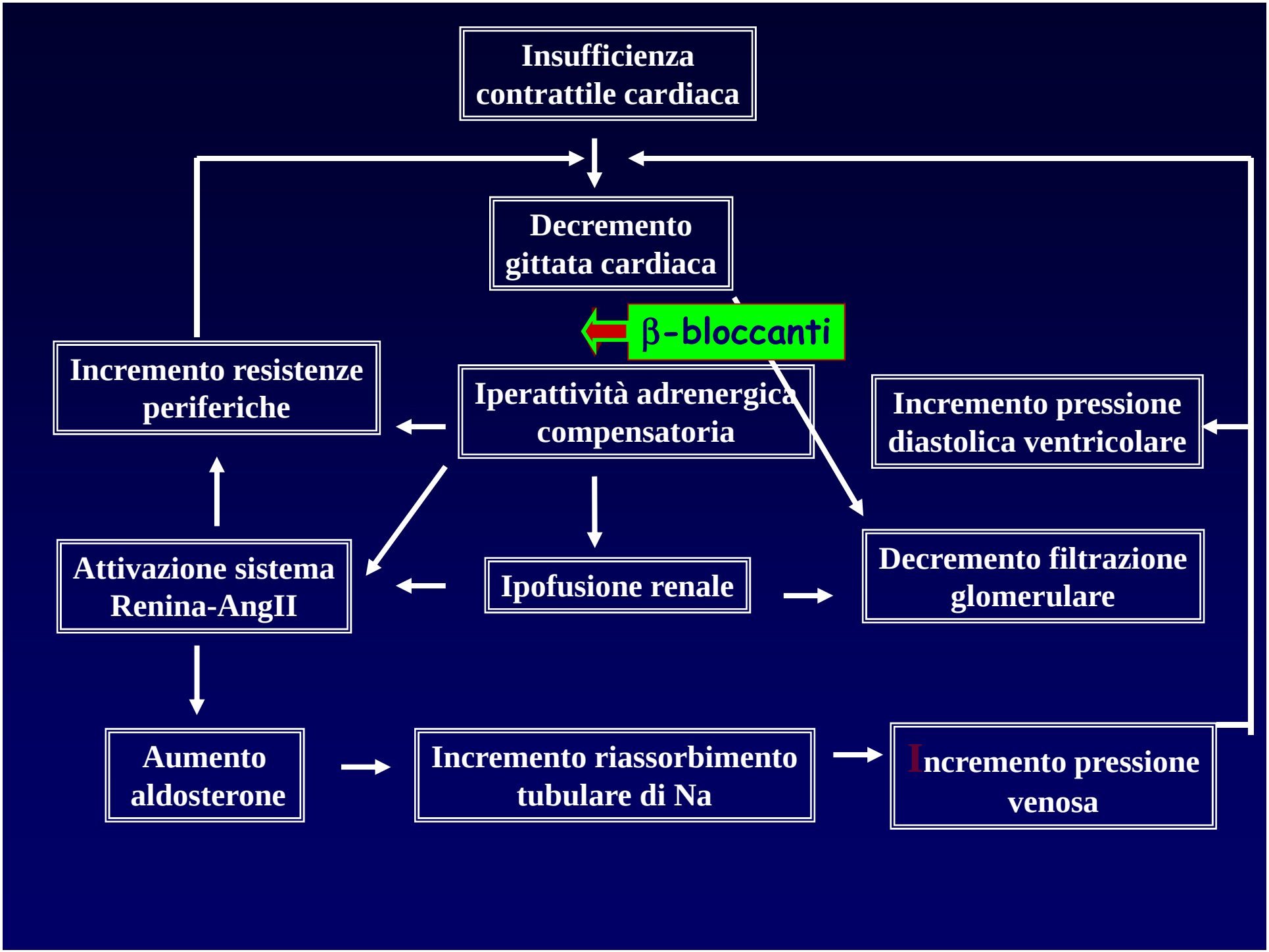
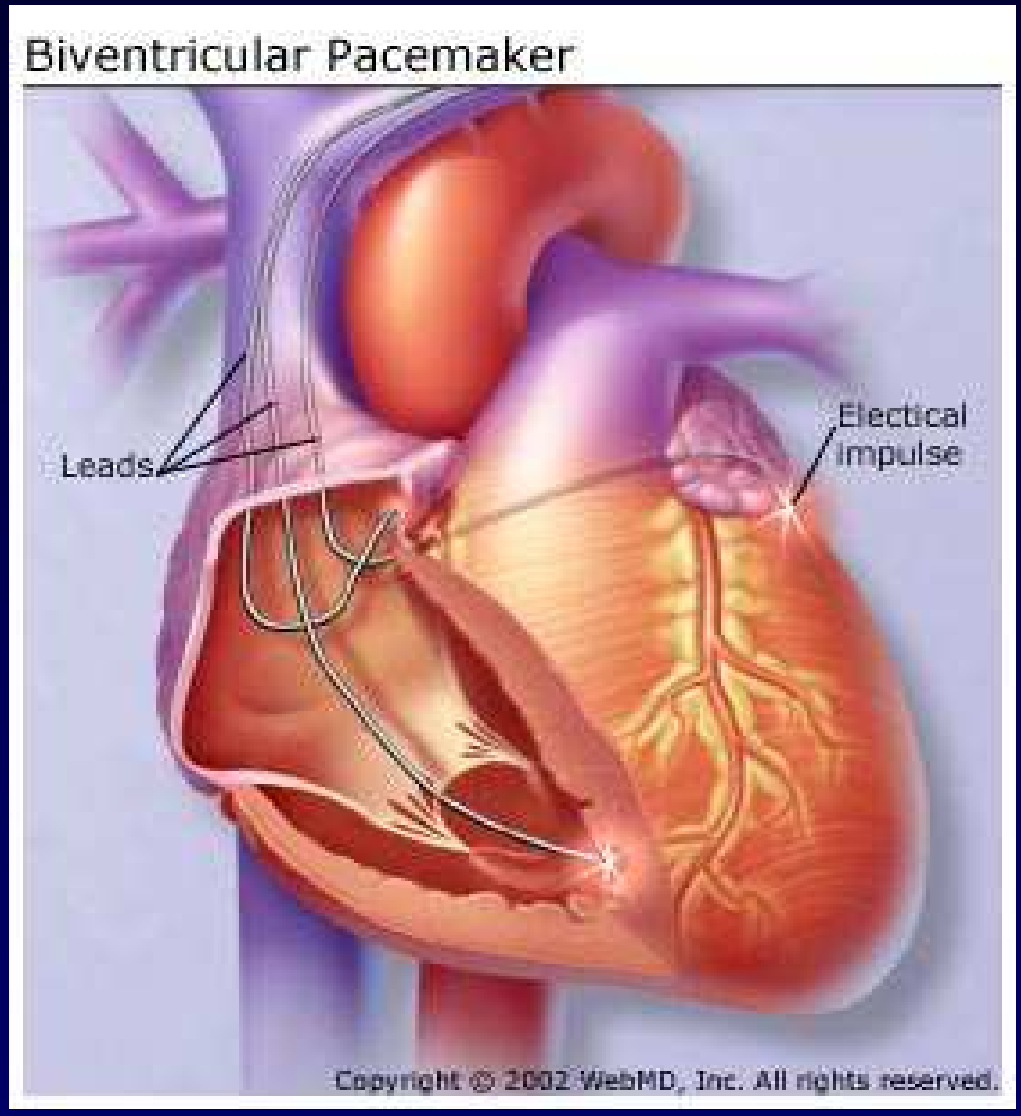
Decremento gittata cardiaca
Incremento resistenze periferiche
Iperattività adrenergica compensatoria
Incremento pressione diastolica ventricolare
Sartan
Attivazione sistema Renina-AngII
Ipofusione renale
Decremento filtrazione glomerulare
Aumento aldosterone
Incremento riassorbimento tubulare di Na
Incremento pressione venosa


Insufficienza contrattile cardiaca
Decremento gittata cardiaca
β-bloccanti
Incremento resistenze periferiche
Iperattività adrenergica compensatoria
Incremento pressione diastolica ventricolare
Attivazione sistema Renina-AngII
Ipofusione renale
Decremento filtrazione glomerulare
Aumento aldosterone
Incremento riassorbimento tubulare di Na
Incremento pressione venosa


Biventricular Pacemaker
Leads
Electical impulse
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FATTORE NATRIURETICO

- IL PEPTIDE NATRIURETICO PROATRIALE (N-ANP) E' UN BUON INDICATORE DI DISFUNZIONE VENTRICOLARE ASINTOMATICA E FATTORE PREDITTIVO DI MORTALITA'

- IL PEPTIDE NATRIURETICO CEREBRALE (BNP) SAREBBE UN FATTORE PREDITTIVO DI MORTALITA' IN PAZIENTI CON SCOMPENSO CRONICO

VASOPRESSINA

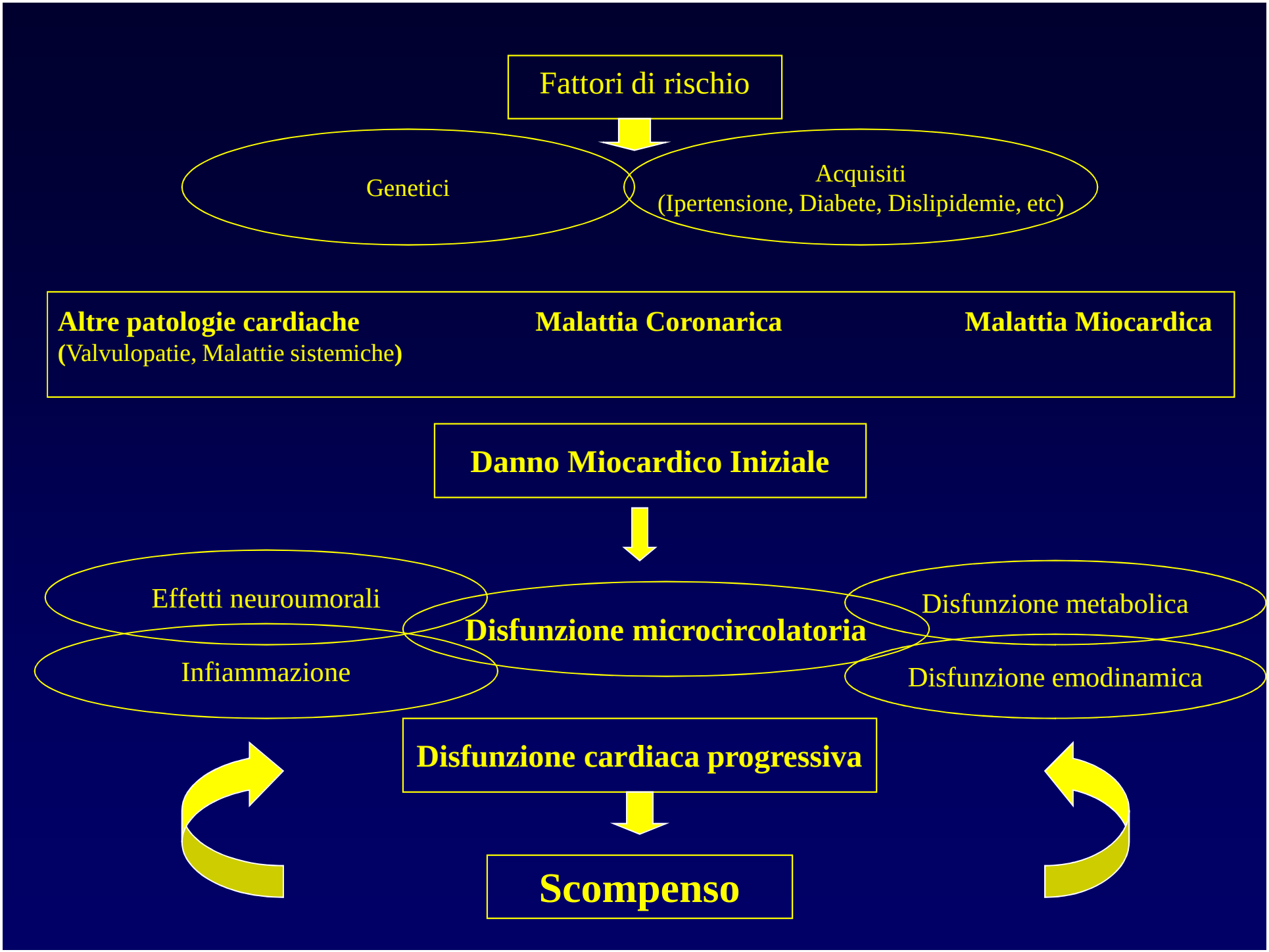
SAREBBE AUMENTATA IN CORSO DI SCOMPENSO

OBIETTIVI TERAPEUTICI NEL TRATTAMENTO CRONICO DELLO SCOMPENSO CON FARMACI CARDIOTONICI

1. **Miglioramento della funzione cardiaca sia a riposo che sotto sforzo**
2. **Miglioramento dell'efficienza cardiaca**
3. **Prevenzione delle complicanze in termini di danno agli organi periferici**
4. **Miglioramento della qualità della vita**

ma soprattutto…….

5. **Aumento della sopravvivenza!!!**


Fattori di rischio
Genetici
Acquisiti
(Ipertensione, Diabete, Dislipidemie, etc)
Altre patologie cardiache
(Valvulopatie, Malattie sistemiche)
Malattia Coronarica
Malattia Miocardica
Danno Miocardico Iniziale
Effetti neuroumorali
Infiammazione
Disfunzione microcircolatoria
Disfunzione metabolica
Disfunzione emodinamica
Disfunzione cardiaca progressiva
Scompenso

1980	1995
Cardiomiopatia dilatativa	**Cardiomiopatia dilatativa**
Cardiomiopatia ipertrofica	**Cardiomiopata ipertrofica**
Cardiomiopatia restrittiva	**Cardiomiopatia restrittiva**
	Cardiomiopatia aritmogena del ventr dx
Malattie muscolari cardiache specifiche	**Cardiomiopatie specifiche**
infettive	infiammatorie
metaboliche	metaboliche
in concomitanza di malattie sistemiche	in concomitanza di malattie sistemiche
eredofamiliari	distrofie muscolari
	disordini neuromuscolari
	ischemiche
	valvolari
	ipertensive
tossiche e da ipersensibilità	tossiche e da ipersensibilità
cardiomiopatia peripartum	cardiomiopatia peripartum
Non classificate	**Non classificate**
(Fibroelastosi, Miocardite di Fiedler)	(Fibroelastosi, miocardio non compattato, cardiomiopatia dilatativa con minima dilatazione, forme con coinvolgimento mitocondriale)

Patologie a possibile evoluzuione verso la disfunzioe ventricolare

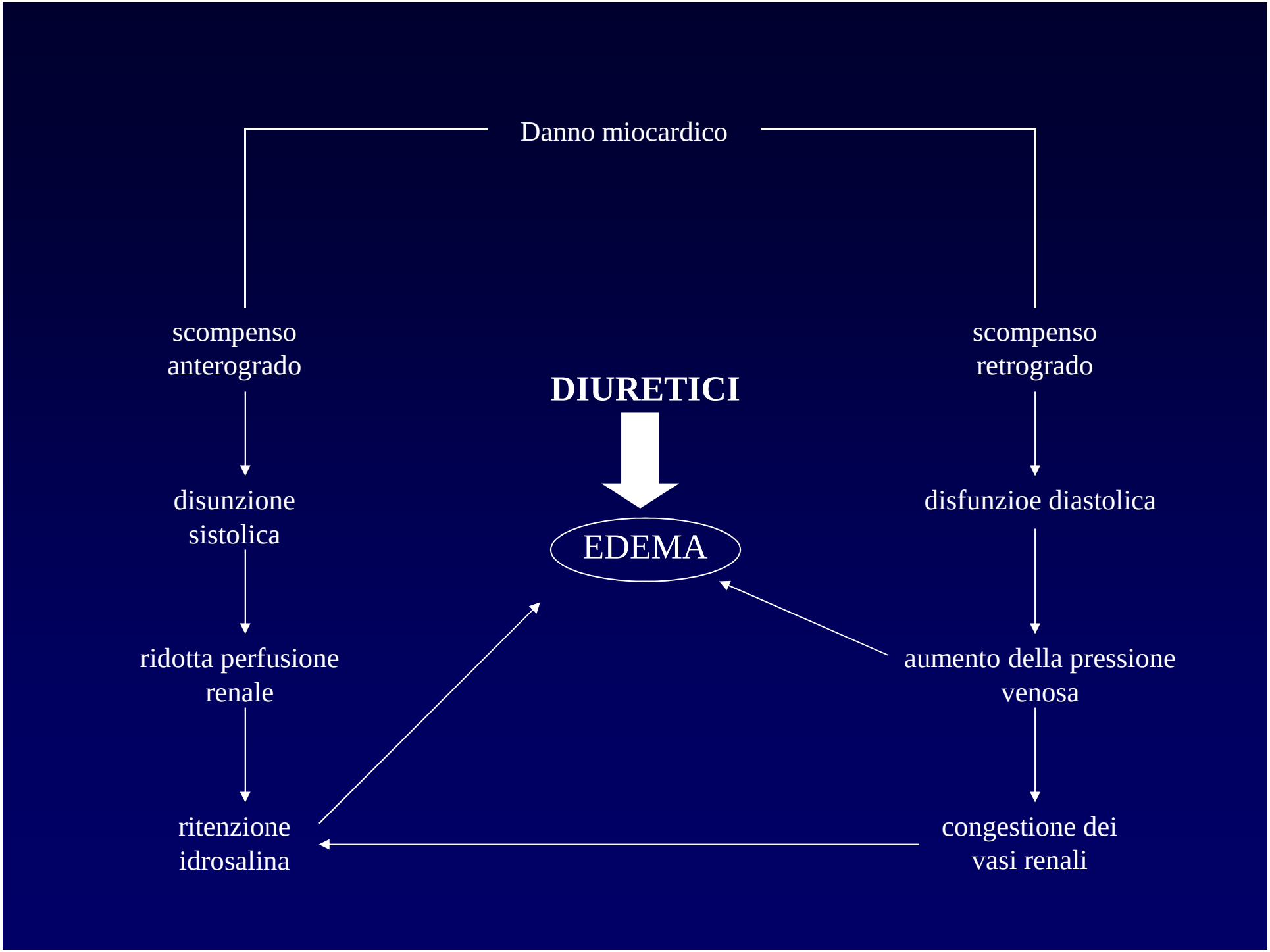
- Ipertensione arteriosa
- Cardiopatia ischemica cronica
- Pregresso infarto miocardico
- Valvulopatia

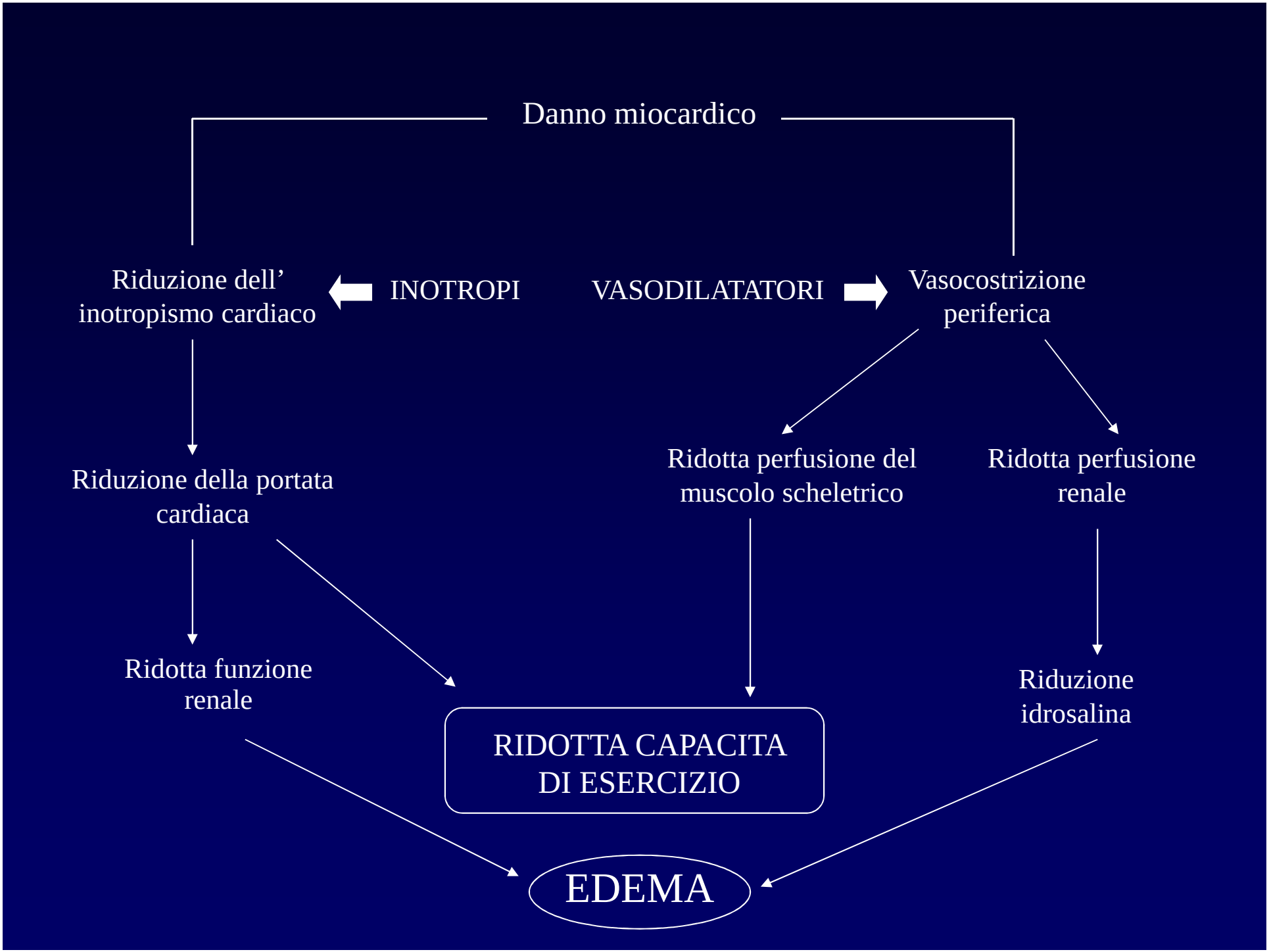
→ Monitorare regolarmente la funzione ventricolare con eco

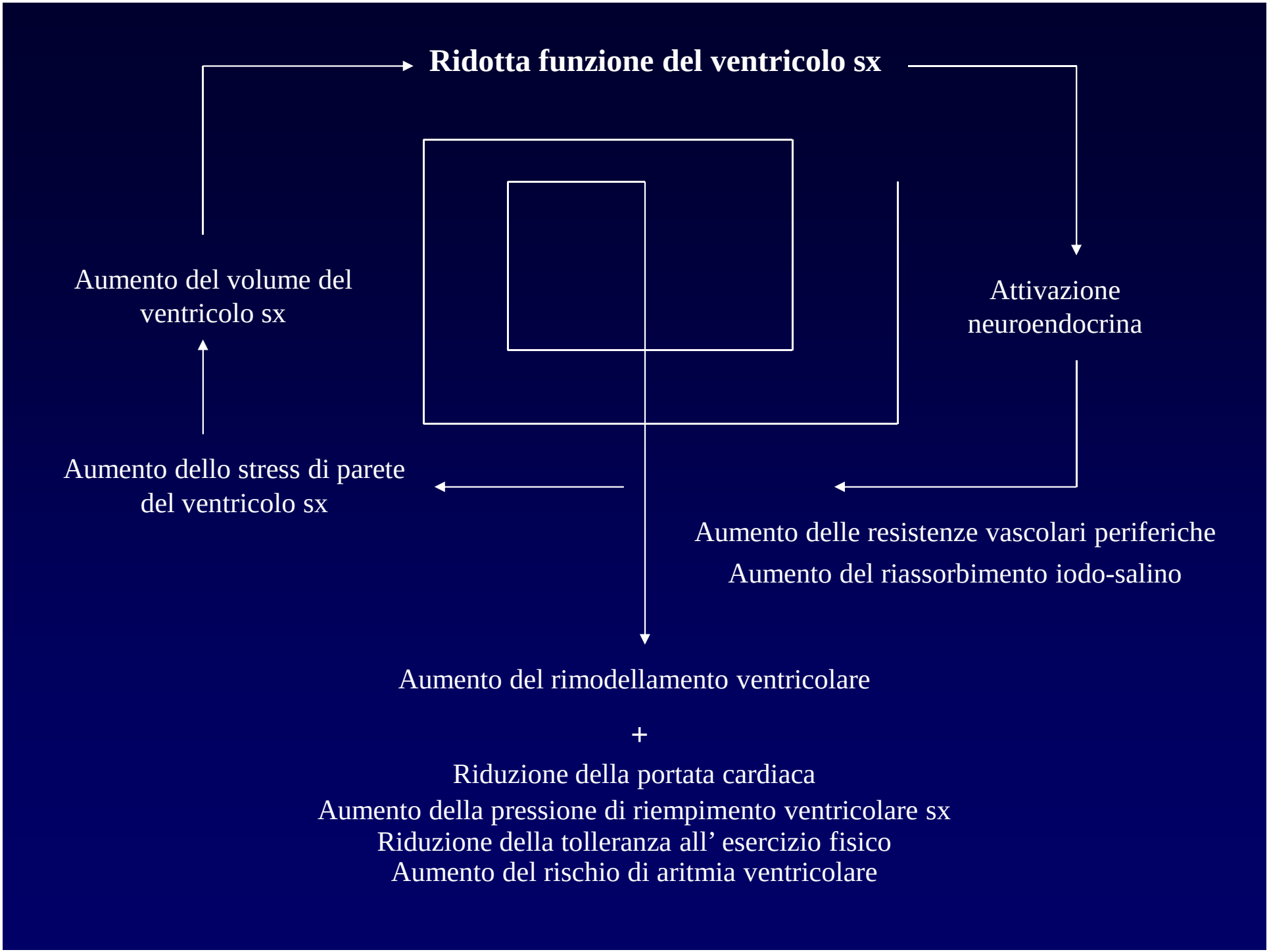
Segni clinici suggestivi di cardiopatia ad evoluzione vs disfunzione ventricolare

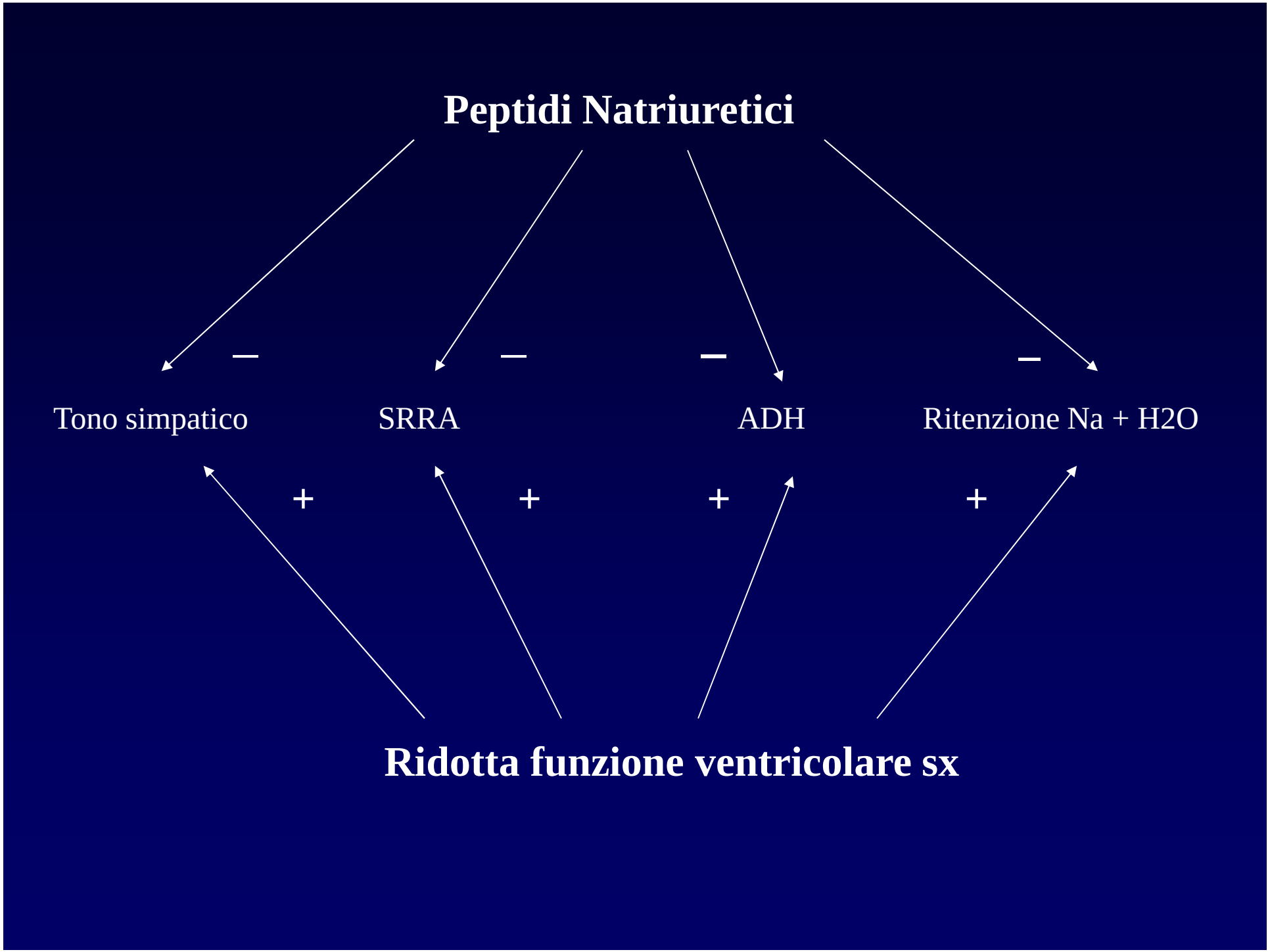
- Tachicardia sinusale
- Blocco di branca sinistra
- Aritmie ventricolari
- Reperto radiografico di cardiomeglia

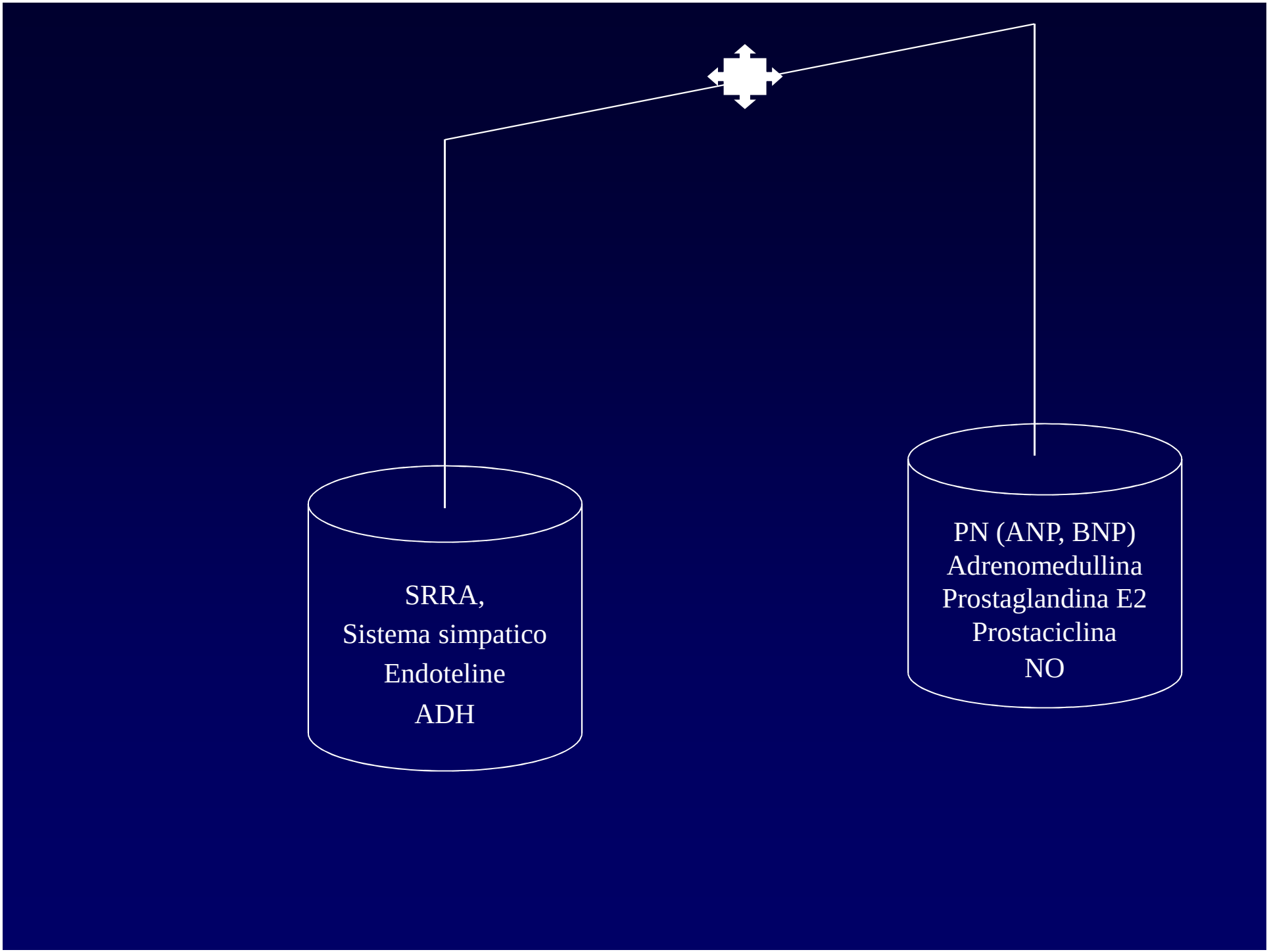
→
- Valutazione della funzione ventricolare a riposo e da sforzo
- Ecocardiogramma
- Angiocardioscintigrafia

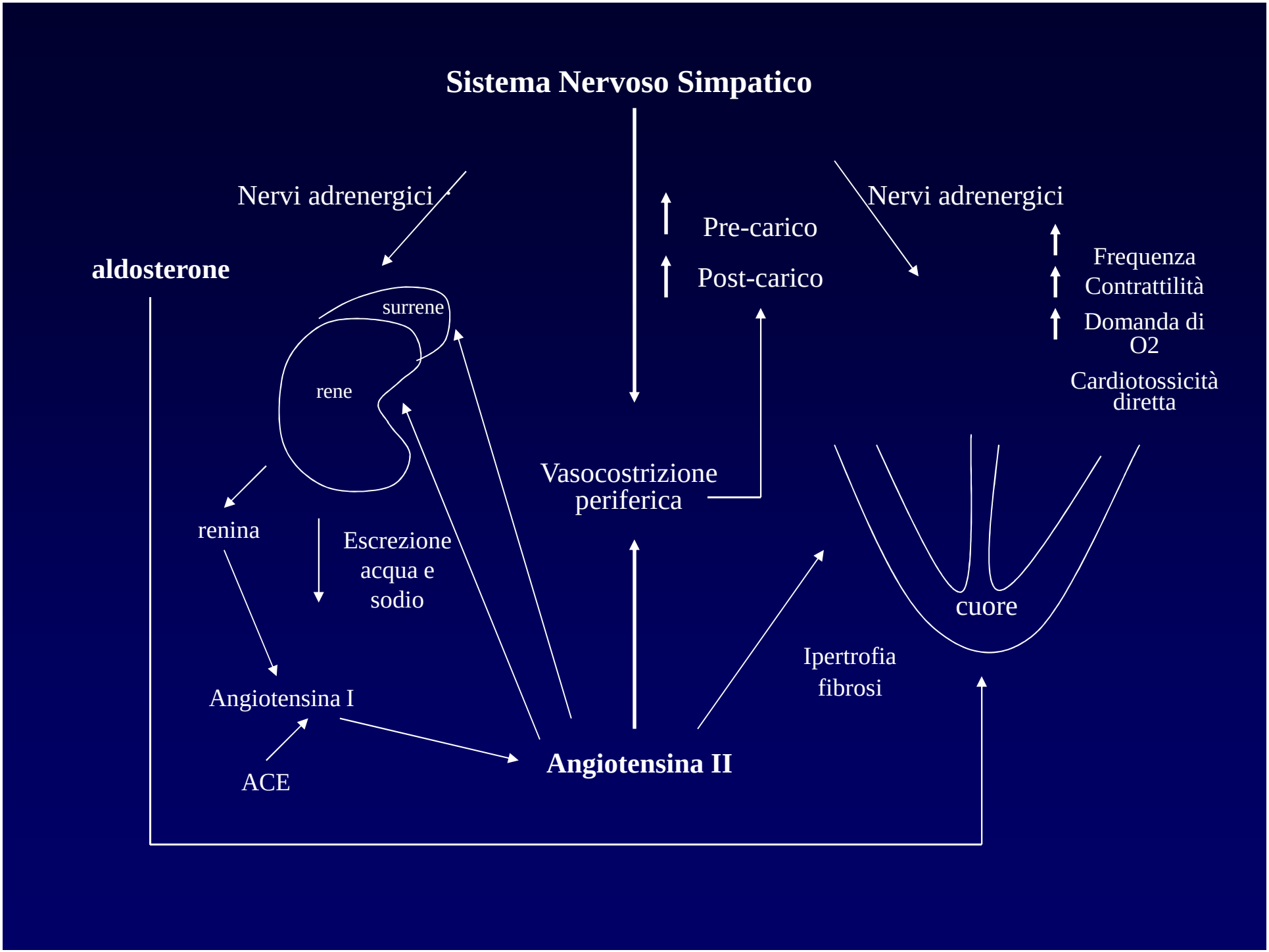

Danno miocardico
scompenso anterogrado
scompenso retrogrado
DIURETICI
disunzione sistolica
disfunzioe diastolica
EDEMA
ridotta perfusione renale
aumento della pressione venosa
ritenzione idrosalina
congestione dei vasi renali


Danno miocardico
Riduzione dell' inotropismo cardiaco
INOTROPI
VASODILATATORI
Vasocostrizione periferica
Riduzione della portata cardiaca
Ridotta perfusione del muscolo scheletrico
Ridotta perfusione renale
Ridotta funzione renale
Riduzione idrosalina
RIDOTTA CAPACITA DI ESERCIZIO
EDEMA


Ridotta funzione del ventricolo sx
Aumento del volume del ventricolo sx
Attivazione neuroendocrina
Aumento dello stress di parete del ventricolo sx
Aumento delle resistenze vascolari periferiche
Aumento del riassorbimento iodo-salino
Aumento del rimodellamento ventricolare
+
Riduzione della portata cardiaca
Aumento della pressione di riempimento ventricolare sx
Riduzione della tolleranza all' esercizio fisico
Aumento del rischio di aritmia ventricolare


Peptidi Natriuretici
–
–
–
–
Tono simpatico
SRRA
ADH
Ritenzione Na + H2O
+
+
+
+
Ridotta funzione ventricolare sx


SRRA,
Sistema simpatico
Endoteline
ADH
PN (ANP, BNP)
Adrenomedullina
Prostaglandina E2
Prostaciclina
NO


Sistema Nervoso Simpatico
Nervi adrenergici
Nervi adrenergici
Pre-carico
Post-carico
Frequenza
Contrattilità
Domanda di O2
Cardiotossicità diretta
aldosterone
surrene
rene
renina
Escrezione acqua e sodio
Vasocostrizione periferica
cuore
Ipertrofia fibrosi
Angiotensina I
ACE
Angiotensina II

SHOCK CARDIOCIRCOLATORIO o EMODINAMICO

Sindrome **evolutiva** con segni e sintomi caratteristici, dovuta ad uno squilibrio fra apporto e fabbisogno di O_2 che conduce allo sviluppo di ipossia **tessutale/cellulare**

Concetto di "Cell Energy Failure"