



Les noves guies d' Insuficiència Cardíaca de la ESC-HFA 2016 a debat

Recomanacions a la insuficiència cardíaca aguda

Josep Masip Utset
Consorci Sanitari Integral

Table 12.1 Factors triggering 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.
Bradycardia.
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

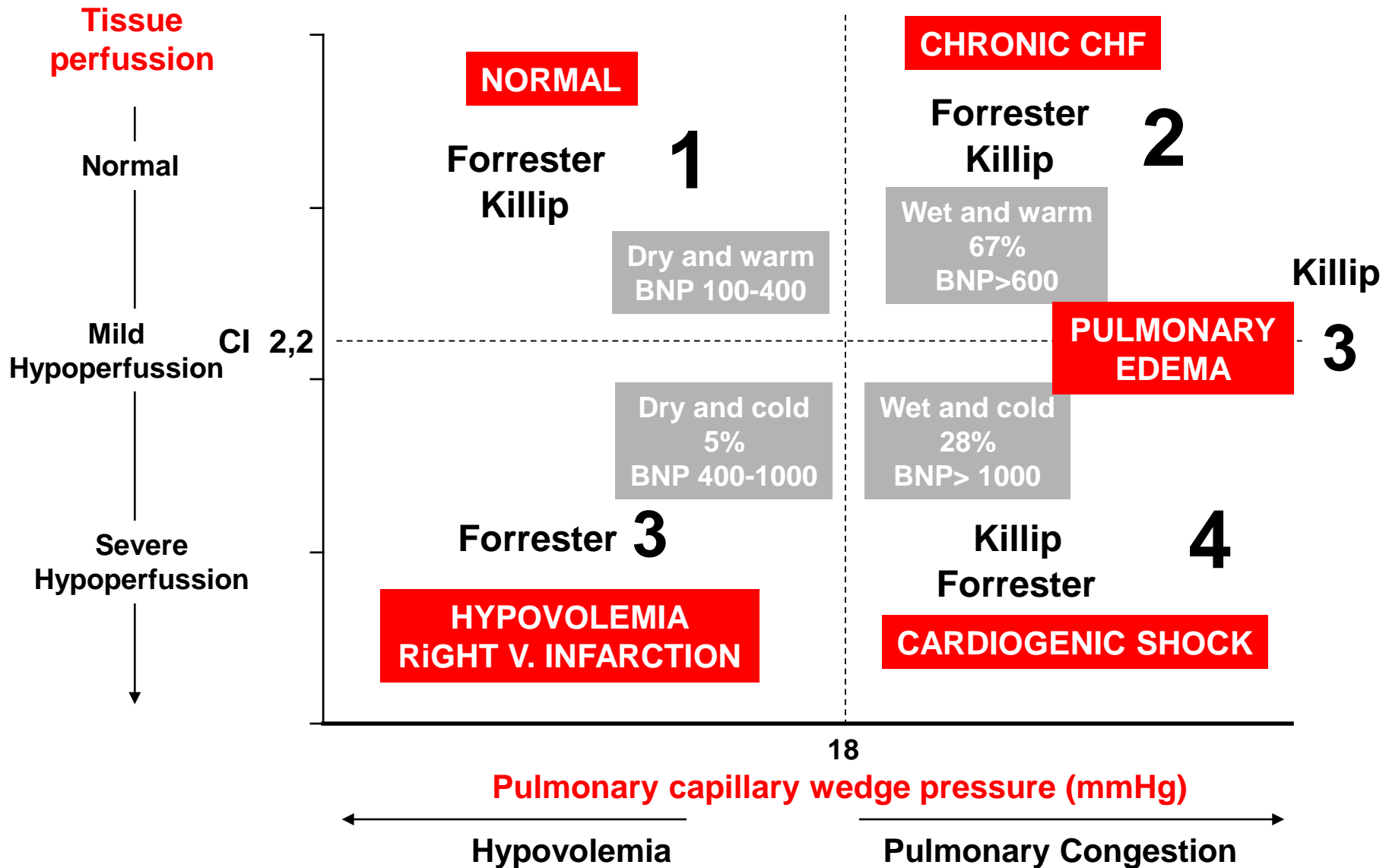
HYPOPERFUSION (+)

- Cold sweated extremities
- Oliguria
- Mental confusion
- Dizziness
- Narrow pulse pressure

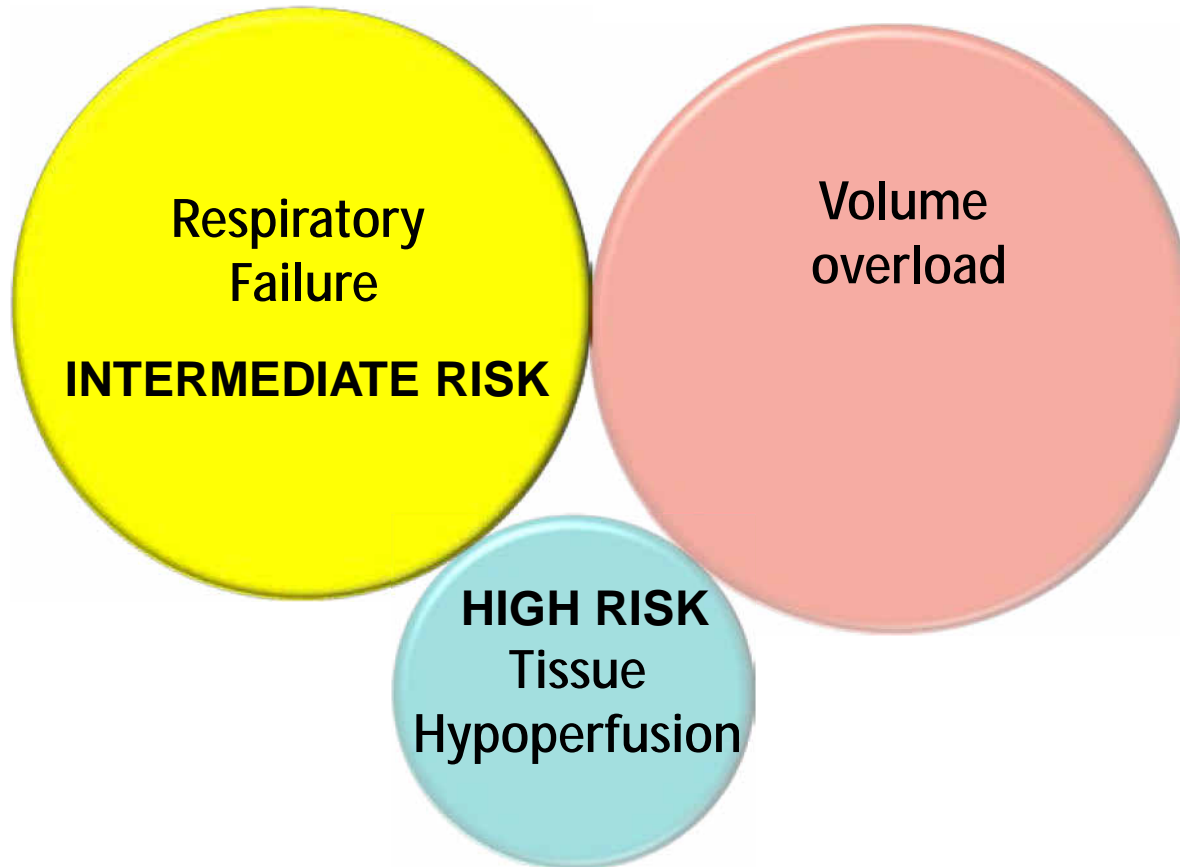
COLD-DRY

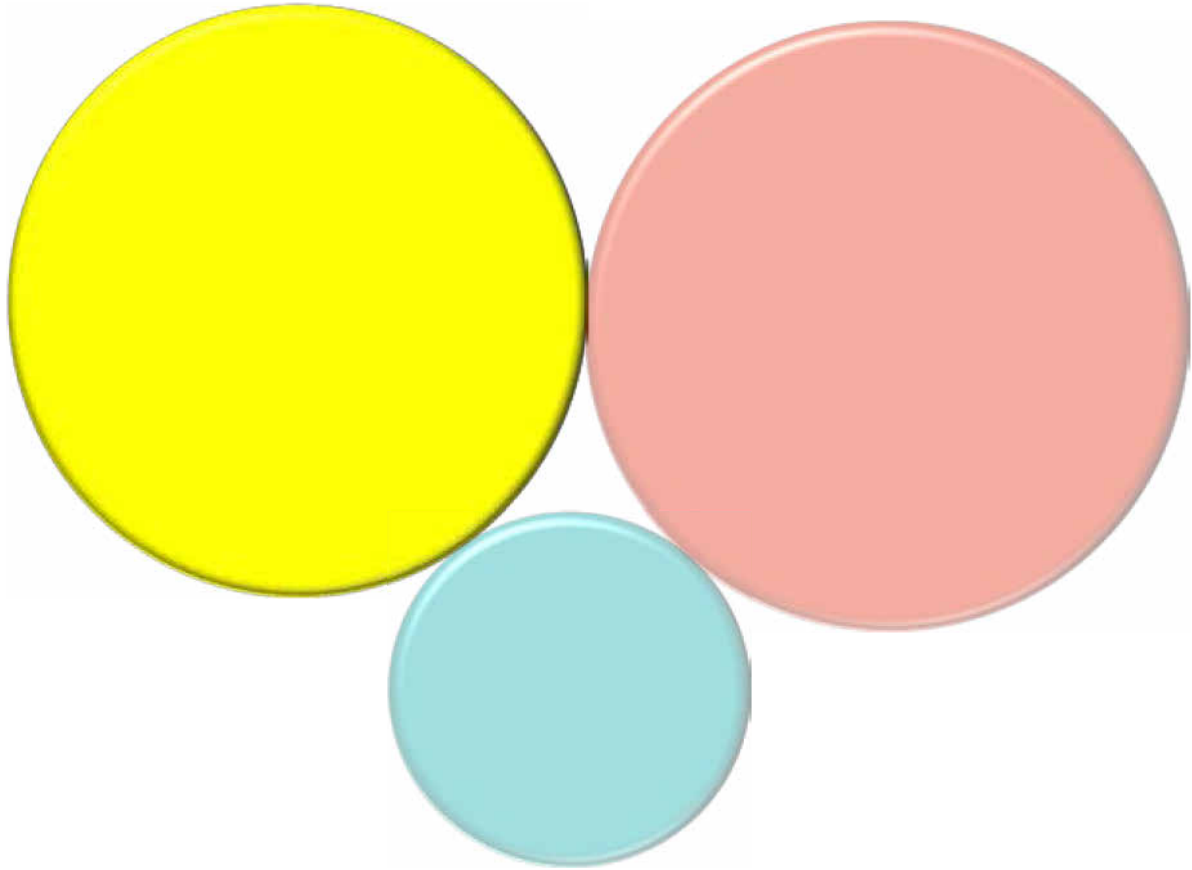
COLD-WET

CLINICAL AND FUNCTIONAL CLASSIFICATION

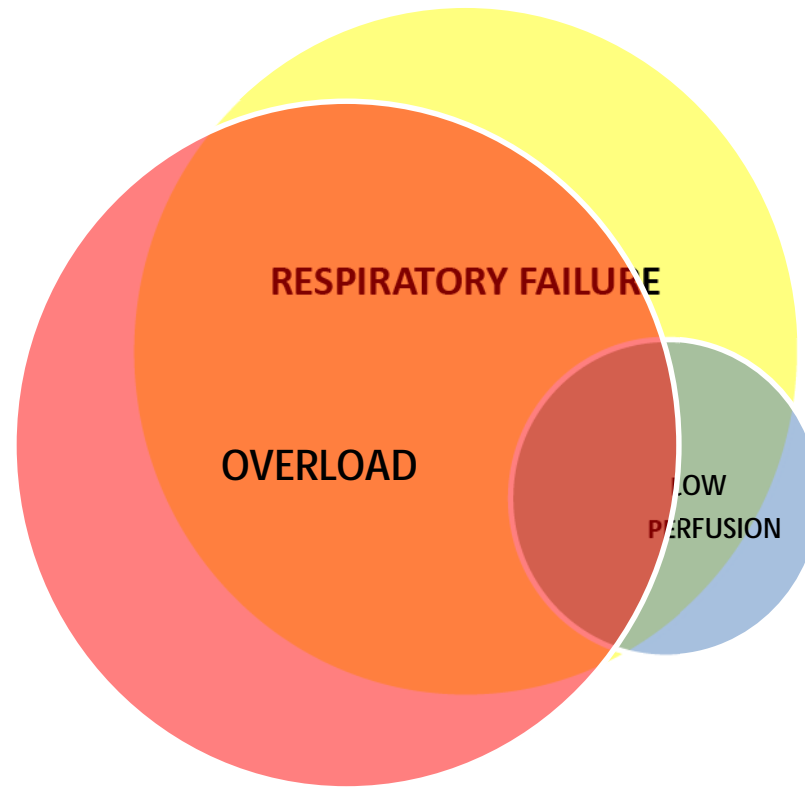


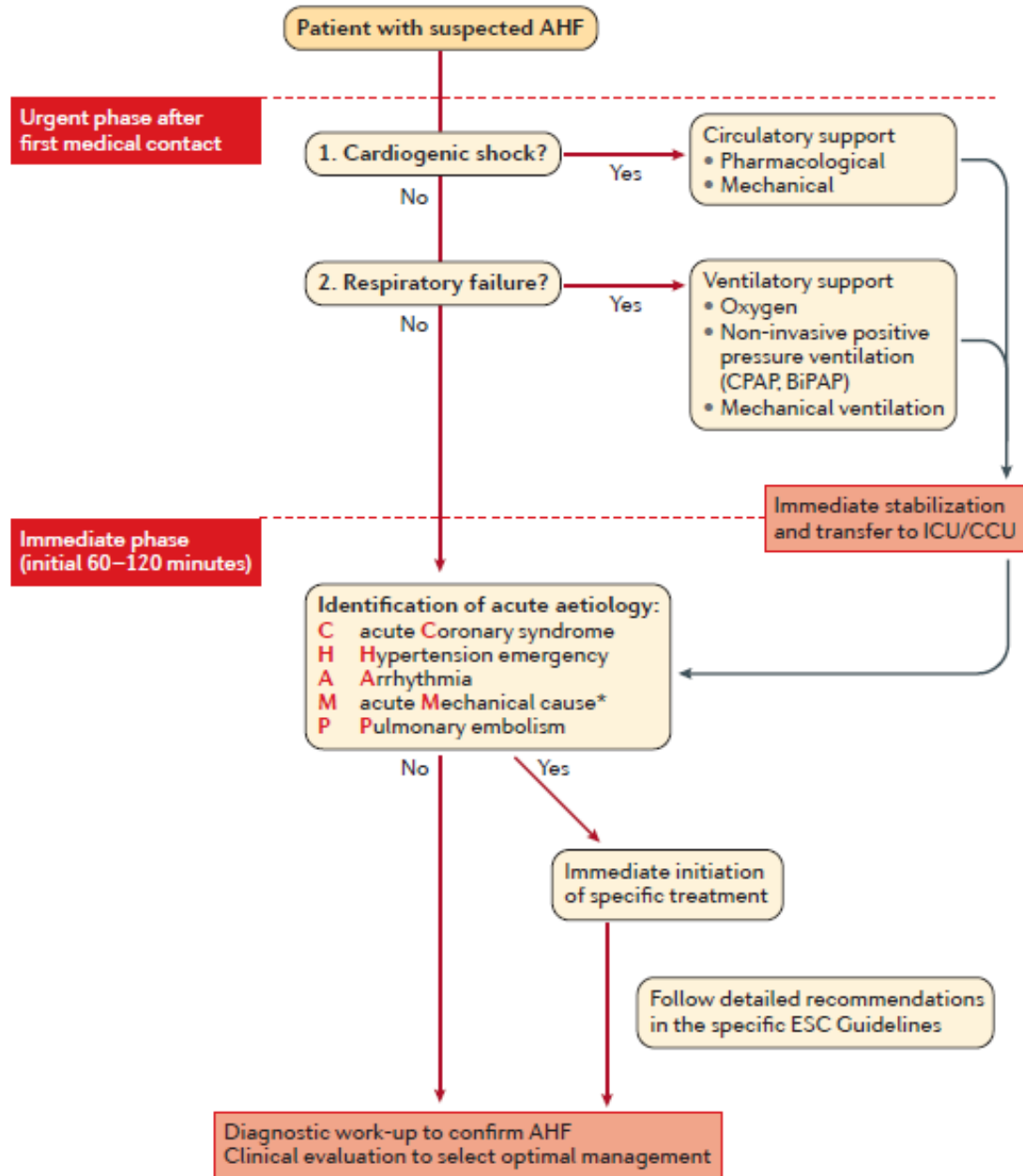
Main alterations in AHF





Main alterations in AHF



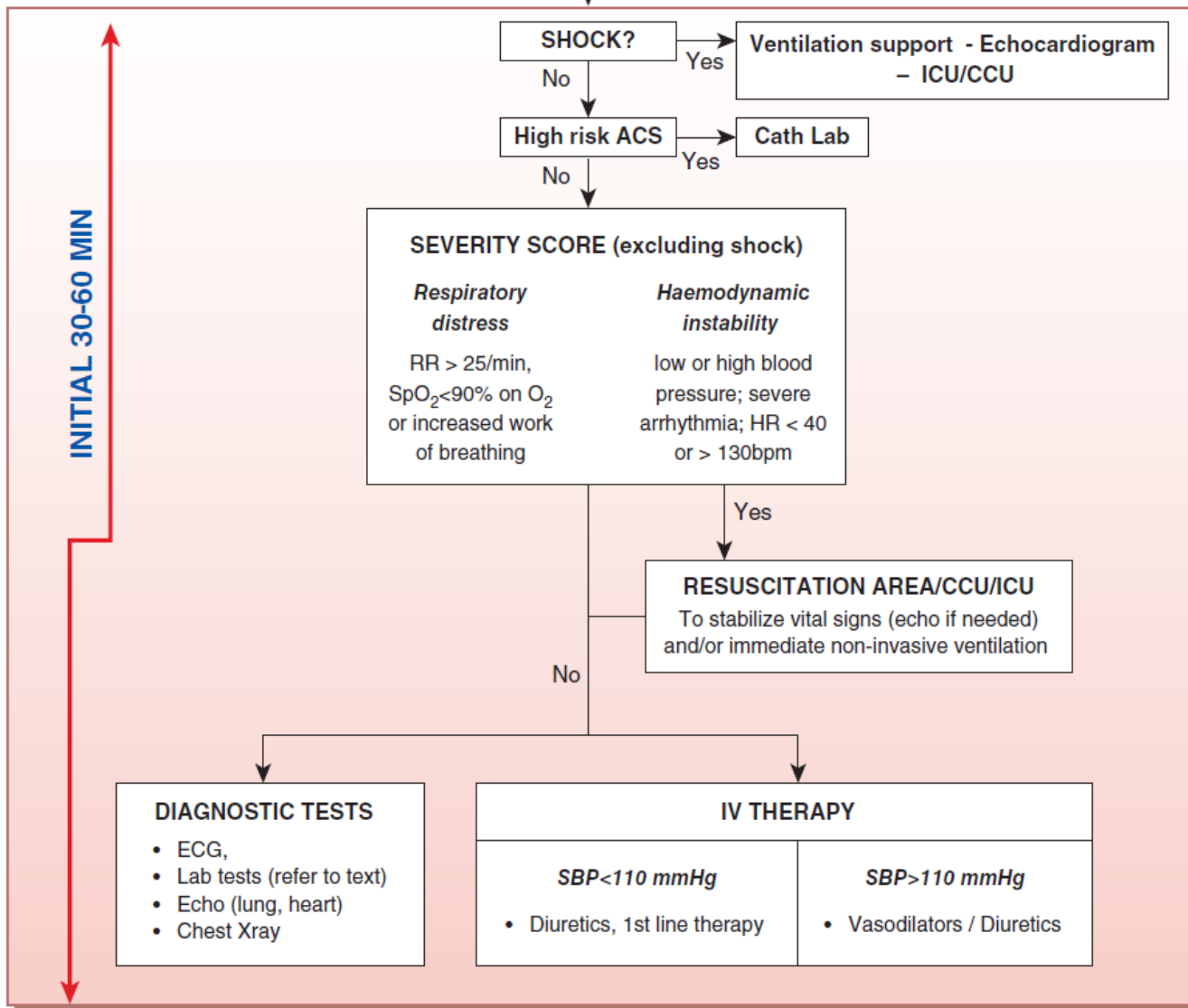


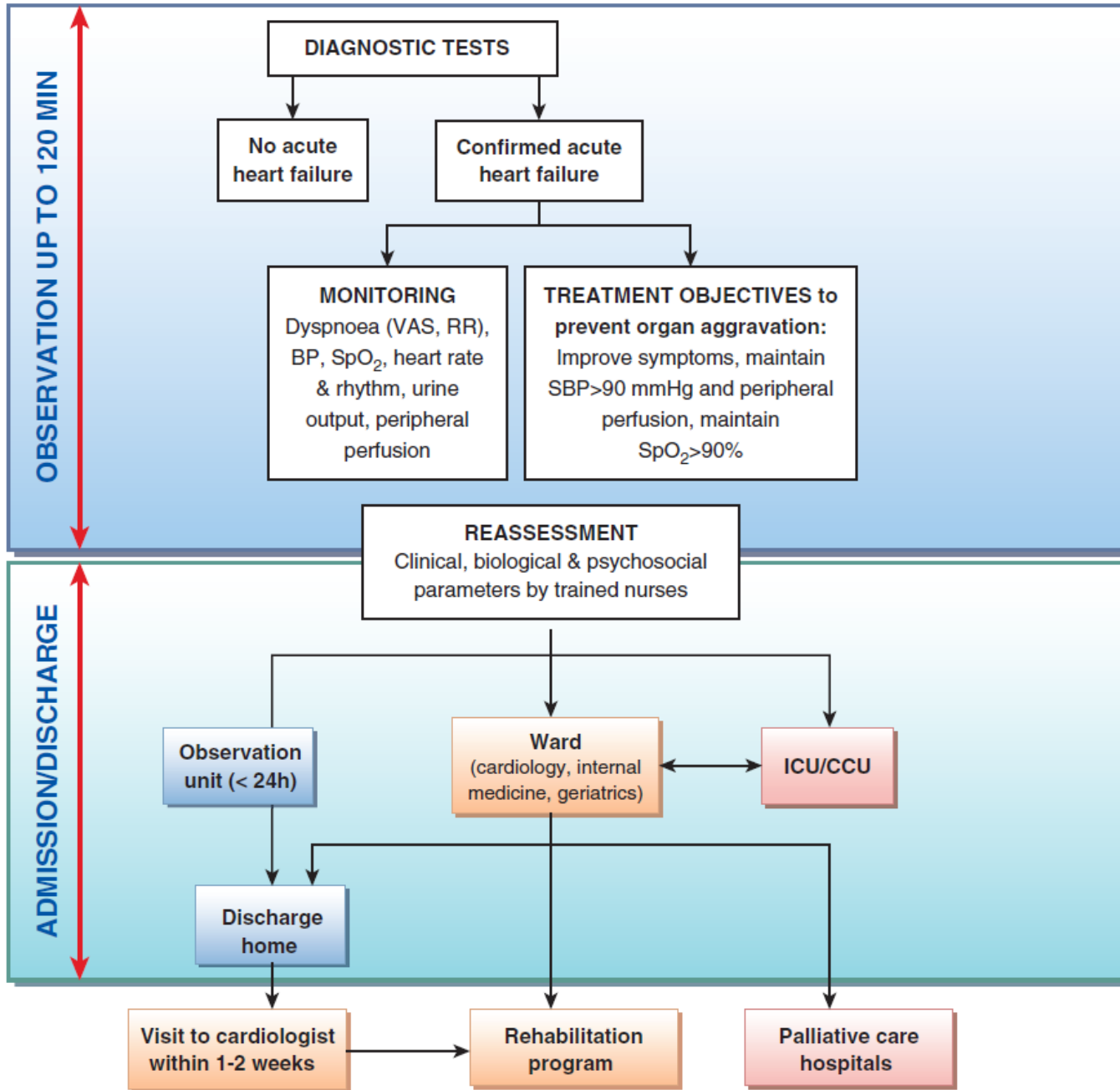


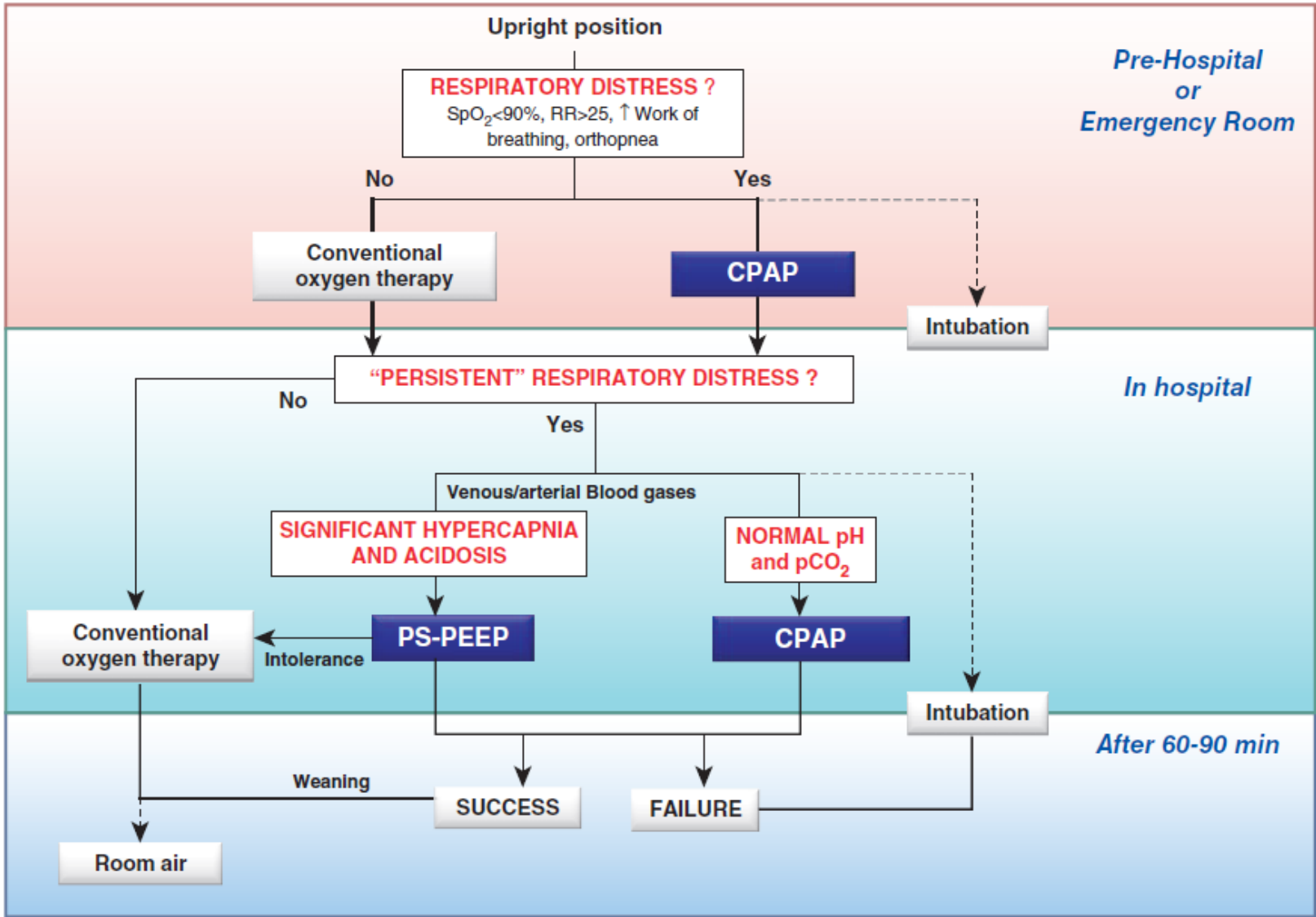
Recommendations on pre-hospital and early hospital management of acute heart failure: a consensus paper from the Heart Failure Association of the European Society of Cardiology, the European Society of Emergency Medicine and the Society of Academic Emergency Medicine – short version

Alexandre Mebazaa^{1*}, M. Birhan Yilmaz², Phillip Levy³, Piotr Ponikowski⁴, W. Frank Peacock⁵, Said Laribi⁶, Arsen D. Ristic⁷, Ekaterini Lambrinou⁸, Josep Masip⁹, Jillian P. Riley¹⁰, Theresa McDonagh¹¹, Christian Mueller¹², Christopher deFilippi¹³, Veli-Pekka Harjola¹⁴, Holger Thiele¹⁵, Massimo F. Piepoli¹⁶, Marco Metra¹⁷, Aldo Maggioni¹⁸, John J.V. McMurray¹⁹, Kenneth Dickstein²⁰, Kevin Damman²¹, Petar M. Seferovic^{22,23}, Frank Ruschitzka²⁴, Adelino F. Leite-Moreira^{25,26}, Abdelouahab Bellou^{27,28}, Stefan D. Anker^{29,30}, and Gerasimos Filippatos³¹

SUSPECTED AHF







Upright position

RESPIRATORY DISTRESS ?

$SpO_2 < 90\%$, $RR > 25$, \uparrow Work of breathing, orthopnea

No

Yes

Conventional oxygen therapy

CPAP

Intubation

Pre-Hospital or Emergency Room

"PERSISTENT" RESPIRATORY DISTRESS ?

No

Yes

In hospital

Venous/arterial Blood gases

SIGNIFICANT HYPERCAPNIA AND ACIDOSIS

NORMAL pH and pCO_2

Conventional oxygen therapy

PS-PEEP

CPAP

Intolerance

Intubation

Weaning

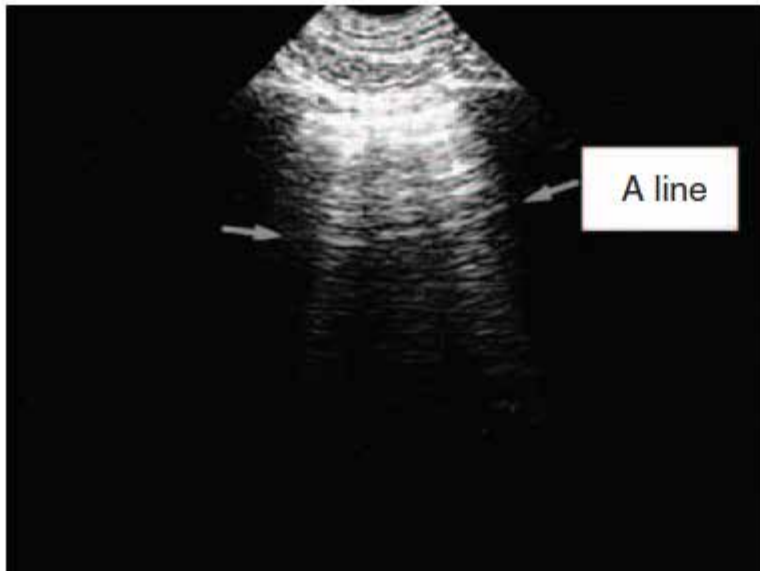
SUCCESS

FAILURE

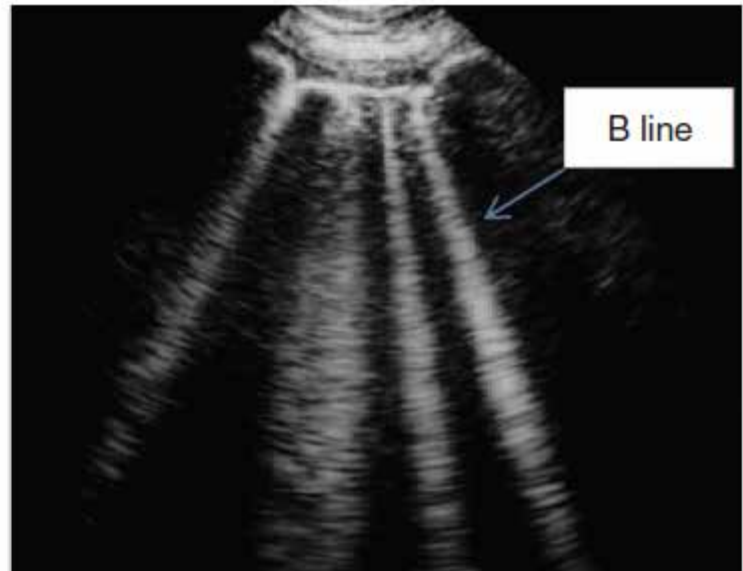
After 60-90 min

Room air

A



B



Chest X-ray

- Up to 20% of patients with AHF is nearly normal
- Supine chest radiographs are of limited value in AHF
- To identify alternative non-cardiac diseases



ECG

- Is rarely normal in AHF (high negative predictive value)
- Helpful in identifying underlying cardiac disease and potential precipitants (rapid AF, ACS)



Echocardiography

- Immediate is mandatory only in patients with hemodynamic instability (particularly in CS) and in patients suspected of acute life-threatening structural or functional cardiac abnormalities (mechanical, valvular, aortic dissection).
- Early in *de novo AHF or unknown cardiac function* (≤ 48 h)
- Pocket-size may be used as an extension of the clinical examination Repeated echocardiography not needed unless relevant deterioration in clinical status



Bedside thoracic ultrasound

- For signs of interstitial edema and pleural effusion if the expertise is available

Pulmonary artery catheter, arterial or central venous line and urinary catheter

- Are not routinely indicated

Plasma natriuretic peptide level (BNP, NT-proBNP or MR-proANP)

Upon presentation is recommended in all patients with acute dyspnea and suspected AHF to help in the differentiation of AHF from non-cardiac causes of acute dyspnea



AHF may be disclosed:
BNP <100 pg/mL
NT-proBNP <300 pg/mL
MR-proANP <120 pg/mL

Predischarge may be considered for prognostic evaluation

Table 12.3 Causes of elevated concentrations of natriuretic peptides^{522–524}

Cardiac	Heart failure Acute coronary syndromes Pulmonary embolism Myocarditis Left ventricular hypertrophy Hypertrophic or restrictive cardiomyopathy Valvular heart disease Congenital heart disease Atrial and ventricular tachyarrhythmias Heart contusion Cardioversion, ICD shock Surgical procedures involving the heart Pulmonary hypertension
Non-cardiac	Advanced age Ischaemic stroke Subarachnoid haemorrhage Renal dysfunction Liver dysfunction (mainly liver cirrhosis with ascites) Paraneoplastic syndrome Chronic obstructive pulmonary disease Severe infections (including pneumonia and sepsis) Severe burns Anaemia Severe metabolic and hormone abnormalities (e.g. thyrotoxicosis, diabetic ketosis)

Other laboratory tests at presentation

Cardiac troponins, BUN (or urea), creatinine, electrolytes (sodium, potassium), glucose, complete blood count, liver function tests and TSH



D-dimer in suspicion of acute pulmonary embolism

Cardiac troponins are elevated in the vast majority of patients with AHF, often without obvious myocardial ischaemia or an acute coronary event, suggesting ongoing myocyte injury or necrosis. Also in acute pulmonary embolism is useful for risk stratification and decision-making

Routine **arterial blood gas** is not needed. Restricted when SpO₂ is not reliable
Venous blood gases might acceptably indicate pH and CO₂

Renal function and electrolytes every 1–2 days or even more frequently

Procalcitonin levels may be considered in patients with AHF with suspected coexisting infection, particularly for the differential diagnosis of pneumonia and to guide antibiotic therapy.

Liver function tests (reduced CO and/or increased CVP). Identify patients of poor prognosis

TSH should be assessed in newly diagnosed (hypothyroidism and hyperthyroidism may precipitate AHF)

Other **biomarkers** have been investigated for their diagnostic and prognostic value in AHF, but none has reached the stage of being recommended for routine clinical use

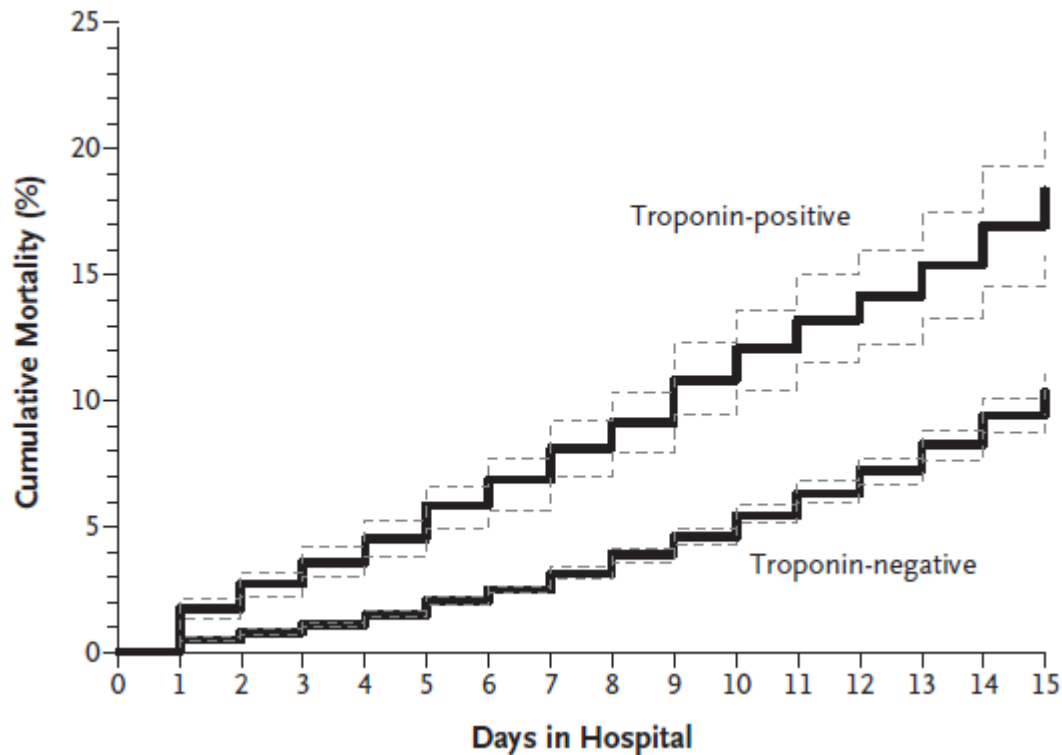
ADHERE Registry :

4240 out of 67920 patients (6.2%) were Troponin+

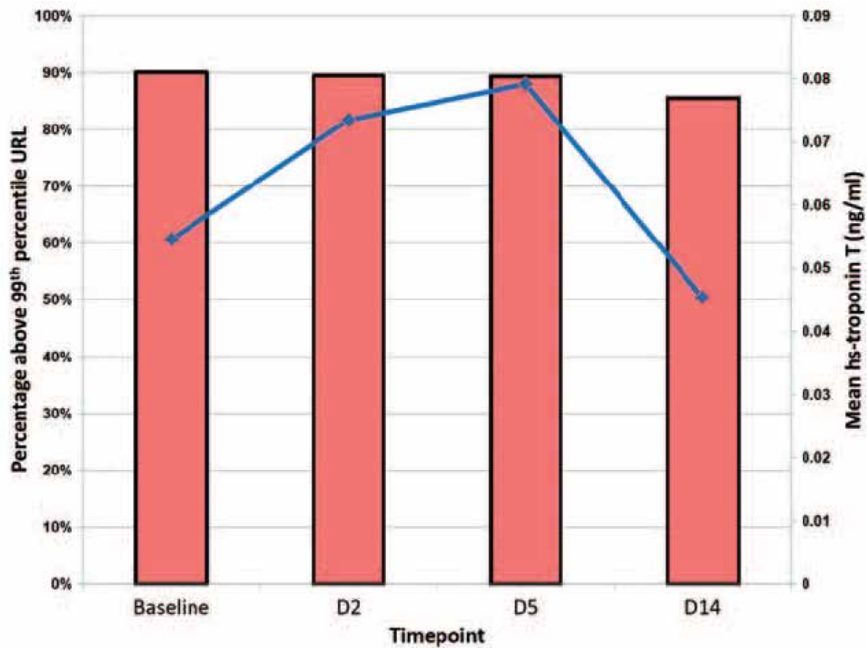
Patients Troponin +: lower SBP on admission,

lower LVEF

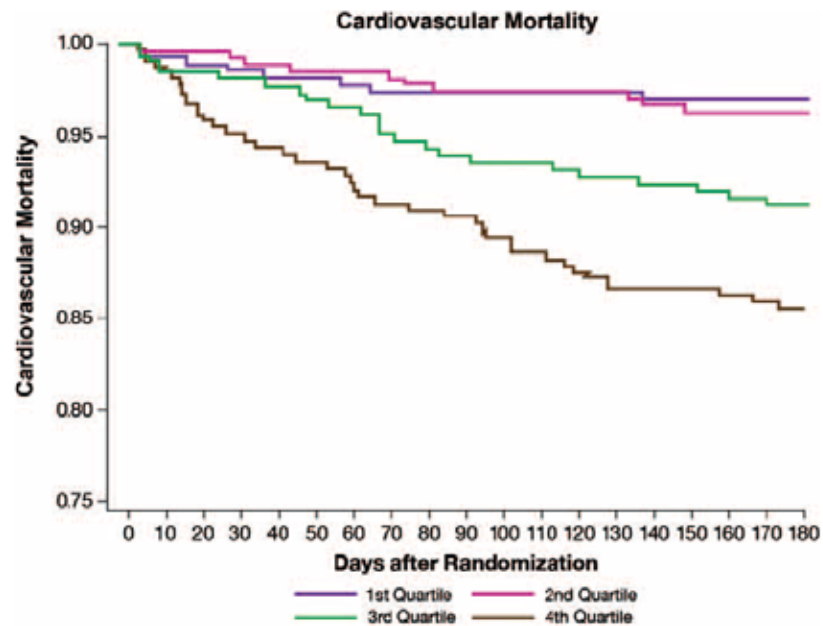
Higher in-hospital mortality (8.0% vs. 2.7%, $P < 0.001$)



Peacock WF. NEJM 2008



Mean hs-cTnT values —◆—



Baseline high-sensitivity cardiac troponin T

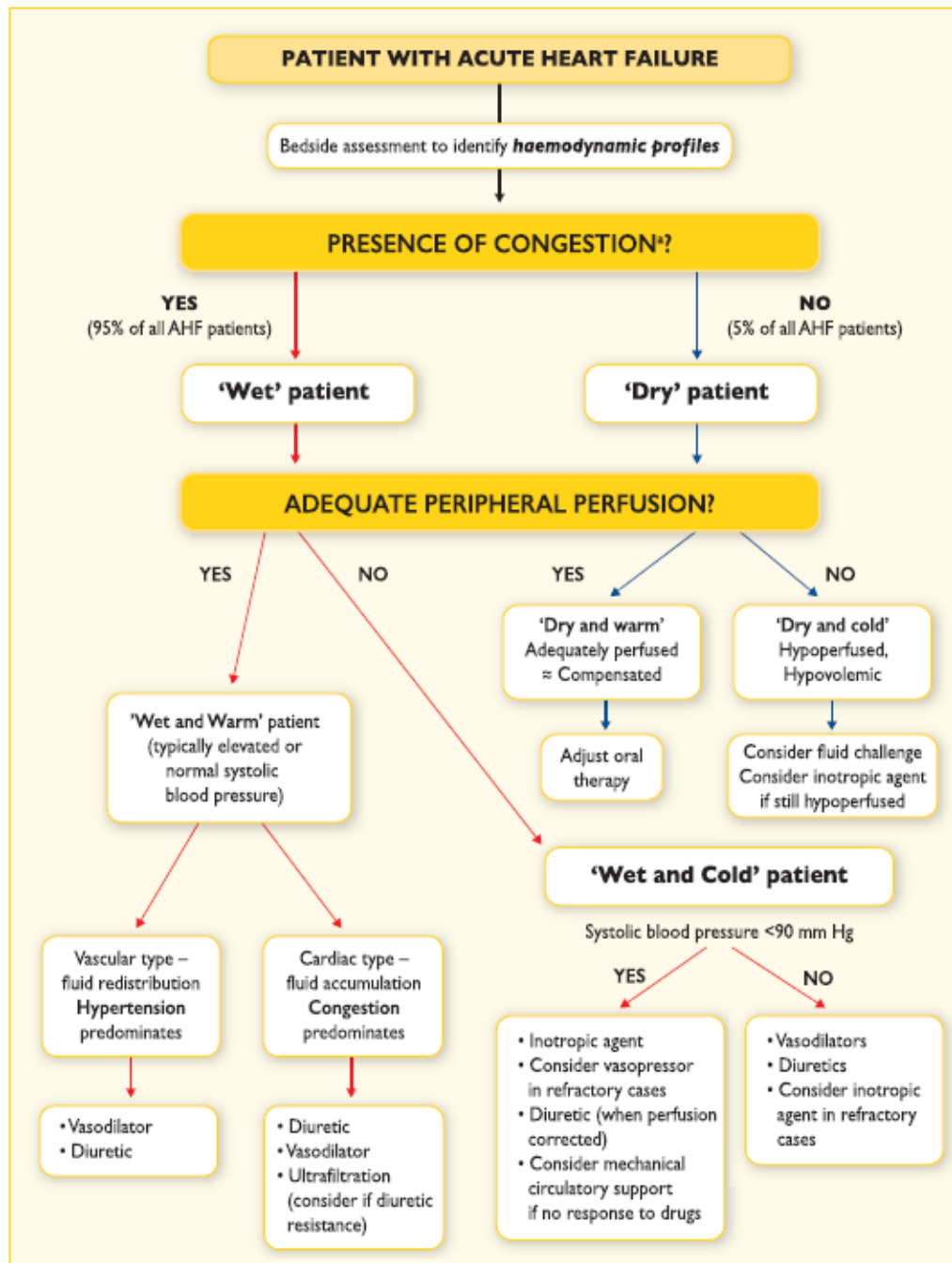
Felker M. Eur J Heart Fail 2015

Recommendations regarding monitoring of clinical status of patients hospitalized due to acute heart failure

Recommendations	Class ^a	Level ^b
Standard non-invasive monitoring of heart rate, rhythm, respiratory rate, oxygen saturation and blood pressure is recommended.	I	C
It is recommended that patients should be weighed daily and have an accurate fluid balance chart completed.	I	C
It is recommended to evaluate signs and symptoms relevant to HF (e.g. dyspnoea, pulmonary rales, peripheral oedema, weight) daily to assess correction of fluid overload.	I	C
Frequent, often daily, measurement of renal function (blood urea, creatinine) and electrolytes (potassium, sodium) during i.v. therapy and when renin-angiotensin-aldosterone system antagonists are initiated is recommended.	I	C
Intra-arterial line should be considered in patients with hypotension and persistent symptoms despite treatment.	IIa	C
Pulmonary artery catheter may be considered in patients who, despite pharmacological treatment present refractory symptoms (particularly with hypotension and hypoperfusion).	IIb	C

Recommendations for the management of patients with acute heart failure: oxygen therapy and ventilatory support

Recommendations	Class ^a	Level ^b	Ref ^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 (8.0 kPa) 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	541–545
Intubation is recommended, if respiratory failure, leading to hypoxaemia (PaO ₂ <60 mmHg (8.0 kPa)), hypercapnia (PaCO ₂ >50 mmHg (6.65 kPa)) and acidosis (pH <7.35), cannot be managed non-invasively.	I	C	



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

AHF with hypoperfusion, diuretics should be avoided before adequate perfusion is attained

Combination requires careful monitoring to avoid hypokalaemia, renal dysfunction and hypovolemia.

Optimal dosing, timing and method of delivery is unclear. In the 'high-dose' arm of the DOSE study, administration of furosemide at 2.5 times the previous oral dose resulted in greater improvement in dyspnea, larger weight change and fluid loss at the cost of transient worsening in renal function. The dose should be the smallest to provide adequate clinical effect.

A bolus of 10–20 mg i.v. torasemide may be considered as an alternative.

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

Intravenous vasodilators are the second most often used agents in AHF for symptomatic relief; however, there is no robust evidence confirming their beneficial effects

Are especially useful in patients with hypertensive AHF, whereas in those with SBP<90 mmHg (or with symptomatic hypotension) should be avoided

Dosing should be carefully controlled to avoid excessive decreases in BP, 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

Inotropes should be reserved for cases with severe CO reduction, compromising vital organs, mainly in hypotensive-AHF (excluding hypovolemia or other reversible causes)

Levosimendan is preferable over dobutamine to reverse the effect of betablockade if beta-blockade is thought to be contributing to hypoperfusion. In patients with hypotension (SBP <85 mmHg) or cardiogenic shock, in combination with other inotropes or vasopressors

ECG monitoring is required for administering inotropes, because they can cause tachycardia, ischemia and arrhythmias, and may increase mortality. In any case, inotropes have to be used with caution starting from rather low doses and up-titrating with close monitoring

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

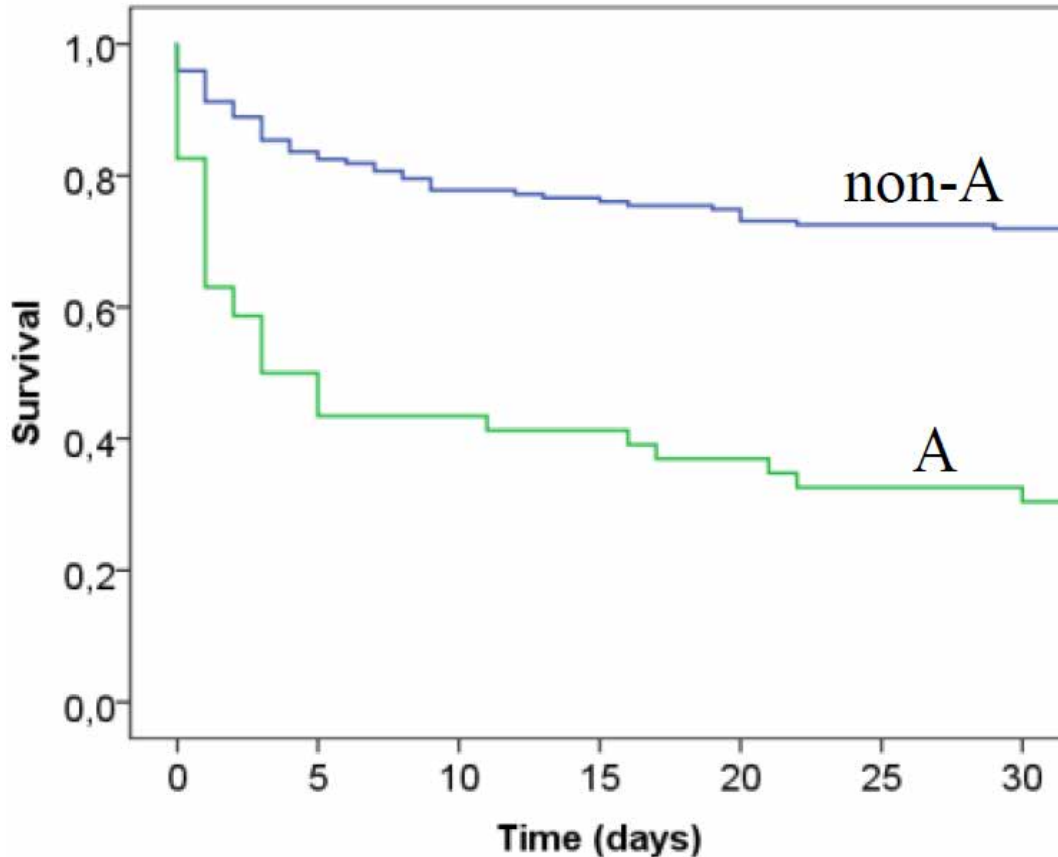
These agents are given to raise BP and redistribute blood to the vital organs. However, this is at the expense of an increase in LV afterload.

Dopamine was compared with norepinephrine in the treatment of various shock patients. A subgroup analysis suggested that norepinephrine would have fewer side effects and lower mortality.

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.



ADRENALINE was independently associated with mortality



Adjusted for center
and gender

The association
remained after
Propensity score
analysis

OR 4.3 (1.5-12.6) $p < 0.0507$

Harjola VP. Eur Heart J 2015

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.	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 is mostly indicated in AF and FC>110 bpm. However, in patients with co-morbidities or other drugs affecting digoxin metabolism and/or the elderly, the maintenance dose may be based on the measurements of digoxin concentration in peripheral blood

Tolvaptan may be used to treat patients with volume overload and resistant hyponatremia (thirst and dehydration are recognized adverse effects).

Routine use of opiates is not recommended and they may only be cautiously considered in patients with severe dyspnea, mostly with pulmonary edema. Dose-dependent side effects include nausea, hypotension, bradycardia and respiratory depression.

There are controversies regarding the potentially elevated mortality risk in patients receiving morphine.

Anxiolytics or sedatives may be needed in a patient with agitation or delirium. Cautious use of benzodiazepines (diazepam or lorazepam) may be the safest approach.

Recommendations regarding oral evidence-based disease-modifying therapies in patients with acute heart failure

Recommendations	Class ^a	Level ^b
In case of worsening of chronic HFrEF, every attempt should be made to continue evidence-based, disease-modifying therapies, in the absence of haemodynamic instability or contraindications.	I	C
In the case of <i>de novo</i> HFrEF, every attempt should be made to initiate these therapies after haemodynamic stabilization.	I	C

Recommendations	Class ^a	Level ^b
Ultrafiltration may be considered for patients with refractory congestion, who failed to respond to diuretic-based strategies.	IIb	B
Renal replacement therapy should be considered in patients with refractory volume overload and acute kidney injury.	IIa	C

Routine use of ultrafiltration is not recommended and should be confined to patients who fail to respond to diuretic based strategies.

The following criteria may indicate the need for initiation of renal replacement therapy in patients with refractory volume overload:

- **oliguria unresponsive to fluid resuscitation measures**
- **severe hyperkalaemia (K>6.5 mmol/L)**
- **severe acidaemia (pH <7.2)**
- **serum urea level >25 mmol/L (150 mg/dL)**
- **serum creatinine >300 mmol/L (>3.4 mg/dL)**

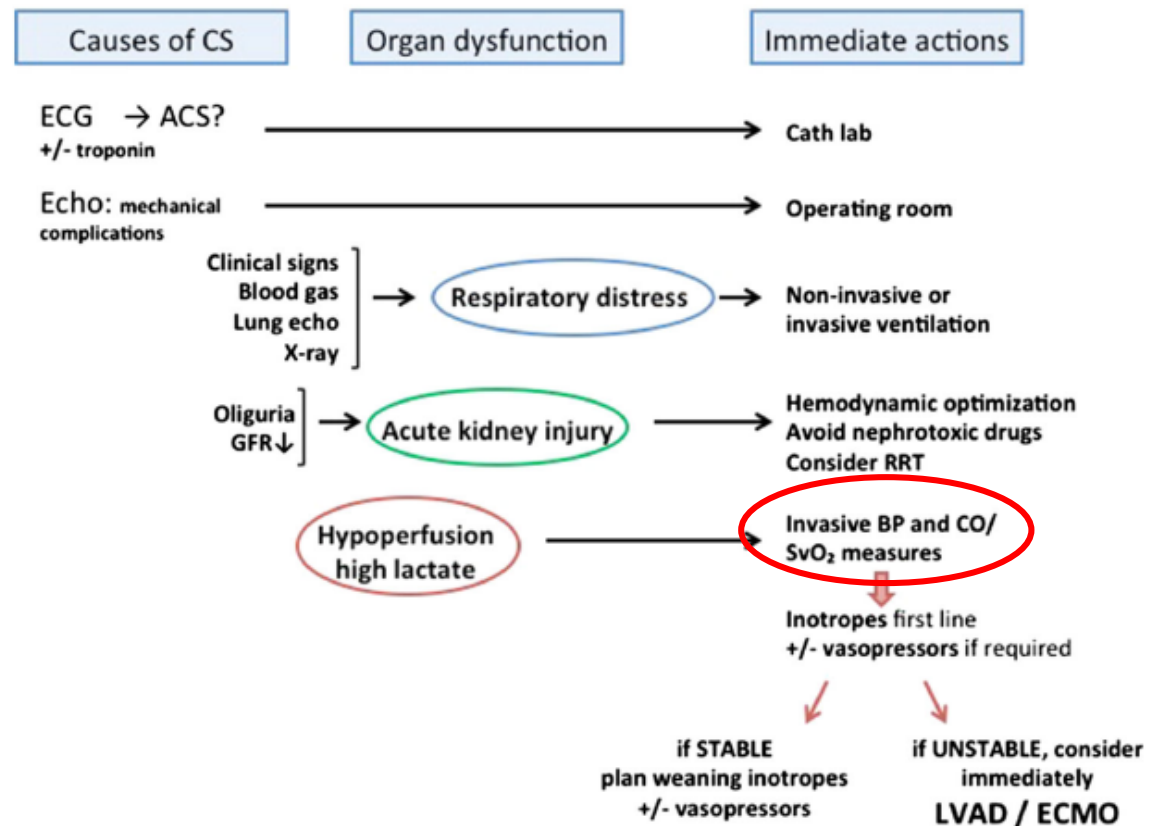
Recommendations regarding management of patients with cardiogenic shock

Recommendations	Class ^a	Level ^b
In all patients with suspected cardiogenic shock, immediate ECG and echocardiography are recommended.	I	C
All patients with cardiogenic shock should be rapidly transferred to a tertiary care center which has a 24/7 service of cardiac catheterization, and a dedicated ICU/CCU with availability of short-term mechanical circulatory support.	I	C
In patients with cardiogenic shock complicating ACS an immediate coronary angiography is recommended (within 2 hours from hospital admission) with an intent to perform coronary revascularization.	I	C
Continuous ECG and blood pressure monitoring are recommended.	I	C
Invasive monitoring with an arterial line is recommended.	I	C
Fluid challenge (saline or Ringer's lactate, >200 ml/15–30 min) is recommended as the first-line treatment if there is no sign of overt fluid overload.	I	C
Intravenous inotropic agents (dobutamine) may be considered to increase cardiac output.	IIb	C
Vasopressors (norepinephrine preferable over dopamine) may be considered if there is a need to maintain SBP in the presence of persistent hypoperfusion.	IIb	B
IABP is not routinely recommended in cardiogenic shock.	III	B
Short-term mechanical circulatory support may be considered in refractory cardiogenic shock depending on patient age, comorbidities and neurological function.	IIb	C



A. Mebazaa
H. Tolppanen
C. Mueller
J. Lassus
S. DiSomma
G. Baksyte
M. Cecconi
D. J. Choi
A. Cohen Solal
M. Christ
J. Masip
M. Arrigo
S. Nouria
D. Ojji
F. Peacock
M. Richards
N. Sato
K. Sliwa
J. Spinar
H. Thiele
M. B. Yilmaz
J. Januzzi

Acute heart failure and cardiogenic shock: a multidisciplinary practical guidance



AHF

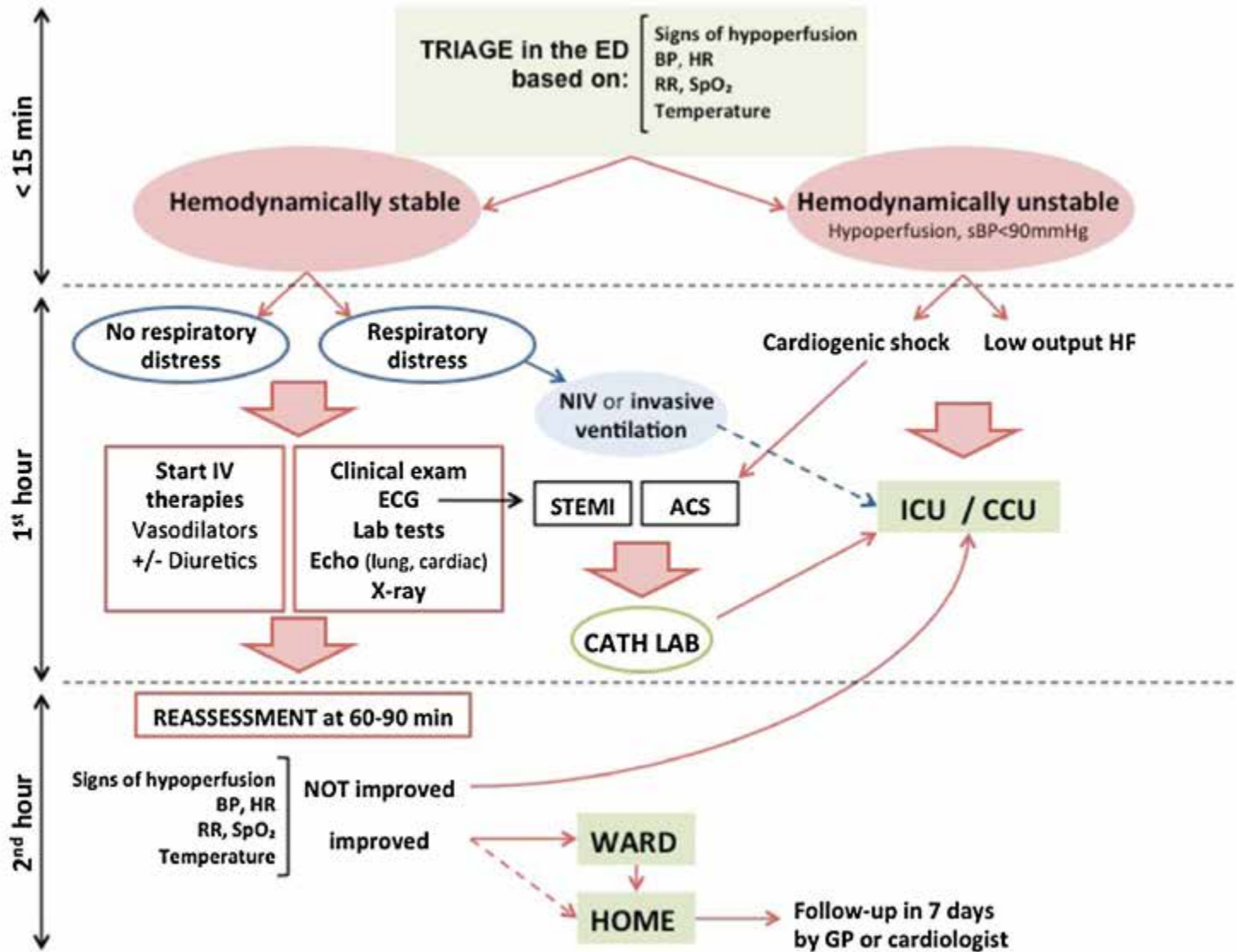


Table 12.6 Goals of treatment in acute heart failure

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: <ul style="list-style-type: none">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.

Multidisciplinary team management

- **Organization of care: Community - Hospital services
Rehabilitation - Palliative care**
- **Discharge planning**
- **Lifestyle advice**
- **Exercise training**

Structured follow-up:

- **Patient education**
- **Optimization of medical treatment**
- **Psychosocial support**
- **Improved access to care**
- **Older, frailty, cognitive impairment**
- **Palliative and end-of-life care**



Review

Practical approach on frail older patients attended for acute heart failure

Francisco J. Martín-Sánchez^{a, h, i, j, k, l, m, n, o, p, q, r, s, t, u, v, w, x, y, z}, Michael Christ^b, Óscar Miró^{c, u}, W. Frank Peacock^d, John J. McMurray^e, Héctor Bueno^{f, g, h}, Alan S. Maiselⁱ, Louise Cullen^{j, k, l}, Martin R. Cowie^m, Salvatore Di Sommaⁿ, Elke Platz^o, Josep Masip^{p, v}, Uwe Zeymer^d, Christiaan Vrints^f, Susanna Price^s, Christian Mueller^{t, w}

Clinical Practice/Education



European Society of Cardiology – Acute Cardiovascular Care Association position paper on safe discharge of acute heart failure patients from the emergency department

European Heart Journal: Acute Cardiovascular Care
1–10
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acc.sagepub.com

Óscar Miró¹, Frank W Peacock², John J McMurray³,
Héctor Bueno⁴, Michael Christ⁵, Alan S Maisel⁶,
Louise Cullen⁷, Martin R Cowie⁸, Salvatore Di Somma⁹,
Francisco J Martín Sánchez¹⁰, Elke Platz¹¹, Josep Masip¹²,
Uwe Zeymer¹³, Christiaan Vrints¹⁴, Susanna Price¹⁵,
Alexander Mebazaa¹⁶ and Christian Mueller¹⁷ for the
Acute Heart Failure Study Group of the ESC Acute
Cardiovascular Care Association

Gaps in knowledge

6. Acute heart failure

- Prospective evaluation of the 'time-to-treatment' concept in AHF
- Evaluation of whether inadequate phenotyping is responsible for the failure of treatments to improve outcome in AHF
- Better definition and treatment of diuretic resistance
- Role of nitrates in the management of AHF
- Treatments improving mortality and morbidity
- Strategies and therapies to prevent early rehospitalization after discharge for a hospital admission for AHF.

Punts forts de les guies de ICA

- **Pèptids natriurètics i troponina**
- **CHAMP approach**
- **Timing i indicació de l'ecocardiograma**
- **Estratègies per iniciar ràpidament el tractament**
- **Criteris d'ubicació de pacients (UCI, sala, Urgències)**
- **Trasllat de pacients en shock**
- **Valoració de l'assistència ventricular**
- **Enfoc multidisciplinar del maneig i implicació de diferents àmbits a l'alta**

Punts dèbils de les guies de ICA

- **Classificació basada en IC crònica avançada (Nohria) que no cobreix tots els supòsits (insuficiència respiratòria)**
- **Desaparició dels fenotips de prèvies guies (EAP, IVD, ICCD entre altres)**
- **No aborda la IC dreta**
- **Enfoc marcadament esbiaixat sota el prisma del cardiòleg (No d'Urgències)**
- **Recomana TSH a urgències**
- **No presenta punts de tall diagnòstics de ICA de BNP o NT-ProBNP**
- **No aborda la monitorització hemodinàmica per ECO**
- **No recomana la monitorització del CO / SvO2, lactat, catèter venós central i sonda urinària en el shock cardiogènic**
- **Poc èmfasi en l'ús de l'eco pulmonar**
- **Poc èmfasi en el possible efecte deleteri de l'adrenalina**

Table 12.2 Definitions of the terms used in Section 12 on acute heart failure

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 (<10.67 kPa) (blood gas analysis).
Hypoxaemic respiratory failure (type I)	PaO ₂ <60 mmHg (<8 kPa)
Hypercapnia	CO ₂ partial pressure (PaCO ₂) in arterial blood >45 mmHg (>6 kPa) (blood gas analysis).
Hypercapnic respiratory failure (type II)	PaCO ₂ >50 mmHg (>6.65 kPa).
Acidosis	pH <7.35
Elevated blood lactate	>2 mmol/L
Oliguria	Urine output <0.5 mL/kg/h

BP = blood pressure; bpm = beats per minute; PaCO₂ = partial pressure of carbon dioxide in arterial blood; PaO₂ = partial pressure of oxygen in arterial blood; SaO₂ = oxygen saturation.

Recommendations regarding diagnostic measurements in patients with suspected acute heart failure	Class^a	Level^b
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
Recommendations for the management of patients with acute heart failure – pharmacotherapy	Class^a	Level^b
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 a continuous infusion, and the dose and duration should be adjusted according to the patients' symptoms and clinical status.	I	B
Inotropic agents are not recommended unless the patient is symptomatically hypotensive or hypoperfused because of safety concern.	III	A
Recommendations regarding management of patients with cardiogenic shock	Class^a	Level^b
In all patients with suspected cardiogenic shock, immediate ECG and echocardiography are recommended.	I	C
All patients with cardiogenic shock should be rapidly transferred to a tertiary care centre which has a 24/7 service of cardiac catheterization, and a dedicated ICU/CCU with availability of short-term mechanical circulatory support.	I	C
Recommendations regarding oral evidence-based disease-modifying therapies in patients with acute heart failure	Class^a	Level^b
In case of worsening of chronic HFrEF, every attempt should be made to continue evidence-based, disease-modifying therapies, in the absence of haemodynamic instability or contra-indications.	I	C