Acute Pulmonary Edema

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BASED ON:

1. ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012

2. HFSA 2010 Recommendation

THE SCOPE OF THE PROBLEM:

PREVALENCE:
1–2% of the adult population in developed countries,
rising to ≥10% among persons $^{24}_{12}$70 years of age.

At least half of patients with HF have a low EF (HF-REF).
CAD is the cause of approximately two-thirds of cases of systolic HF, although hypertension and diabetes are contributing factors, LESS COMMON: previous viral infection, alcohol abuse, chemotherapy and 'idiopathic' dilated cardiomyopathy.

HF-PEF have a different epidemiological and etiological profile:
older pts. more often female and obese, less likely have CAD,
but more likely have hypertension and atrial fibrillation.
Patients with HF-PEF have a better prognosis.

ACUTE HEART FAILURE:

ACUTE HEART FAILURE (AHF) is the term used to describe the rapid onset, or change in symptoms and signs of HF.

It is a life-threatening condition that requires immediate medical attention and urgent hospital admission.

Heart failure is the leading cause of hospitalization in patients 65 years of age

In-hospital mortality is excessive and readmission is common, despite advances in pharmacotherapy and device therapy

AHF arises as a result of deterioration of a previously diagnosed HF

In AHF-REF most common triggers are arrhythmias or discontinuation of diuretic therapy

In AHF-PEF most common triggers are volume overload or severe hypertension

AHF may also be the first presentation of HF (*'de novo'* AHF)

The 'acuteness' may vary, days or weeks of deterioration, or developing within hours to minutes (depending of etiology).

Patients may present with a spectrum of conditions:

ranging from life-threatening pulmonary edema or cardiogenic shock.

CLASIFICATION OF ACUTE HEART FAILURE

CLINICAL STATUS	HR	SBP mmHg	CI L/min/m2	PCWP mmHg	Congestion Killip/Forrester	Diuresis	Hypo-perfusion	End-organ hypo-perfusion
I. Acute decompensate CHF	+/−	Low normal /High	Low normal /High	Mild elevation	K II/F II	+	+/−	−
II. Acute HF with hypertension	Usually increased	High	+/−	>18	K II-IV/FII-III	+/−	+/−	+, with CNS symptoms
III. Acute HF with pulmonary edema	+	Low normal	Low	Elevated	KIII/FII	+	+/−	−
IVa. Cardiogenic shock*/low output syndrome	+	Low normal	Low, <2.2	>16	K III-IV/F I-III	low	+	+
IVb. Severe cardiogenic shock	>90	<90	<1.8	>18	K IV/F IV	Very low	++	+
V. High output HF	+	+/−	+	+/−	KII/FI-II	+	−	−
VI Acute right heart failure	Usually low	Low	Low	Low	F I	+/−	+/−, acute onset	+/−

*Legend: *differentiation of low cardiac output syndrome is subjective; SBP=systolic blood pressure; CI=cardiac index; PCWP=pulmonary capillary wedge pressure; CNS*

Data from UC for Cardiology for 2012

ICU hospitalizations	3400	% of total	Mortality rate (%)
AHF	**390**	**11,5**	**9,1%**
AHF type	**Prevalence of AHF**	**%**	**Mortality rate**
I. Acute decompensate CHF	106	27,2	25 (23,6%)
II. Acute HF with hypertension	52	13,3	3 (5,8%)
III. Acute HF with pulmonary edema	169	43,3	17 (10,1%)
IVb. Severe cardiogenic shock	52	13,3	35 (67,3%)
VI. Acute right heart failure	11	2,8	3(27,3%)

PRECIPITANTS AND CAUSES OF ACUTE HEART FAILURE

Events leading to rapid deterioration

·Rapid arrhythmia or severe bradycardia/conduction disturbances
·Acute coronary syndrome
·Mechanical complication of acute coronary syndrome (rupture of IVS, mitral valve chordal rupture, right ventricular infarction)
·Acute pulmonary embolism
·Hypertensive crisis
·Aortic dissection
·Surgery and per operative problems
·Peripartum cardiomyopathy

Events leading to less rapid deterioration

·Infective endocarditis
·Anemia
·Kidney dysfunction
·Non-adherence to diet/drug therapy
·Iatrogenic causes (drug interactions)
·Arrhythmia/bradycardia/conduction disturbances
·Uncontrolled hypertension
·Hyperthyroidism or hypothyroidism
·Alcohol and drug abuse

ACUTE HF—DIAGNOSIS

- The diagnosis of ADHF should be based primarily on **signs and symptoms.**

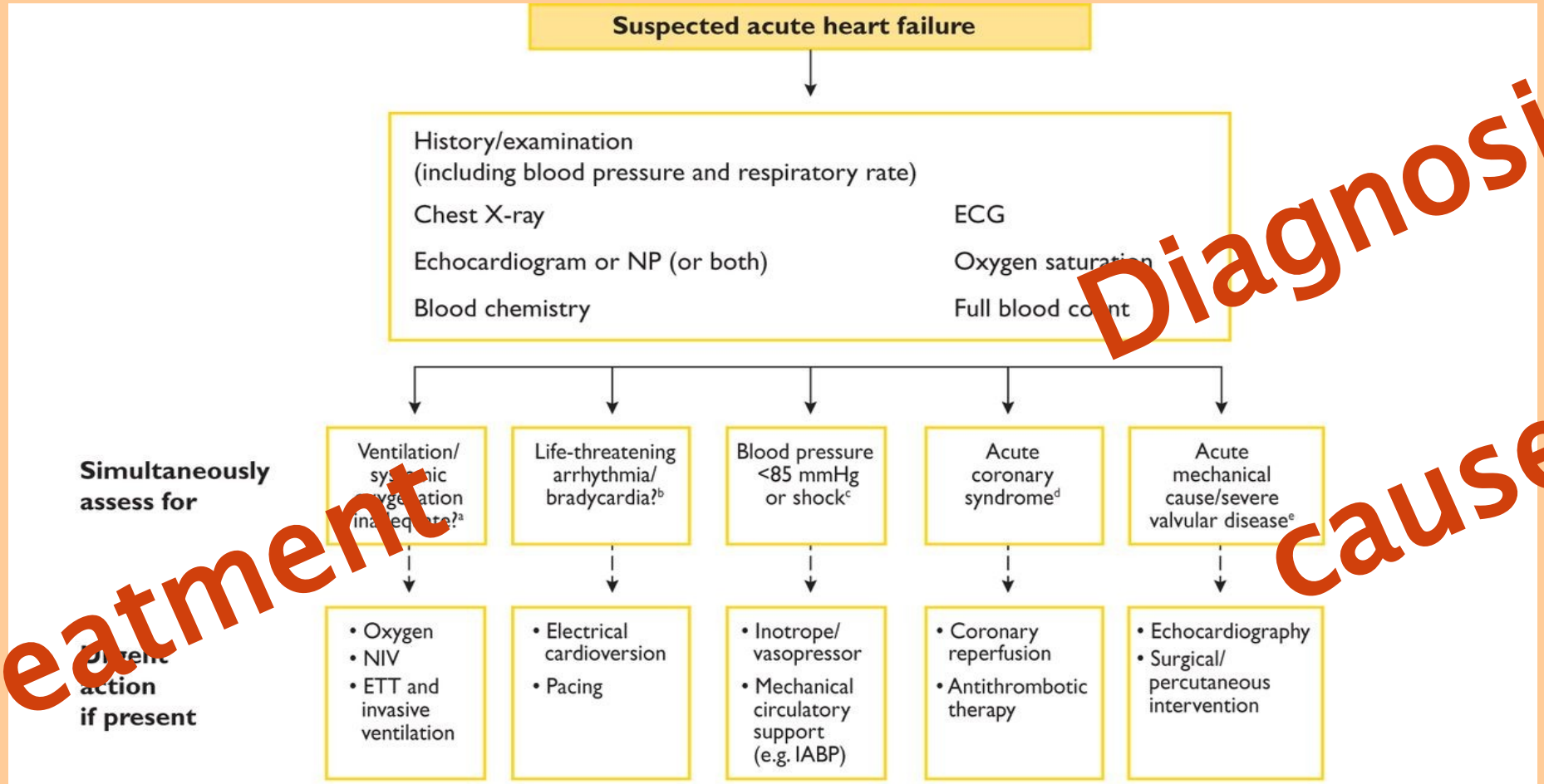
Strength of Evidence = C

- When the diagnosis is uncertain, determination of **BNP or NT-proBNP** concentration is recommended in patients being evaluated for dyspnea who have signs and symptoms compatible with HF.

Strength of Evidence = A

- The natriuretic peptide concentration should not be interpreted in isolation, but in the context of all available clinical data, and with the knowledge of cardiac and non-cardiac factors that can raise it's levels.

INITIAL ASSESSMENT AND MONITORING OF PATIENTS
Three parallel assessments are ongoing



ECG = electrocardiogram; ETT = endotracheal tube; IABP = intra-aortic balloon pump; NIV = non-invasive ventilation; NP = natriuretic peptide.
[a]For example, respiratory distress, confusion SpO_2 <90%, or PaO_2 <60 mmHg (8.0 kPa).
[b]For example, ventricular tachycardia, third-degree atrioventricular block.
[c]Reduced peripheral and vital organ perfusion—patients often have cold skin and urine output ≤15 ml/h and/or disturbance of consciousness.
[d]Percutaneous coronary revascularization (or thrombolysis) indicated if ST-segment elevation or new left bundle branch block.
[e]Vasodilators should be used with great caution, and surgery should be considered for certain acute mechanical complications (e.g. inter-ventricular septal rupture, mitral valve papillary muscle rupture).

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Diagnosis

cause

reatment

ACUTE HF—Hospital Admission

- **Hospital admission** <u>is recommended</u> **when:**
  - **Evidence of severely decompensated HF, including:**
    - Hypotension
    - Worsening renal failure
    - Altered mentation
  - **Dyspnea at rest**
    - Typically reflected by resting tachypnea
    - Less commonly reflected by oxygen saturation < 90%
  - **Hemodynamically significant arrhythmia**
    - Including new onset of rapid atrial fibrillation
  - **Acute coronary syndromes**

Strength of Evidence = C

- **Hospital admission** <u>should be considered</u> **when:**
  - **Worsened congestion**
  - **Signs and symptoms of pulmonary or systemic congestion**
  - **Major electrolyte disturbance**
  - **Associated co morbid conditions**
    - **Pneumonia, pulmonary embolus, diabetic ketoacidosis, symptoms suggestive of TIA or stroke**
  - **Repeated ICD firings**
  - **Previously undiagnosed HF with signs and symptoms of systemic or pulmonary congestion**

Strength of Evidence = C

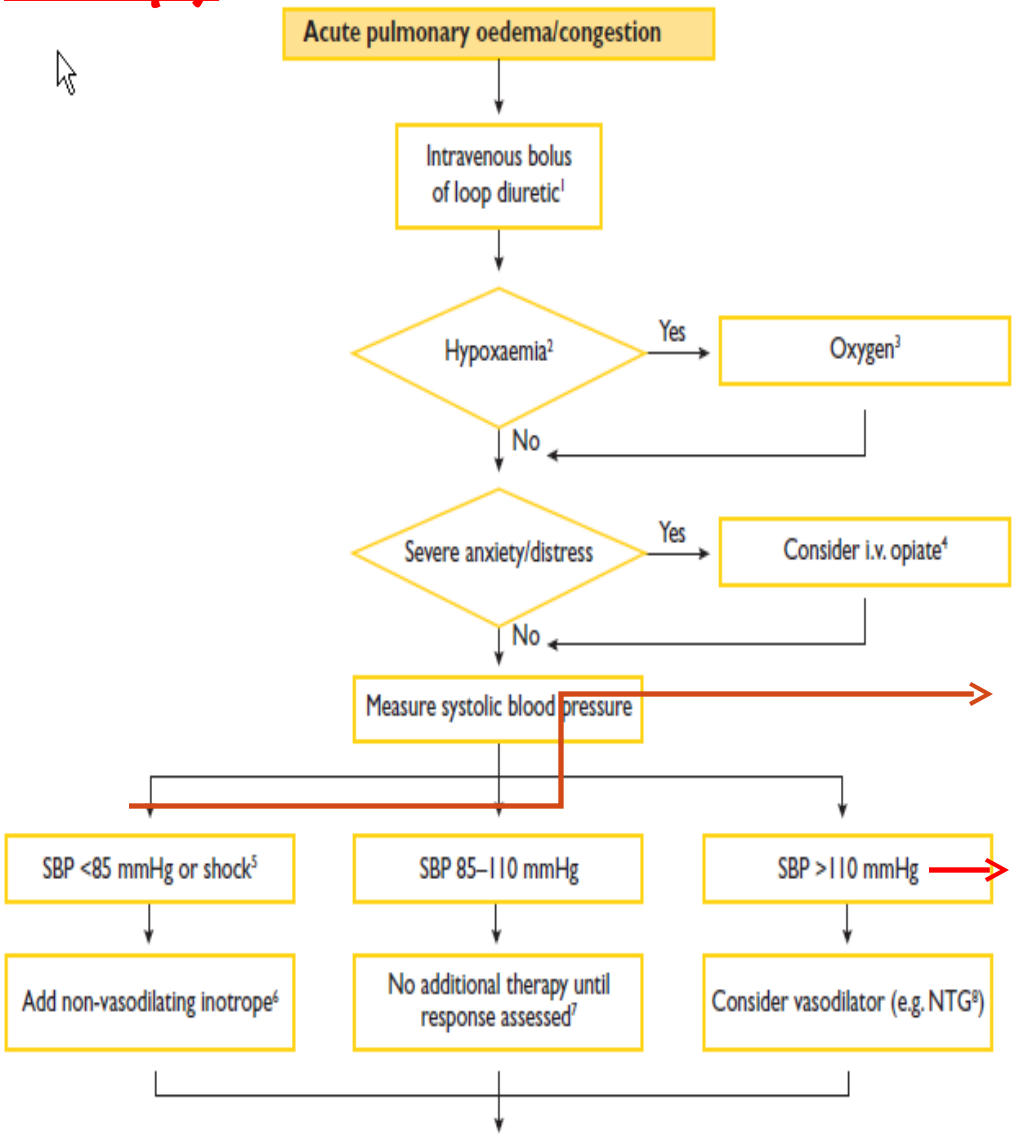
ACUTE HF—TREATMENT GOALS *Strength of Evidence = C*

Immediate (ED/ICU/CCU)
• Treat symptoms
• Restore oxygenation
• Improve haemodynamics and organ perfusion
• Limit cardiac and renal damage
• Prevent thrombo-embolism
• Minimize ICU length of stay
Intermediate (in hospital)
• Stabilize patient and optimize treatment strategy
• Initiate and up-titrate disease-modifying pharmacological therapy
• Consider device therapy in appropriate patients
• Identify aetiology and relevant co-morbidities
Pre-discharge and long-term management
• Plan follow-up strategy
• Enrol in disease management programme, educate, and initiate appropriate lifestyle adjustments
• Plan to up-titrate/optimize dose of disease-modifying drugs
• Ensure assessed for appropriate device therapy
• Prevent early readmission
• Improve symptoms, quality of life, and survival

- Improve symptoms, especially congestion and low output symptoms
- Restore normal oxygenation
- Optimize volume status
- Identify etiology
- Identify and address precipitating factors
- Optimize chronic oral therapy
- Minimize side effects
- Identify patients who might benefit from revascularization or device therapy
- Identify risk of thromboembolism and need for anticoagulant therapy
- Educate patients concerning medications and self assessment

TREATMENT OF ACUTE HEART FAILURE

remains largely opinion-based with little good evidence to guide therapy



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INITIAL THERAPY:

1. DIURETIC therapy
2. OXYGEN therapy

Routine administration of supplemental oxygen:

·Is recommended in the presence of hypoxia *saturation <90% or PAo2 <8,0 kPa*

·Is not recommended in the absence of hypoxia

Strength of Evidence = C

(NEW in 2010)

Use of non-invasive positive pressure ventilation may be considered for severely dyspnoic patients with clinical evidence of pulmonary edema.

Strength of Evidence = C

3. MORPHINE - 4-8 mg of plus 10 mg metoclopramide, repeat as needed

DEPENDING ON THE RESPONSE:

Start an i.v. infusion of DOBUTAMINE 2.5 µg/kg/min, doubling dose every 15 min according to response or tolerability. A dose >20 µg/kg/min is rarely needed.

Start i.v. infusion of NTG 10 ≒g/min and doubled every 10 min depending on response and tolerability. Dose> 100 ≒g/min is rarely necessary

[14][7] APPROPRIATE RESPONSE: reducing breathlessness, adequate diuresis (>100 ml/hour urine within the first 2 hours), increased oxygen saturation, improved organ perfusion, reduced HR and respiratory frequency after 1-2 hours of treatment.

ACUTE HF—Fluid Overload and Diuretics

Doses of diuretics commonly used to treat heart failure (HF-REF/ HF-PEF / acute / chronic)

Diuretics	Initial dose (mg)		Usual daily dose (mg)	
Loop diuretics[a]				
Furosemide	20–40		40–240	
Bumetanide	0.5–1.0		1–5	
Torasemide	5–10		10–20	
Thiazides[b]				
Bendroflumethiazide	2.5		2.5–10	
Hydrochlorothiazide	25		12.5–100	
Metolazone	2.5		2.5–10	
Indapamide[c]	2.5		2.5–5	
Potassium-sparing diuretics[d]				
	+ACEi/ ARB	−ACEi/ ARB	+ACEi/ ARB	−ACEi/ ARB
Spironolactone/ eplerenone	12.5–25	50	50	100–200
Amiloride	2.5	5	5–10	10–20
Triamterene	25	50	100	200

Initial treatment with LOOP DIURETICS—given i.v. is recommended *(Strength of Evidence=B):*

- At doses needed to produce diuresis sufficient to achieve optimal volume status with relief of signs and symptoms of congestion (edema, elevated JVP, dyspnea), without inducing an excessively rapid reduction in:
  - intravascular volume (which may result in symptomatic hypotension and/or worsening renal function), or
  - serum electrolytes (which may precipitate arrhythmias or muscle cramps)
- In patients already on DTh-2,5 higher dose is recommended *(Strength of Evidence=C)*

MONITORING:

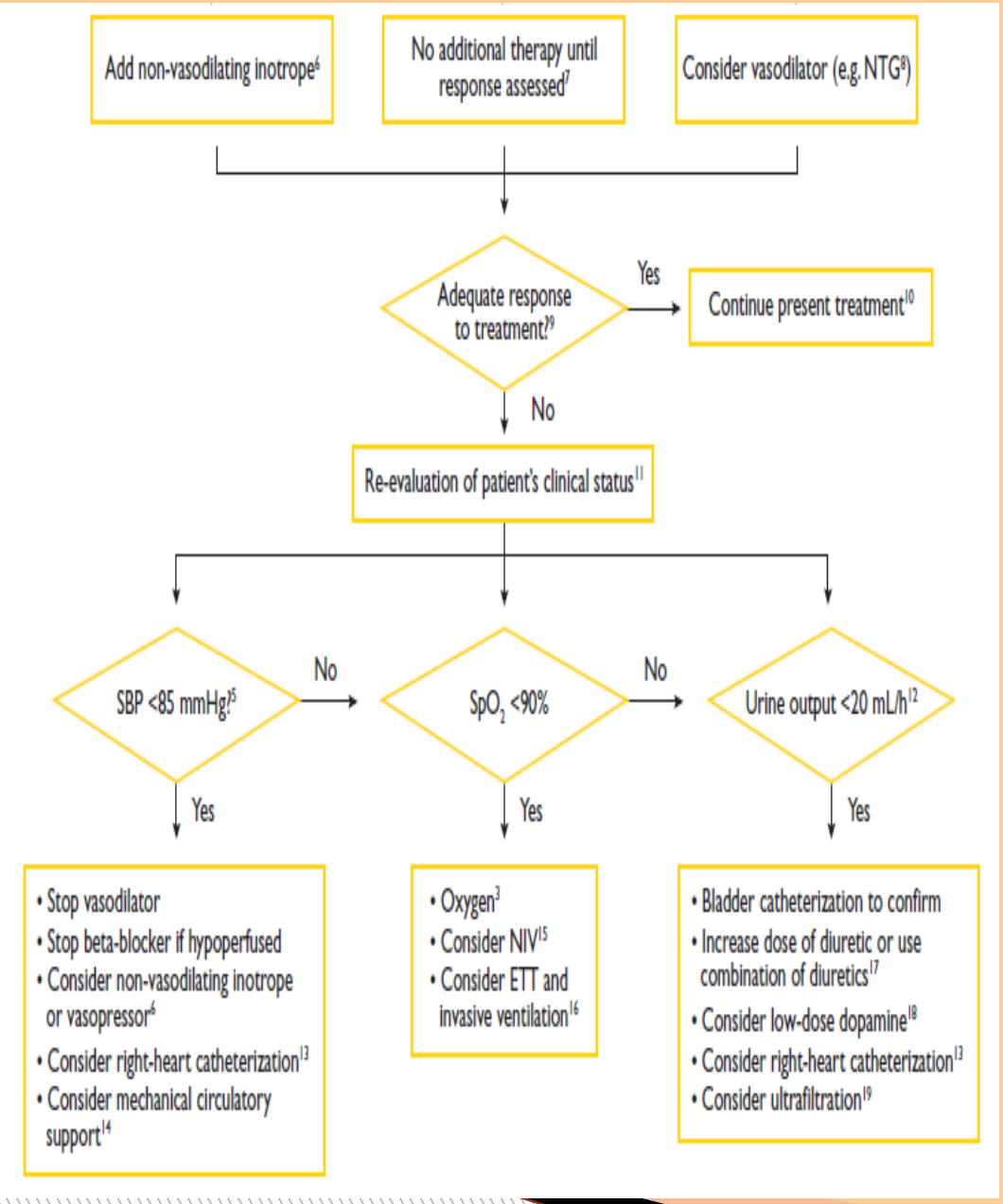
- Observation for development of a side effects (renal dysfunction, electrolyte abnormalities, symptomatic hypotension)
- Monitoring of serum potassium and magnesium levels at least daily and maintained in the normal range.
- Observation for the development of renal dysfunction *(patients with moderate to severe renal dysfunction and evidence of fluid retention should continue to be treated with diuretics, because in the presence of severe fluid overload, renal dysfunction may improve with diuresis).*

B Thyazides are not recommended if estimated GFR is <30 mL/min, except when prescribed synergistically with loop diuretics.

C Indapamide is a non-thiazide sulfonamide.

D A mineralocorticoid antagonist (MRA) spironolactone/eplerenone is always preferred. Amiloride and triamterene should not be combined with an MRA.

TREATMENT OF ACUTE HEART FAILURE



DEPENDING ON THE RESPONSE:

[14][7] Inappropriate response: <100 ml/h diuresis more than 1-2 hours is inadequate initial response IV diuretic

[14][7] In patients with:
persistently low blood pressure / shock, think about alternative diagnoses, pulmonary arterial catheterization in order to identify inappropriate left ventricular filling pressure and adjustment of vasoactive therapy

Absence/low diuresis: *Strength of Evidence = C*

1. Doubling the dose of loop diuretic furosemide 500 mg equivalent (doses of 250 mg and higher should be given in the form of infusion over 4 h)
2. In the absence of a response start IV infusion of dopamine 2,5 ≒g/kg/min. Higher doses are not recommended to enhance diuresis
3. In the absence of adequate diuresis in a patient who remains in pulmonary edema, veno-venous ultrafiltration

Other alternatives: *Strength of Evidence = C*
Continuous infusion of a loop diuretic
Addition of a second type of diuretic orally (metolazone or spironolactone) or intravenously (chlorothiazide)
Restricting sodium and fluid

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INOTROPS in the treatment of ACUTE HEART FAILURE

Strength of Evidence = C

	Bolus	Infusion rate
Dobutamine	No	2–20 µg/kg/min (β+)
Dopamine	No	<3 µg/kg/min: renal effect (δ+)
		3–5 µg/kg/min; inotropic (β+)
		>5 µg/kg/min: (β+), vasopressor (α+)
Milrinone	25–75 µg/kg over 10–20 min	0.375–0.75 µg/kg/min
Enoximone	0.5–1.0 mg/kg over 5–10 min	5–20 µg/kg/min
Levosimedan[a]	12 µg/kg over 10 min (optional)[b]	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

Intravenous inotropes (milrinone or dobutamine) may be considered to relieve symptoms and improve end-organ function in patients with:

- Reduced LVEF
- And diminished peripheral perfusion or end-organ dysfunction (low output syndrome)

Particularly if these patients:

- Have systolic blood pressure <90 mmHg,
- Have symptomatic hypotension despite adequate filling pressure,

Intravenous inotropes (milrinone or dobutamine) are not recommended unless left heart filling pressures are known to be elevated or cardiac index is severely impaired based on:

- direct measurement or
- clear clinical signs.

It is recommended that administration of intravenous inotropes (milrinone or dobutamine) be accompanied by continuous or frequent blood pressure monitoring and continuous monitoring of cardiac rhythm.

If symptomatic hypotension or worsening tachyarrhythmia develop during administration of these agents, discontinuation or dose reduction should be considered.

VASODILATORS in the treatment of ACUTE HEART FAILURE

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
Nesiritide[a]	Bolus 2 μg/kg + infusion 0.01 μg/kg/min	Hypotension	

In the absence of symptomatic hypotension, i.v. nitroglycerin, nitroprusside or nesiritide **may be considered** as an addition to diuretic therapy for rapid improvement of congestive symptoms in patients admitted with ADHF.

Strength of Evidence = B

IV vasodilators (intravenous nitroglycerin or nitroprusside) and diuretics are recommended for rapid symptom relief in patients with acute pulmonary edema or severe hypertension. *Strength of Evidence=C*

Intravenous vasodilators **may be considered** in patients with ADHF who have persistent severe HF despite aggressive treatment with diuretics and standard oral therapies.

- Nitroprusside *Strength of Evidence=B*
- Nitroglycerine, nesiritide *Strength of Evidence=C*

MONITORING:

- Frequent blood pressure monitoring **is recommended** *Strength of Evidence = B*
- They should be decreased in dosage or discontinued if symptomatic hypotension or worsening renal function develops

ACUTE HF— Patient Monitoring *(Strength of Evidence = C)*

Frequency	Value	Specifics
At least daily	Weight	Determine after voiding in the morning Account for possible increased food intake due to improved appetite
At least daily	Fluid intake and output	Monitoring of daily weights, intake, and output is recommended to assess clinical efficacy of diuretic therapy. Routine use of a Foley catheter is not recommended for monitoring volume status. However, placement of a catheter is recommended when close monitoring of urine output is needed or if a bladder outlet obstruction is suspected of contributing to worsening renal function.
More than daily	Vital signs	Orthostatic blood pressure, if indicated Oxygen saturation daily until stable
At least daily	Signs	Edema, ascites, pulmonary rales, hepatomegaly, increased jugular venous pressure, hepatojugular reflux, liver tenderness
At least daily	Symptoms	Orthopnea, paroxysmal nocturnal dyspnea or cough, nocturnal cough, dyspnea, fatigue, lightheadedness
At least daily	Electrolytes	Potassium, sodium
At least daily	Renal function	BUN, serum creatinine

ACUTE HF—Hemodynamic Monitoring

- The routine use of invasive hemodynamic monitoring in patients with ADHF is not recommended.

Strength of Evidence = A

- Invasive hemodynamic monitoring should be considered in a patient:
  - Who is refractory to initial therapy
  - Whose volume status and cardiac filling pressures are unclear
  - Who has clinically significant hypotension (typically SBP < 80 mm Hg) or worsening renal function during therapy
  - Or who is being considered for cardiac transplant and needs assessment of degree and reversibility of pulmonary hypertension
  - Or in whom documentation of an adequate hemodynamic response to the inotropic agent is necessary when chronic outpatient infusion is being considered

Strength of Evidence = C

ACUTE HF—VT Prophylaxis

(NEW in 2010)

- Venous thromboembolism prophylaxis with low dose unfractionated heparin, low molecular weight heparin or fondaparinux to prevent proximal deep venous thrombosis and pulmonary embolism is recommended for patients who are admitted to hospital with ADHF and who are not already anticoagulated and have no contraindication to anticoagulation.

Strength of Evidence = B

- Venous thromboembolism prophylaxis with a mechanical device (intermittent pneumatic compression devices or graded compression stockings) to prevent proximal deep venous thrombosis and pulmonary embolism should be considered for patients who are admitted to the hospital with ADHF, who are not already anticoagulated, and who have a contraindication to anticoagulation.

ACUTE HF—Fluid / Sodium Restriction

- FLUID RESTRICTION (<2 L/daily):
  - Is recommended in patients with moderate hyponatremia (serum sodium < 130 mEq/L)
  - Should be considered to assist in treatment of fluid overload in other patients.

  Strength of Evidence = C
- In patients with severe (serum sodium < 125 mEq/L) or worsening hyponatremia, stricter fluid restriction may be considered.

  Strength of Evidence = C
- SODIUM RESTRICTION
- A low sodium diet (2 g daily) is recommended for most hospitalized patients.

  Strength of Evidence = C
- In patients with recurrent or refractory volume overload, stricter sodium restriction may be considered. *Strength of Evidence = C*

TREATMENT OF ACUTE HEART FAILURE
Patirnts with pulmonary congestion without shock

Patients with pulmonary congestion/oedema without shock		
An i.v. loop diuretic is recommended to improve breathlessness and relieve congestion. Symptoms, urine output, renal function, and electrolytes should be monitored regularly during use of i.v. diuretic.	I	B
High-flow oxygen is recommended in patients with a capillary oxygen saturation <90% or PaO_2 <60 mmHg (8.0 kPa) to correct hypoxaemia.	I	C
Thrombo-embolism prophylaxis (e.g. with LMWH) is recommended in patients not already anticoagulated and with no contraindication to anticoagulation, to reduce the risk of deep venous thrombosis and pulmonary embolism.	I	A
Non-invasive ventilation (e.g. CPAP) should be considered in dyspnoeic patients with pulmonary oedema and a respiratory rate >20 breaths/min to improve breathlessness and reduce hypercapnia and acidosis. Non-invasive ventilation can reduce blood pressure and should not generally be used in patients with a systolic blood pressure <85 mmHg (and blood pressure should be monitored regularly when this treatment is used).	IIa	B
An i.v. opiate (along with an antiemetic) should be considered in particularly anxious, restless, or distressed patients to relieve these symptoms and improve breathlessness. Alertness and ventilatory effort should be monitored frequently after administration because opiates can depress respiration.	IIa	C
An i.v. infusion of a nitrate should be considered in patients with pulmonary congestion/oedema and a systolic blood pressure >110 mmHg, who do not have severe mitral or aortic stenosis, to reduce pulmonary capillary wedge pressure and systemic vascular resistance. Nitrates may also relieve dyspnoea and congestion. Symptoms and blood pressure should be monitored frequently during administration of i.v. nitrates.	IIa	B
An i.v. infusion of sodium nitroprusside may be considered in patients with pulmonary congestion/oedema and a systolic blood pressure >110 mmHg, who do not have severe mitral or aortic stenosis, to reduce pulmonary capillary wedge pressure and systemic vascular resistance. Caution is recommended in patients with acute myocardial infarction. Nitroprusside may also relieve dyspnoea and congestion. Symptoms and blood pressure should be monitored frequently during administration of i.v. nitroprusside.	IIb	B
Inotropic agents are NOT recommended unless the patient is hypotensive (systolic blood pressure <85 mmHg), hypoperfused, or shocked because of safety concerns (atrial and ventricular arrhythmias, myocardial ischaemia, and death).	III	C

TREATMENT OF ACUTE HEART FAILURE
Patients with hypotension, hypoperfusion or shock

Patients with hypotension, hypoperfusion or shock		
Electrical cardioversion is recommended if an atrial or ventricular arrhythmia is thought to be contributing to the patient's haemodynamic compromise in order to restore sinus rhythm and improve the patient's clinical condition.	I	C
An i.v. infusion of an inotrope (e.g. dobutamine) should be considered in patients with hypotension (systolic blood pressure <85 mmHg) and/or hypoperfusion to increase cardiac output, increase blood pressure, and improve peripheral perfusion. The ECG should be monitored continuously because inotropic agents can cause arrhythmias and myocardial ischaemia.	IIa	C
Short-term mechanical circulatory support should be considered (as a 'bridge to recovery') in patients remaining severely hypoperfused despite inotropic therapy and with a potentially reversible cause (e.g. viral myocarditis) or a potentially surgically correctable cause (e.g. acute interventricular septal rupture).	IIa	C
An i.v. infusion of levosimendan (or a phosphodiesterase inhibitor) may be considered to reverse the effect of beta-blockade if beta-blockade is thought to be contributing to hypoperfusion. The ECG should be monitored continuously because inotropic agents can cause arrhythmias and myocardial ischaemia, and, as these agents are also vasodilators, blood pressure should be monitored carefully.	IIb	C
A vasopressor (e.g. dopamine or norepinephrine) may be considered in patients who have cardiogenic shock, despite treatment with an inotrope, to increase blood pressure and vital organ perfusion. The ECG should be monitored as these agents can cause arrhythmias and/or myocardial ischaemia. Intra-arterial blood pressure measurement should be considered.	IIb	C
Short-term mechanical circulatory support may be considered (as a 'bridge to decision') in patients deteriorating rapidly before a full diagnostic and clinical evaluation can be made.	IIb	C

TREATMENT OF ACUTE HEART FAILURE
Patients with ACS

Patients with an ACS		
Immediate primary PCI (or CABG in selected cases) is recommended if there is an ST elevation or a new LBBB ACS in order to reduce the extent of myocyte necrosis and reduce the risk of premature death.	I	A
Alternative to PCI or CABG: *Intravenous thrombolytic therapy is recommended, if PCI/CABG cannot be performed, if there is ST-segment elevation or new LBBB, to reduce the extent of myocyte necrosis and reduce the risk of premature death.*	I	A
Early PCI (or CABG in selected patients) is recommended if there is non-ST elevation ACS in order to reduce the risk of recurrent ACS. Urgent revascularization is recommended if the patient is haemodynamically unstable.	I	A
Eplerenone is recommended to reduce the risk of death and subsequent cardiovascular hospitalization in patients with an EF ≤40%.	I	B
An ACE inhibitor (or ARB) is recommended in patients with an EF ≤40%, after stabilization, to reduce the risk of death, recurrent myocardial infarction, and hospitalization for HF.	I	A
A beta-blocker is recommended in patients with an EF ≤40%, after stabilization, to reduce the risk of death and recurrent myocardial infarction.	I	B
An i.v. opiate (along with an antiemetic) should be considered in patients with ischaemic chest pain to relieve this symptom (and improve breathlessness). Alertness and ventilatory effort should be monitored frequently after administration because opiates can depress respiration.	IIa	C

TREATMENT OF ACUTE HEART FAILURE
Patients with rapid ventricular rate/ bradycardia or heart block

Patients with AF and a rapid ventricular rate		
Patients should be fully anticoagulated (e.g. with i.v. heparin), if not already anticoagulated and with no contraindication to anticoagulation, as soon as AF is detected to reduce the risk of systemic arterial embolism and stroke.	I	A
Electrical cardioversion is recommended in patients haemodynamically compromised by AF and in whom urgent restoration of sinus rhythm is required to improve the patient's clinical condition rapidly.	I	C
Electrical cardioversion or pharmacological cardioversion with amiodarone should be considered in patients when a decision is made to restore sinus rhythm non-urgently ('rhythm control' strategy). This strategy should only be employed in patients with a first episode of AF of <48 h duration (or in patients with no evidence of left atrial appendage thrombus on TOE).	I	C
Intravenous administration of a cardiac glycoside should be considered for rapid control of the ventricular rate.	I	C
Dronedarone is not recommended because of safety concerns (increased risk of hospital admission for cardiovascular causes and an increased risk of premature death), particularly in patients with an EF ≤40%.	III	A
Class I antiarrhythmic agents are not recommended because of safety concerns (increased risk of premature death), particularly in patients with LV systolic dysfunction.	III	A
Patients with severe bradycardia or heart block		
Pacing is recommended in patients haemodynamically compromised by severe bradycardia or heart block to improve the patient's clinical condition.	I	C

ACUTE HF— Discharge Criteria

Recommended for <u>all</u> HF patients *Strength of Evidence = C*	•Exacerbating factors addressed •Near optimal volume status achieved •Transition from intravenous to oral diuretics successfully completed •Patient and family education completed, including clear discharge instructions •Near optimal pharmacologic therapy achieved, including ACEI and BB (for patients with reduced LVEF) or intolerance documented •Follow-up clinic visit scheduled, usually for 7-10 days
Should be considered for patients with <u>advanced HF or recurrent admissions for HF</u> *Strength of Evidence = C*	Oral medication regimen stable for 24 hours No intravenous vasodilator or inotropic agent for 24 hours Ambulation prior to discharge to assess functional capacity after therapy Plans for post-discharge management (scale present in home, visiting nurse or telephone follow up generally no longer than 3 days after discharge) Referral for disease management, if available

ACUTE HF—Discharge Planning, Patient education

- It **is recommended** that every effort be made to utilize the hospital stay for assessment and improvement of patient adherence via patient and family education and social support services.

  Strength of Evidence = B

- DISCHARGE PLANNING **is recommended** as part of the management of patients with ADHF. Discharge planning should address the following issues:
  - Details regarding medication, dietary sodium restriction and recommended activity level
  - Follow-up by phone or clinic visit early after discharge to reassess volume status
  - Medication and dietary compliance
  - Alcohol moderation and smoking cessation
  - Monitoring of body weight, electrolytes and renal function
  - Consideration of referral for formal disease management

  Strength of Evidence = C

THANK YOU

QUESTIONS/ DISSCUSION