Acute Cardiovascular Care Association
Clinical Decision-Making
Toolkit

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A Registered Branch of the ESC

EUROPEAN SOCIETY OF CARDIOLOGY®

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The Acute Cardiovascular Care Association Clinical Decision-Making Toolkit

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Preface

Acute cardiovascular care has become very complex over the past years. Every professional involved faces challenges in the diagnosis, risk stratification and treatment of these patients. Many times critical decisions have to be made in very short periods of time, often in difficult clinical environments with limited resources.

The optimal management of patients with acute CV conditions requires a deep understanding of the CV anatomy and physiology, an important clinical training, advanced skills in a variety of diagnostic and therapeutic techniques, and a good knowledge of the functioning and resources provided by the local healthcare system.

In spite of these difficulties, an important part of acute CV care is initially delivered by non-experts. The Toolkit has been designed to provide guidance for rapid clinical decision-making to the non-experts involved in the initial management of patients with acute CV conditions as well as to the future experts, currently in training.

We decided to design the Toolkit as simply as possible, based mostly on algorithms and tables, easy to use in the usual environments where initial acute cardiovascular care is provided (ambulances, ER, CCUs, ICUs…). The Toolkit is an instrument to help make, accurately, the first decisions when managing patients presenting with the main CV symptoms or acute CV syndromes. Its content is based either on the latest clinical practice guidelines or the clinical experience of a number of European experts in each field when guidelines are not available. The Toolkit does not replace textbooks and other sources of information that need to be consulted to reach an optimal management of these patients.

All the effort put in by all authors and persons involved in the development of the Toolkit will be worthwhile if it means that one single additional patient with an acute CV syndrome survives or has a better outcome in Europe.

Héctor Bueno, MD, PhD, FESC
August 2013

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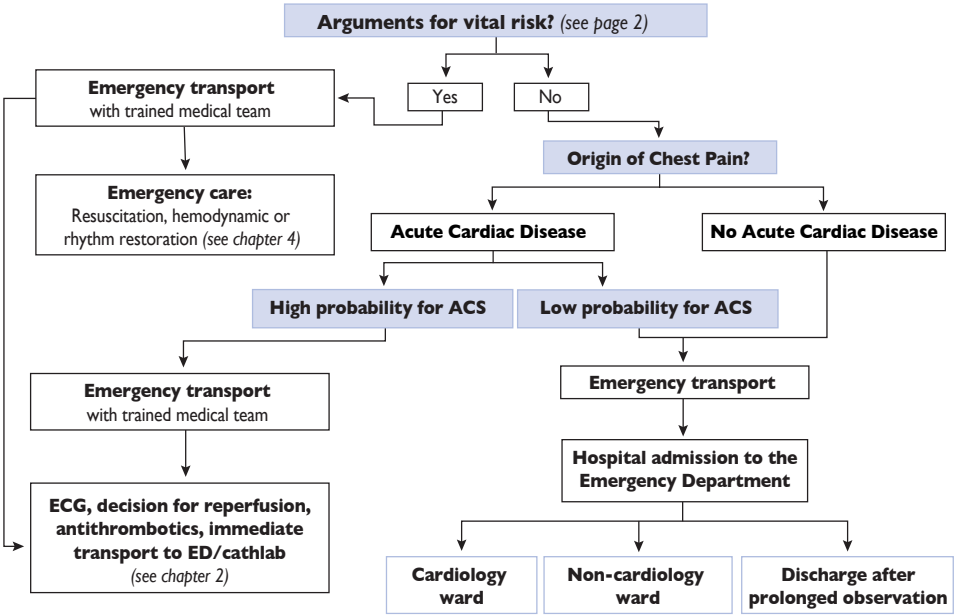
Chapter 1
KEY SYMPTOMS

FACTORS TO BE CONSIDERED IN THE EVALUATION AFTER THE FIRST CALL FOR CHEST PAIN

FIRST CALL FOR CHEST PAIN	Higher risk / probability	Lower risk / probability
Arguments for vital risk	• Cardiorespiratory arrest, syncope / loss of consciousness, neurological defect • Dyspnea • Nausea – vomiting • Arrhythmias – tachycardia	• Normal consciousness • Normal breathing *(see page 7)* • Normal heart rhythm
Context, CV risk	Age >40 years, previous CV disease (MI, stroke, PE), modifiable CV risk factors (smoker, HTN, hypercholesterolemia, diabetes), chronic CV treatment	• Age <40 years, • No previous CV disease • No CV risk factors • No chronic treatment
Chest Pain	Medial / lateral thoracic pain, intense, with dyspnea	• Depends on position / palpation / movements • Variable intensity, short duration (<1 min.) • Hyperthermia
Cardiac Ischemic Pain	Retro-sternal, constriction, jaw/cervical/arm/back irradiation, spontaneous, prolonged >20 min. + dyspnea, sweating, lightheadedness, nausea	• Lateral, abdominal irradiation • No neuro-vegetative symptoms

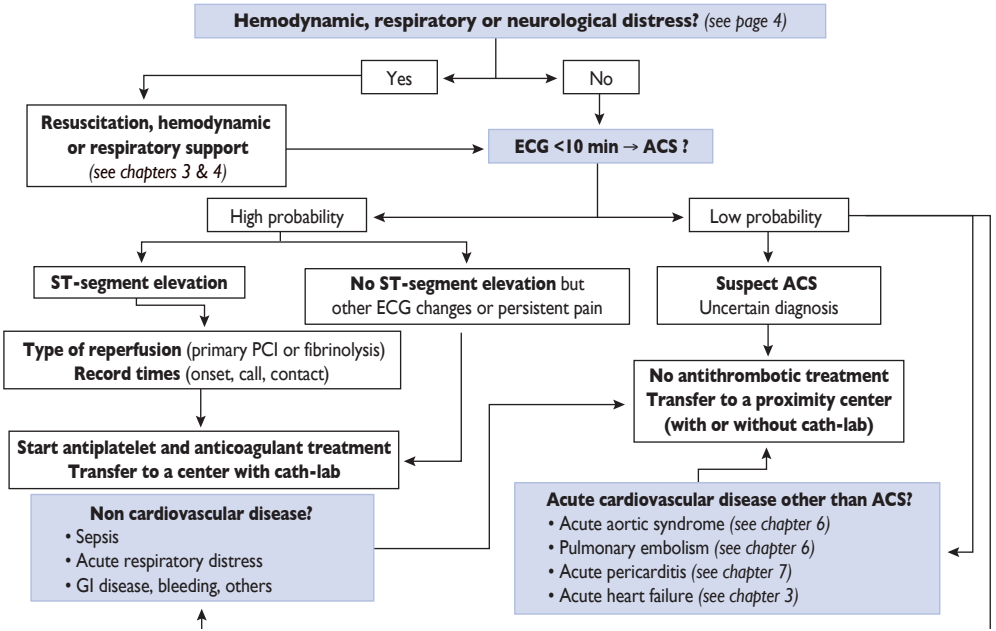
APPROACH AFTER FIRST CALL FOR OUT-OF-HOSPITAL CHEST PAIN



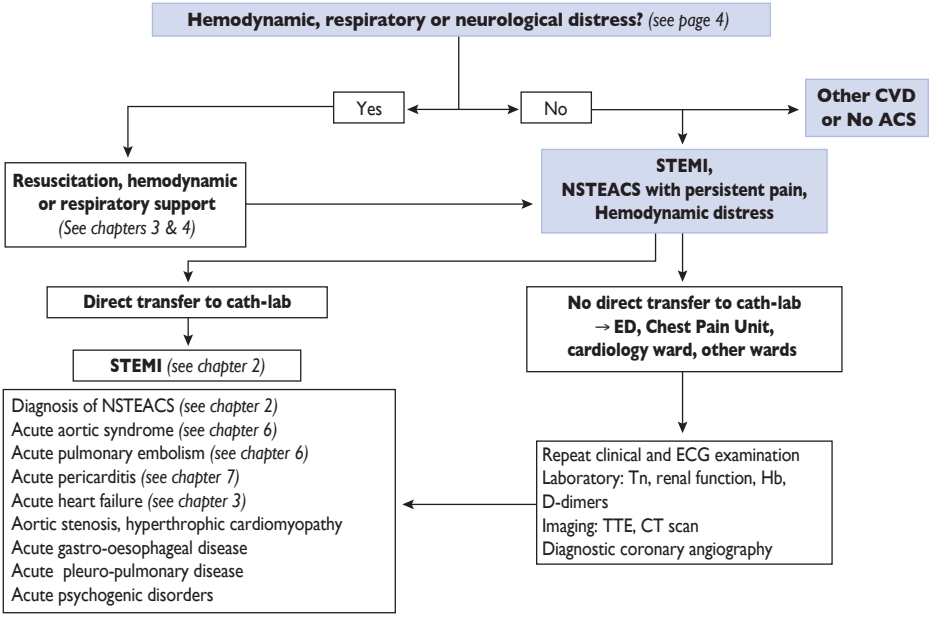
FACTORS TO BE CONSIDERED IN THE EVALUATION DURING THE FIRST MEDICAL CONTACT FOR CHEST PAIN

FIRST MEDICAL CONTACT	Higher risk / probability	Lower risk / probability
Hemodynamic, respiratory, neurological distress	• Cardiopulmonary arrest, hypotension, tachycardia, shock • Dyspnea, hypoxemia, lung rales (Killip class >2) • ECG: ST segment deviation	• Normal consciousness, no motion defects • Normal HR and BP • Normal breathing and SpO_2, no loss of pulse
Probability for ACS	• Context, typical symptoms consistent with myocardial ischemia • ECG changes • Bedside Tn	• No CV risk, atypical symptoms, normal ECG • Negative bedside Tn only if onset of pain >6 hours *(see page 22)*
STEMI **NSTEACS** **Uncertain diagnosis** *(see page 22)*	• ECG criteria for STEMI (see page 33) • ST depression or normal ECG • Normal ECG → Repeat 12-lead ECG recording	• Other ST-segment abnormalities not related to STEMI *(see page 24)*
Type of reperfusion **Time assessment**	• **Primary PCI or thrombolysis?** Primary PCI if delay <120 (preferably <90) min or <60 min if onset of pain <120 min. Consider age, anterior wall location • **Times:** Onset of pain, call, first medical contact, ECG, door, balloon inflation or needle (lytic drug) administration	• No reperfusion if delay >12 h, no symptoms, no ST-segment elevation

FIRST MEDICAL CONTACT IN PATIENTS WITH CHEST PAIN (HOME-AMBULANCE)



MANAGEMENT OF PATIENTS WITH CHEST PAIN (EMERGENCY ROOM)



DYSPNEA: DIFERENTIAL DIAGNOSIS

50% have ≥2 diagnoses, which may result in acute respiratory failure*!

Basic measures

- ❒ BP, HR, respiratory rate, SpO_2 & temperature
- ❒ Start oxygen to target SpO_2 94-98%
- ❒ Start i.v. line & monitor patient

Criteria for transfer to ICU
(despite treatment for 30 minutes)

- ❒ Respiratory rate >35/min
- ❒ SpO_2 <85%
- ❒ SBP <90 mmHg
- ❒ HR >120 bpm

Investigations:
- ❒ **ECG**
- ❒ **Chest X-ray**
- ❒ **Blood count**
- ❒ **Tn**
- ❒ **BNP**
- ❒ **ABG**
- ❒ **D-dimers if suspicion of PE**

Acute heart failure

Acute coronary syndrome

Pneumonia

Exacerbated COPD or other chronic lung disease

Pulmonary embolism

Other causes, including
- ❒ Asthma
- ❒ Severe sepsis
- ❒ Tumor
- ❒ Pneumothorax
- ❒ Pleural effusion/ascites
- ❒ Anxiety disorder
- ❒ Anemia
- ❒ Bronchitis
- ❒ Metabolic acidosis
- ❒ Neurologic disease

* Defined as ≥1 criterion:
- Respiratory rate ≥25/min
- PaO_2 ≤75 mmHg
- SpO_2 ≤92% in ambient air
- $PaCO_2$ ≥45 mmHg with arterial pH ≤7.35

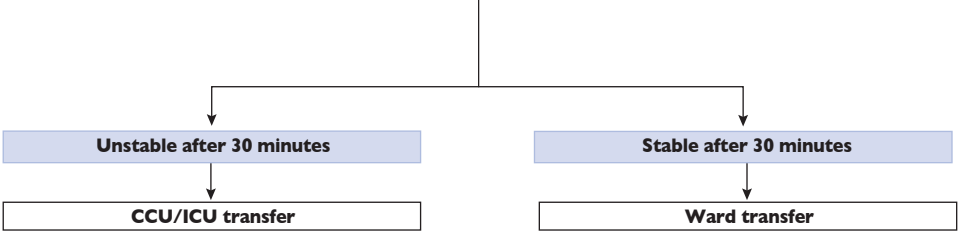
Reference: Ray P et al. Acute respiratory failure in the elderly: etiology, emergency diagnosis and prognosis. Critical Care (2006), 10 (3) :R82.

DYSPNEA: ACUTE HEART FAILURE *(see chapter 3.1)*

BASIC WORK-UP

- ❒ **Immediate 12-lead ECG, cardiac monitor, BP, respiratory rate, pulse oximetry**
- ❒ **Clinical findings**
  Most commonly: lower extremity edema, jugular venous distension, rales; work up for underlying cardiac disease and triggers
- ❒ **Laboratory findings**
  Complete blood count, chemistries, cardiac enzymes, BNP, TSH, ABG as needed
- ❒ **Chest X-ray (lung ultrasound)**
- ❒ **Echocardiogram**
  During admission (earlier if decompensated aortic stenosis or endocarditis are suspected)
- ❒ **Coronary angiography**
  Emergent in patients with ACS; delayed in patients with suspected coronary artery disease

❒ Positioning	Keep head of bed elevated above level of legs
❒ Oxygen	Up to 12 L/min via non-rebreather, titrate oxygen saturation to 95%
❒ Nitroglycerin	1-2 SL tablets or 2-3 patches 10 mg (1st choice). In pulmonary edema with severe shortness of breath: NTG drip 0.05% (100 mg in 200 ml) - Start with 25 µg/min = 3 ml/h, check BP after 5 and 10 min - Increase dose per SHO/attending recommendations by 25 µg/min at a time as long as SBP >90 mmHg - Additional BP check 5 and 10 min after each increase in dosing - Check BP every 20 min once a steady drip rate is reached
❒ Furosemide	40-120 mg i.v. (adjust based on kidney function and clinical findings; monitor creatinine)
❒ Morphine	2 mg i.v. (preceeded by 10 mg i.v. metoclopramide PRN)
❒ Consider digoxin	0.5 (-1.0) mg i.v. in patients with atrial fibrillation
❒ Anticoagulation	Therapeutic dosing in ACS and atrial fibrillation: Enoxaparin 1 mg/kg body weight as 1st dose



Reference: Ware L B and Matthay M A.. Acute Pulmonary Edema. New Engl J Med (2005); 353:2788-2796.

DYSPNEA: ACUTE PULMONARY EMBOLISM *(see chapter 6.2)*

Priorities: 1. Vital signs 2. Diagnostic screening dependent upon clinical stratification

ABG, ECG, chest X-ray plus clinical assessment of PE probability (risk factors) plus monitoring

Hemodynamically unstable

Initiate transfer to ICU

Immediate TTE (if available)

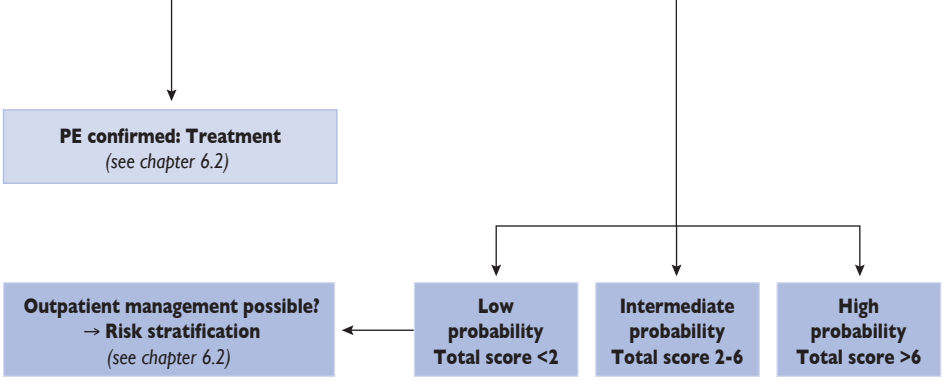
Result inconclusive → CT-angio

Right ventricular dysfunction

Hemodynamically stable

Wells criteria for PE:

	Score
❒ Clinical signs and symptoms of deep vein thrombosis (DVT)	+ 3.0
❒ No alternative diagnosis (or alternative diagnosis less likely than PE)	+ 3.0
❒ Heart rate >100/min	+ 1.5
❒ Immobilization or operation within the last 4 weeks	+ 1.5
❒ Previous DVT or PE	+ 1.5
❒ Hemoptysis	+ 1.0
❒ Malignant tumor with treatment within the last 6 months or palliative care	+ 1.0



Stein PD, Woodard PK, Weg JG, et al. Diagnostic pathways in acute pulmonary embolism: recommendations of the PIOPED II investigators. Am J Med (2006);119:1048–55.
Goldhaber SZ. Pulmonary embolism. Lancet (2004); 363 (9417) 1295-1305
Agnelli G and Becattini C. Acute Pulmonary Embolism. New Engl J Med (2010); 363:266-274

DYSPNEA: COPD EXACERBATION

❒ **Verify diagnosis (DD: PE, acute heart failure, pneumothorax)**
❒ **Oxygen administration → SpO_2 target 88-92% (Beware of carbonarcosis: ABC after 1 h)**

Definition:
Known COPD and/or
- ❒ Progressive dyspnea and/or
- ❒ Change in quantitiy and color of sputum and/or
- ❒ Heavy coughing

→ ❒ COPD classification (GOLD)
→ ❒ Etiology

❒ History, clinical examination (blood pressure, pulse, oxygen saturation, vigilance)

↓

- ❒ Laboratory findings: Blood count, coagulation, ProCT, perhaps BNP, D-Dimers
- ❒ Chest X-ray; ECG (exclusion of differential diagnoses)
- ❒ Sputum cultures (always in case of hospitalisation or previous outpatient antibiotic treatment)

→ ❒ Hospitalisation indicated?
→ ❒ Evaluate ICU criteria
→ ❒ NIV indicated?

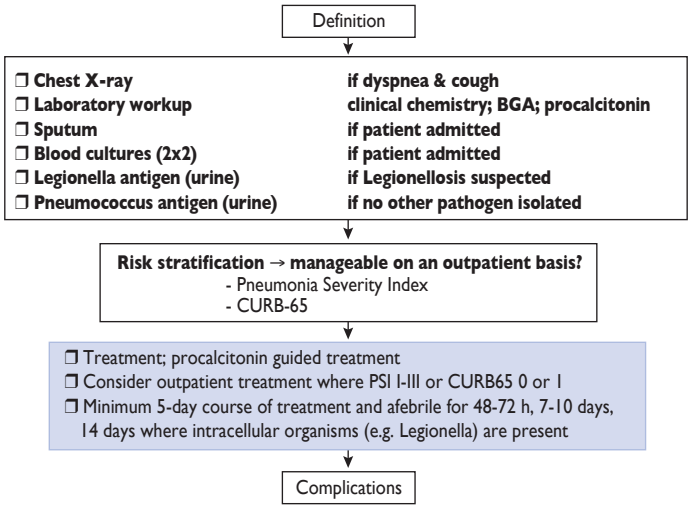
↓

- ❒ **Oxygen therapy** 2-(4) l; target saturation 90%
- ❒ **Salbutamol/ipratropium inhalations** ≥4-6 x/d, if needed long-term inhalation
- ❒ **Systemic steroids** prednisone 0.5 mg/kg of body weight for 5 days
- ❒ Antibiotic treatment should be considered; always indicated in stage Gold IV
- ❒ Physiotherapy

→ ❒ Follow-up

DYSPNEA: COMMUNITY-ACQUIRED PNEUMONIA

Objective: diagnostics, risk stratification & empirical immediate treatment <2(-4) hrs.



Copyrights: Mandell LA et al . Infectious Diseases Society of America/American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults. Clin Infect Dis. (2007);44 Suppl 2:S27-72. - Halm EA and Teirstein AS. Management of Community-Acquired Pneumonia New Engl J Med (2002); 347:2039-2045 - Woodhead M et al. Guidelines for the management of adult lower respiratory tract infections ERJ December 1, (2005); 26 (6) 1138-1180

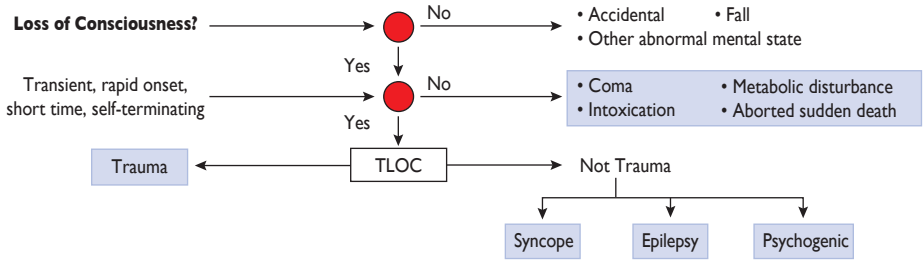
SYNCOPE: Assessment of patients with transient loss of consciousness (TLOC)

Syncope is a transient loss of consciousness due to global cerebral hypoperfusion (usually, itself due to a period of low blood pressure) characterised by rapid onset, short duration, spontaneous and complete recovery.

The differentiation between syncope and non-syncopal conditions with real or apparent LOC can be achieved in most cases with a **detailed clinical history** but sometimes can be extremely difficult. The following questions should be answered:

- Was LOC complete?
- Was LOC transient with rapid onset and short duration?
- Did the patient recover spontaneously, completely and without sequelae?
- Did the patient lose postural tone?

If the answers to these questions are positive, the episode has a high likelihood of being syncope. If the answer to one or more of these questions is negative, exclude other forms of LOC before proceeding with syncope evaluation.



Reference: Sutton R. Clinical classification of syncope. Prog Cardiovasc Dis. (2013) ; 55(4):339-44.

SYNCOPE: DIAGNOSTIC CRITERIA (I)
Diagnostic criteria with initial evaluation

Vasovagal syncope is diagnosed if syncope is precipitated by emotional distress or orthostatic stress and is associated with typical prodrome.

Situational syncope is diagnosed if syncope occurs during or immediately after specific triggers.

Orthostatic syncope is diagnosed when it occurs after standing up and there is documentation of orthostatic hypotension.

Arrhythmia related syncope is diagnosed by ECG when there is:

- Persistent sinus bradycardia <40 bpm in awake or repetitive sinoatrial block or sinus pauses >3 s
- Mobitz II 2nd or 3rd degree AV block
- Alternating left and right BBB
- VT or rapid paroxysmal SVT
- Non-sustained episodes of polymorphic VT and long or short QT interval
- Pacemaker or ICD malfunction with cardiac pauses

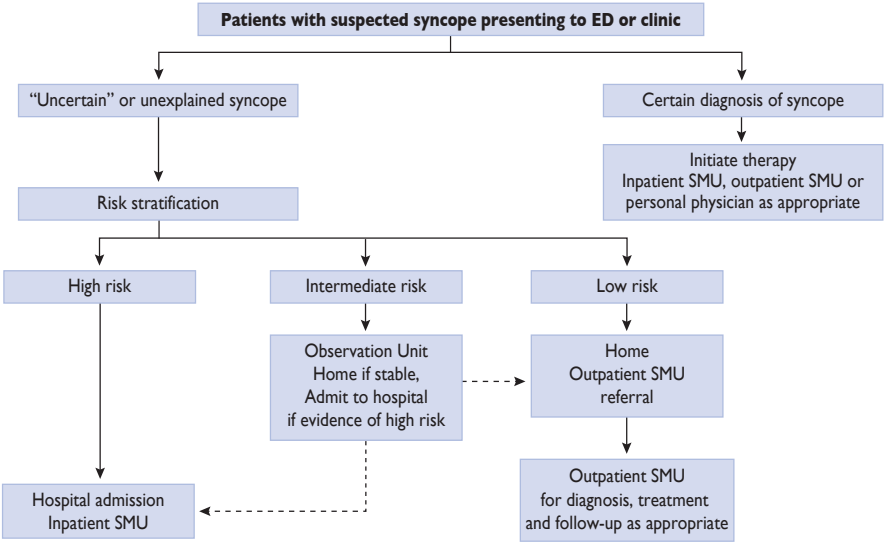
Cardiac ischemia related syncope is diagnosed when syncope presents with ECG evidence of acute ischemia with or without myocardial infarction.

Cardiovascular syncope is diagnosed when syncope presents in patients with prolapsing atrial myxoma, severe aortic stenosis, pulmonary hypertension, pulmonary embolus or acute aortic dissection.

Reference: Moya A et al. Eur Heart J(2009) 30, 2631–2671 (1)

SYNCOPE: Evaluation and risk stratification of patients with suspected syncope

Once syncope is considered to be the likely diagnosis, risk stratification is required to determine further management.



SYNCOPE: DIAGNOSTIC CRITERIA (2)
Diagnostic criteria with provocation maneuvers

CAROTID SINUS MASSAGE	ORTHOSTATIC HYPOTENSION
Indications • CSM is indicated in patients >40 years with syncope of unknown aetiology after initial evaluation; • CSM should be avoided in patients with previous MI, TIA or stroke within the past 3 months and in patients with carotid bruits (except if carotid Doppler studies excluded significant stenosis).	**Recommendations: Active standing** **Indications** • Manual intermittent determination with sphygmomanometer of BP supine and, when OH is suspected, during active standing for 3 min is indicated as initial evaluation; • Continuous beat-to-beat non-invasive pressure measurement may be helpful in cases of doubt.
Diagnostic criteria • CSM is diagnostic if syncope is reproduced in presence of asystole longer than 3 s and/or a fall in systolic BP >50 mmHg.	**Diagnostic criteria** • The test is diagnostic when there is a symptomatic fall in systolic BP from baseline value ≥20 mmHg or diastolic BP ≥10 mmHg or a decrease in systolic BP to <90 mmHg; • The test should be considered diagnostic when there is an asymptomatic fall in systolic BP from baseline value ≥20 mmHg or diastolic BP >10 mmHg or a decrease in systolic BP to <90 mmHg.

Reference: Moya A et al. Eur Heart J(2009) 30, 2631–2671 (2)

TREATMENT ACCORDING TO TYPE OF SYNCOPE (I)

Treatment of reflex syncope	Treatment of orthostatic hypotension
• Explanation of the diagnosis, provision of reassurance and explanation of risk of recurrence are in all patients • Isometric PCM are indicated in patients with prodrome • Cardiac pacing should be considered in patients with dominant cardioinhibitory CSS • Cardiac pacing should be considered in patients with frequent recurrent reflex syncope, age > 40 years and documented spontaneous cardioinhibitory response during monitoring • Midodrine may be indicated in patients with VVS refractory to lifestyle measures • Tilt training may be useful for education of patients but long-term benefit depends on compliance • Cardiac pacing may be indicated in patients with tilt-induced cardioinhibitory response with recurrent frequent unpredictable syncope and age > 40 after alternative therapy has failed • Triggers or situations inducing syncope must be avoided as much as possible • Hypotensive drugs must be modified or discontinued • Cardiac pacing is not indicated in the absence of a documented cardioinhibitory reflex • Beta-adrenergic blocking drugs are not indicated	• Adequate hydration and salt intake must be maintained • Midodrine should be administered as adjunctive therapy if needed • Fludrocortisone should be administered as adjunctive therapy if needed • PCM may be indicated • Abdominal binders and/or support stockings to reduce venous pooling may be indicated • Head-up tilt sleeping (>10°) to increase fluid volume may be indicated • Triggers or situations inducing syncope must be avoided as much as possible • Hypotensive drugs administered for concomitant conditions must be discontinued or reduced

TREATMENT ACCORDING TO TYPE OF SYNCOPE (2)

Treatment of arrhythmic syncope

Cardiac Pacing

- Pacing is indicated in patients with sinus node disease in whom syncope is demonstrated to be due to sinus arrest (symptom-ECG correlation) without a correctable cause
- Pacing is indicated in sinus node disease patients with syncope and abnormal CSNRT
- Pacing is indicated in sinus node disease patients with syncope and asymptomatic pauses > 3 sec. (with possible exceptions of young trained persons, during sleep and in medicated patients)
- Pacing is indicated in patients with syncope and 2nd degree Mobitz II, advanced or complete AV block
- Pacing is indicated in patients with syncope, BBB and positive EPS
- Pacing should be considered in patients with unexplained syncope and BBB
- Pacing may be indicated in patients with unexplained syncope and sinus node disease with persistent sinus bradycardia itself asymptomatic
- Pacing is not indicated in patients with unexplained syncope without evidence of any conduction disturbance

Catheter ablation

- Catheter ablation is indicated in patients with symptom/arrhythmia ECG correlation in both SVT and VT in the absence of structural heart disease (with exception of atrial fibrillation)
- Catheter ablation may be indicated in patients with syncope due to the onset of rapid atrial fibrillation

Antiarrhythmic drug therapy

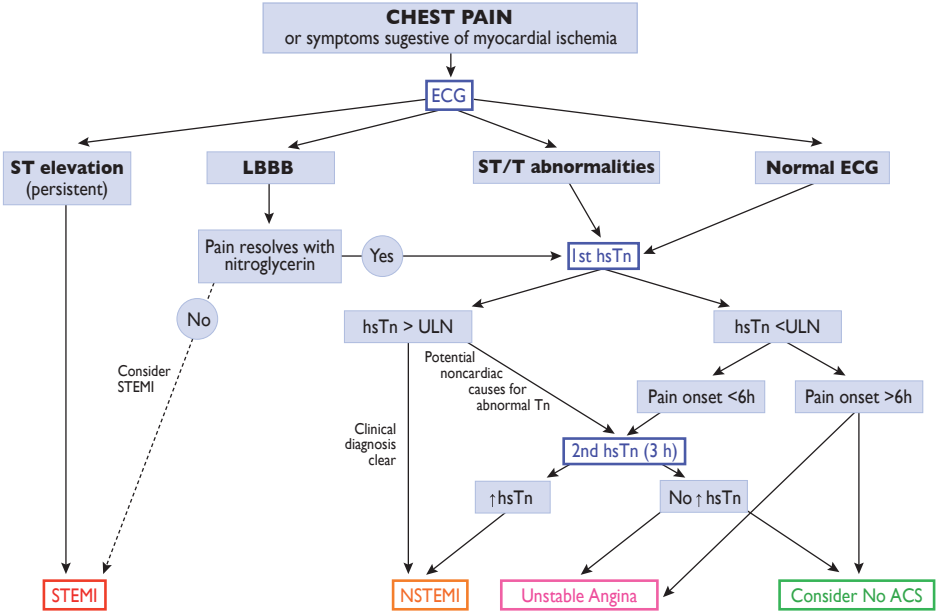
- Antiarrhythmic drug therapy, including rate control drugs, is indicated in patients with syncope due to onset of rapid atrial fibrillation
- Drug therapy should be considered in patients with symptom/arrhythmia ECG correlation in both SVT and VT when catheter ablation cannot be undertaken or has failed

Implantable Cardioverter Defibrillator (ICD)

- ICD is indicated in patients with documented VT and structural heart disease
- ICD is indicated when sustained monomorphic VT is induced at EPS in patients with previous myocardial infarction
- ICD should be considered in patients with documented VT and inherited cardiomyopathies or channelopathies

Chapter2
ACUTE CORONARY SYNDROMES

ACUTE CORONARY SYNDROMES: DIAGNOSIS



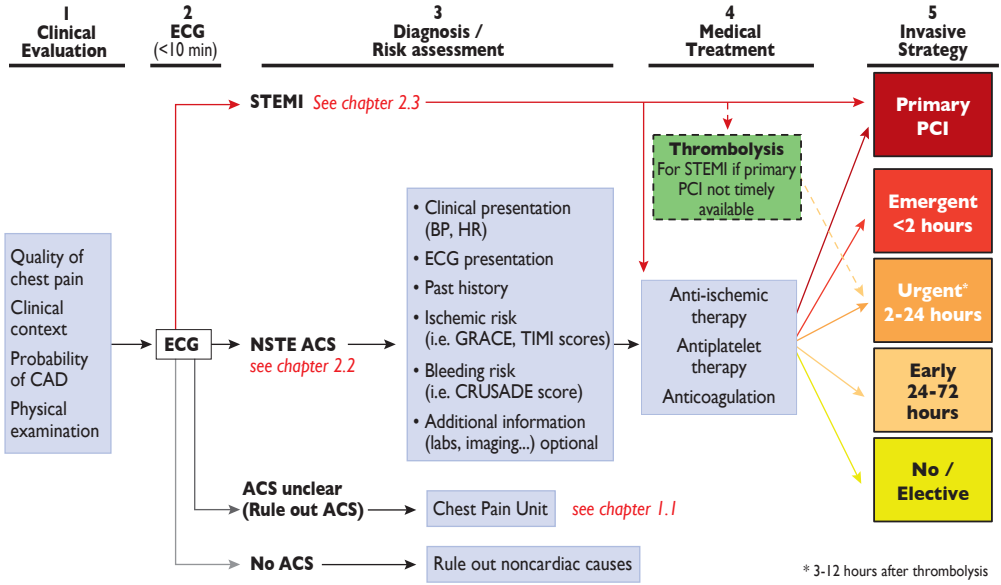
ACUTE CORONARY SYNDROMES: DIFFERENTIAL DIAGNOSIS (1)

CAUSES OF CHEST PAIN NOT RELATED TO ACS	CAUSES OF TROPONIN ELEVATION NOT RELATED TO ACS
PRIMARY CARDIOVASCULAR • Acute pericarditis, pericardial effusion • Acute myocarditis • Severe hypertensive crisis • Stress cardiomyopathy (Tako-Tsubo syndrome) • Hypertrophic cardiomyopathy, aortic stenosis • Severe acute heart failure • Acute aortic syndrome (dissection, hematoma) • Pulmonary embolism, pulmonary infarction • Cardiac contusion	**PRIMARY CARDIOVASCULAR** • Acute myo(peri)carditis • Severe hypertensive crisis • Pulmonary edema or severe congestive heart failure • Stress cardiomyopathy (Tako-Tsubo syndrome) • Post- tachy- or bradyarrhythmias • Cardiac contusion, ablation, pacing, cardioversion, or endomyocardial biopsy • Aortic dissection, aortic valve disease or hypertrophic cardiomyopathy • Pulmonary embolism, severe pulmonary hypertension
PRIMARY NON-CARDIOVASCULAR • Oesophageal spasm, oesophagitis, GER • Peptic ulcer disease, cholecystitis, pancreatitis • Pneumonia, bronchitis, asthma attack • Pleuritis, pleural effusion, pneumothorax • Pulmonary embolism, severe pulmonary hypertension • Thoracic trauma • Costochondritis, rib fracture • Cervical / thoracic vertebral or discal damage • Herpes Zoster	**PRIMARY NON-CARDIOVASCULAR** • Renal dysfunction (acute or chronic) • Critical illness (sepsis, respiratory failure…) • Acute neurological damage (i.e. stroke, subarachnoid hemorrhage) • Severe burns (affecting >30% of body surface area) • Rhabdomyolysis • Drug toxicity (chemotherapy with adriamycin, 5-fluorouracil, herceptin, snake venoms…) • Inflammatory or degenerative muscle diseases • Hypothyroidism • Infiltrative diseases (amyloidosis, hemochromatosis, sarcoidosis) • Scleroderma

ACUTE CORONARY SYNDROMES: DIFFERENTIAL DIAGNOSIS (2)
CAUSES OF REPOLARISATION ABNORMALITIES IN THE ECG NOT RELATED TO ACS

ST-SEGMENT ELEVATION	NEGATIVE T WAVES
Fixed • LV aneurysm • LBBB, WPW, hypertrophic cardiomyopathy, LVH • Pacemaker stimulation • Early repolarisation (elevated J-point) **Dynamic** • Acute (myo)pericarditis • Pulmonary embolism • Electrolytic disturbances (hyperkaliemia) • Acute brain damage (stroke, subarachnoid hemorrhage)	• Normal variants, i.e. women (right precordial leads), children, teenagers • Evolutive changes post MI • Chronic IHD • Acute (myo)pericarditis, cardiomyopathies • BBB, LVH, WPW • Post-tachycardia or pacemaker stimulation • Metabolic or ionic disturbances
ST-SEGMENT DEPRESSION	**PROMINENT T WAVES**
Fixed • Abnormal QRS (LBBB, WPW, pacemaker stimulation…) • LVH, hypertrophic cardiomyopathy • Chronic IHD **Dynamic** • Acute (myo)pericarditis • Acute pulmonary hypertension • Electrolytic disturbances (hyperkalemia) • Intermitent LBBB, WPW, pacing • Post-tachycardia / cardioversion • Severe hypertensive crisis • Drug effects (digoxin) • Shock, pancreatitis • Hyperventilation	• Normal variants, i.e. early repolarisation • Metabolic or ionic disturbances (i.e. hyperkalemia) • Acute neurological damage (stroke, subarachnoid hemorrhage)

GENERAL APPROACH TO THE PATIENT WITH CHEST PAIN / SUSPECTED ACS



Reference: Hamm CW et al . Eur Heart J (2011) 32 (23): 2999-3054 [1]

NON ST-SEGMENT ELEVATION ACS: RISK STRATIFICATION

ISCHEMIC RISK

GRACE RISK SCORE

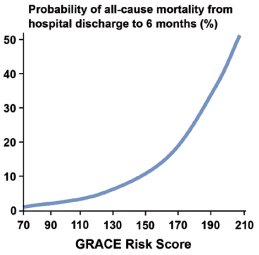
Predictive Factors

- Age
- HR*
- SBP*
- Creatinine (mg/dl)*
- Killip class*
- Cardiac arrest
- ST-segment deviation
- Elevated cardiac markers

* On admission

Outcomes

Death or death/MI in-hospital and at 6 months



Risk calculation

http://www.outcomes.org/grace

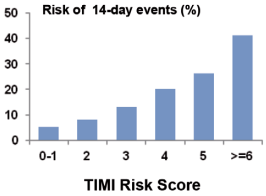
TIMI RISK SCORE

Predictive Factors

- Age ≥65 years
- At least 3 risk factors for CAD
- Significant (>50%) coronary stenosis
- ST deviation
- Severe anginal symptoms (>2 events in last 24 h)
- Use of aspirin in last 7 days
- Elevated serum cardiac markers

Outcome

All-cause mortality / new or recurrent MI / severe recurrent ischemia requiring urgent revascularisation at 14 days



Risk calculation

(1 point each)

http://www.timi.org

BLEEDING RISK

CRUSADE RISK SCORE

Predictive Factors

- Sex
- HR*
- SBP*
- Creatinine (mg/dl)*
- Baseline hematocrit*
- GFR: Cockcroft-Gault*
- Diabetes
- Prior vascular disease
- Signs of congestive heart failure*

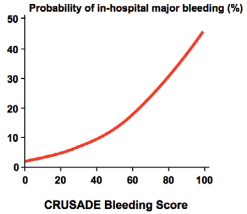
* On admission

Outcome

In-hospital major bleeding

Risk calculation

www.crusadebleedingscore.org

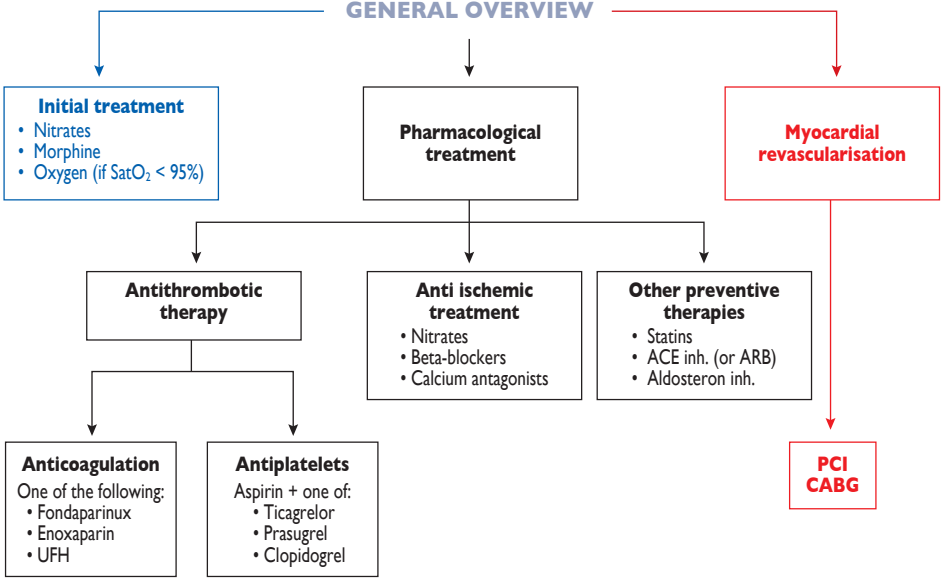


Eagle KA et al. A validated prediction model for all forms of acute coronary syndrome: estimating the risk of 6-month post-discharge death in an international registry. JAMA. (2004) ;291(22):2727-33

Antman EM, et al. The TIMI risk score for unstable angina/non-ST elevation MI: A method for prognostication and therapeutic decision making. JAMA. (2000) ;284(7):835-42

Subherwal S, et al Baseline risk of major bleeding in non-ST-segment-elevation myocardial infarction: the CRUSADE (Can Rapid risk stratification of Unstable angina patients Suppress ADverse outcomes with Early implementation of the ACC/AHA Guidelines) Bleeding Score. Circulation (2009) ;119(14):1873-82

NON ST-SEGMENT ELEVATION ACS: TREATMENT (I)
GENERAL OVERVIEW



NON ST-SEGMENT ELEVATION ACS: TREATMENT (2)
DOSING AND RECOMMENDATIONS FOR PHARMACOLOGICAL THERAPIES

Drug	Loading dose	Maintenance dose	Considerations	Major contraindications (in addition to specific allergies)
INITIAL TREATMENT				
Nitrates	2–3 mg i.v. 1–2 puffs s.l.			Severe hypotension
Morphine	2–3 mg i.v.	–	If pain refractary to nitrates	
Oxygen	FiO_2 needed for SpO_2 >95%		Only if SpO_2 <95%	
ANTITHROMBOTIC THERAPY			First line treatment. Careful use if active/very high bleeding risk	
Aspirin	300 mg oral	75–150 mg QD		
Ticagrelor	180 mg oral	90 mg BID	Preferred in moderate-high risk patients at diagnosis	Previous intracerebral hemorrhage
Prasugrel	60 mg oral	10 mg QD	Preferred in clopi-naïve with CAD and PCI planned If tica/prasu not available	Previous stroke/TIA Weight <60 kg, Age >75 years*
Clopidogrel	300–600 mg oral	75 mg QD	Preferred if no immediate cath	
Fondaparinux	2.5 mg s.c.	2.5 mg s.c. QD	If >75 years, No LD and MD 0.75 mg/Kg/12 h	GFR <30 ml/min/1,73m^2
Enoxaparin	30 mg i.v. + 1 mg/kg s.c.	1 mg/Kg/BID	Consider if anticoagulation needed for other reasons	
UFH	4000 IU i.v.	1000 IU/h	Consider only if immediate cath	
Bivalirudin	0.75 mg/kg i.v.	1.75 mg/kg/h ≤4 h		

Reference: Hamm CW et al . Eur Heart J (2011) ;32 (23): 2999-3054 [2]

* Unless dose reduced to 5 mg

NON ST-SEGMENT ELEVATION ACS: TREATMENT (3)
DOSING AND RECOMMENDATIONS FOR PHARMACOLOGICAL THERAPIES (cont.)

Drug	Loading dose	Maintenance dose	Considerations	Major contraindications (in addition to specific allergies)
ANTI-ISCHEMIC TREATMENT				
Nitrates	–	Titrated according to BP	oral/topic/iv available	Hypotension
Beta-blockers			Preferred over calcium channel blockers	Coronary spasm, severe brachycardia, AV block, severe bronchospasm
Atenolol	25–100 mg oral	25–100 mg QD	Only if normal LVEF	
Carvedilol	3,125–25 mg oral	3,125–25 mg BID	Preferred if LVSD/HF	
Bisoprolol	1,25–10 mg oral	1,25–10 mg QD	Preferred if LVSD/HF	
Metoprolol	25–100 mg oral	25–100 mg BID	Preferred if LVSD/HF	
Calcium antagonists			Consider if BB contraindicated First option in vasospastic angina	
Verapamil	80–120 mg oral	80–240 mg TID-QD		Bradycardia, HF, LVSD
Diltiazem	60–120 mg oral	60–300 mg TID-QD		Bradycardia, HF, LVSD
Amlodipine	5–10 mg oral	5–10 mg QD		Hypotension

NON ST-SEGMENT ELEVATION ACS: TREATMENT (3)
DOSING AND RECOMMENDATIONS FOR PHARMACOLOGICAL THERAPIES (Cont.)

Drug	Loading dose	Maintenance dose	Considerations	Major contraindications (in addition to specific allergies)
OTHER THERAPIES				
Statins	–	**	Use initially in all patients	
ACE inhibitors	–	**	LVSD, HF, HTN. Consider in all others	Hypotension
Angiotensin RB	–	**	Same as ACEI (preferred if ACEI-related cough)	Severe KD
Aldosterone RB	–	25 mg QD	In NSTEMI + LVEF <40% and HF or diabetes	Hyperkaliemia

** Multiple drugs and doses available.

Reference: Hamm CW et al . Eur Heart J (2011); 32 (23): 2999-3054 [3]

NON ST-SEGMENT ELEVATION ACS: TREATMENT (4)
INDICATIONS AND TIMING OF INVASIVE STRATEGY

Clinical situation	Timing
Severe clinical or electrical instability: Cardiogenic shock, severe heart failure, acute mitral regurgitation, refractory symptoms, ventricular arrhythmias	**Within first 2 hours**
Significant troponin rise / fall **ST changes in ECG** **Other risk markers** • DM • Renal insufficiency (eGFR <60 ml/min/1.73 m^2) • Reduced LV function (LVEF <40%) • Early postinfarction angina • Recent coronary revascularisation • Intermediate-high GRACE risk score	**Within first 24 hours**
Other non low-risk patients	**Within first 72 hours**
Low risk patients **Non candidates for coronary revascularisation**	**No invasive strategy**

Reference: Hamm CW et al . Eur Heart J (2011); 32 (23): 2999-3054 [4]

STEMI: ELECTROCARDIOGRAPHIC DIAGNOSIS

STEMI is diagnosed according to the presence of the following acute ischemic ECG changes:

In the absence of LVH and LBBB:

- New ST elevation at the J point in 2 contiguous leads with ≥0.2 mV in men or ≥ 0.15 mV in women in leads V_2-V_3 and/or ≥0.1 mV in other leads
  → Contiguous leads mean lead groups such as anterior leads (V_1-V_6), inferior leads (II, III, aVF) or lateral/apical leads (I, aVL).

In the presence LBBB or ST depression:

- New LBBB, and symptoms suggestive of ACS
- ST depression in leads V_1–V_3 indicate inferobasal myocardial ischemia (especially when the terminal T-wave is positive)

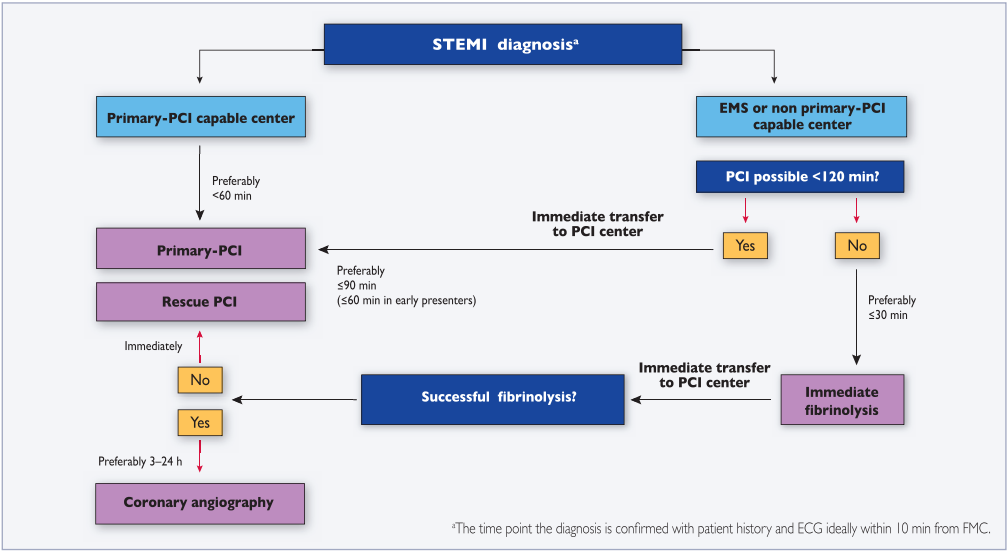
In suspected posterior (circumflex artery- related) or right ventricle-related infarction:

- ST elevation in V_7 (at the left posterior axillary line), V_8 (at the left midscapular line), and V_9 (at the left paraspinal border), using a cut-point >0.05 mV.
  → Capture a overlooked left dominant circumflex using posterior leads in the fifth interspace.

- ST elevation in right precordial leads (V_3R and V_4R), using a cut-off point >0.05 mV, and >0.1 mV in men <30 years.
  → Capture suspected right ventricular infarction using right precordial leads.

Reference: Hamm CW et al . Eur Heart J (2011); 32 (23): 2999-3054 [5]

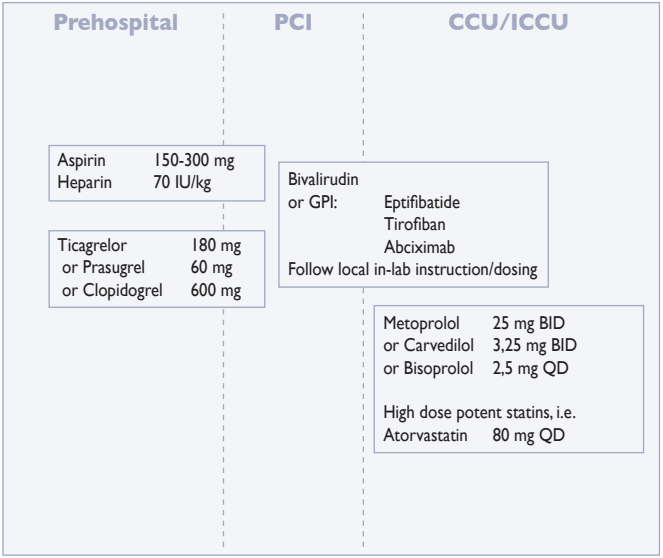
STEMI TREATMENT (I)
GENERAL OVERVIEW OF INITIAL MANAGEMENT



[a]The time point the diagnosis is confirmed with patient history and ECG ideally within 10 min from FMC.

Reference: Steg G et al. Eur Heart J. (2012);33:2569-619 [6]

STEMI TREATMENT (2)
PRIMARY PCI - FIRST 24 HOURS AND DAYS 2-7



Medication Titration Day 2-7

Aspirin	75–100 mg QD
Ticagrelor	90 mg BID
or Prasugrel	10/5 mg QD
or Clopidogrel	75 mg QD
(if ticagrelor/prasugrel unavailable)	

Metoprolol	200 mg QD
or carvedilol	25 mg BID
or bisoprolol	5 mg BID

or Ca-antagonist (see NSTEACS chapter)

Start ACEI or ARB in LVSD, CHF or DM or to control BP

Aldosterone RB in LVSD, CHF or DM

Start or continue anti-hyperglycemic medication

Reference: Steg G et al. Eur Heart J. (2012);33:2569-619 [7]

STEMI TREATMENT (3)
FIBRINOLYSIS: DOSING AND CONTRAINDICATIONS

Doses of fibrinolytic agents		
	Initial treatment	**Specific contraindications**
Streptokinase (SK)	1.5 million units over 30–60 min i.v.	Prior SK or anistreplase
Alteplase (tPA)	15 mg i.v. bolus 0.75 mg/kg over 30 min (up to 50 mg) then 0.5 mg/kg over 60 min i.v. (up to 35 mg)	
Reteplase (rt-PA)	10 units + 10 units i.v. bolus given 30 min apart	
Tenecteplase (TNK–tPA)	Single i.v. bolus: 30 mg if <60 kg 35 mg if 60 to <70 kg 40 mg if 70 to <80 kg 45 mg if 80 to <90 kg 50 mg if ≥90 kg	

Reference: Steg G et al. Eur Heart J. (2012);33:2569-619 [8]

Contraindications to fibrinolytic therapy
Absolute
Previous intracranial hemorrhage or stroke of unknown origin at any time
Ischemic stroke in the preceding 6 months
Central nervous system damage or neoplasms or arteriovenous malformation
Recent major trauma/surgery/head injury (within the preceding 3 weeks)
Gastrointestinal bleeding within the past month
Known bleeding disorder (excluding menses)
Aortic dissection
Non-compressible punctures in the past 24 h (e.g. liver biopsy, lumbar puncture)
Relative
Ischemic stroke more than 6 months ago
Transient ischemic attack in the preceding 6 months
Oral anticoagulant therapy
Pregnancy or within 1 week postpartum
Refractory hypertension (systolic blood pressure >180 mmHg and/or diastolic blood pressure >110 mmHg)
Advanced liver disease
Infective endocarditis
Active peptic ulcer
Prolonged or traumatic resuscitation

Reference: Steg G et al. Eur Heart J. (2012);33:2569-619 [8]

STEMI TREATMENT (4)
DOSING OF ANTITHROMBOTIC DRUGS USED FOR STEMI TREATMENT

Doses of antiplatelet co-therapies	
With primary PCI	
Aspirin	Loading dose of 150–300 mg orally or of 80–150 mg i.v. if oral ingestion is not possible, followed by a maintenance dose of 75–100 mg QD.
Clopidogrel	Loading dose of 600 mg orally, followed by a maintenance dose of 75 mg QD.
Prasugrel	Loading dose of 60 mg orally, followed by a maintenance dose of 10 mg QD. In patients with body weight <60 kg, a maintenance dose of 5 mg is recommended. In patients >75 years, prasugrel is generally not recommended, but a dose of 5 mg should be used if treatment is deemed necessary.
Ticagrelor	Loading dose of 180 mg orally, followed by a maintenance dose of 90 mg BID.
Abciximab	Bolus of 0.25 mg/kg i.v. and 0.125 µg/kg/min infusion (maximum 10 µg/min) for 12 h.
Eptifibatide	Double bolus of 180 µg/kg i.v. (given at a 10-min interval) followed by an infusion of 2.0 µg/kg/min for 18 h.
Tirofiban	25 µg/kg over 3 min i.v., followed by a maintenance infusion of 0.15 µg/kg/min for 18 h.
With fibrinolytic therapy	
Aspirin	Starting dose 150–500 mg orally or i.v. dose of 250 mg if oral ingestion is not possible.
Clopidogrel	Loading dose of 300 mg orally if aged ≤75 years, followed by a maintenance dose of 75 mg QD.

Doses of antiplatelet co-therapies (Cont.)	
Without reperfusion therapy	
Aspirin	Starting dose 150–500 mg orally.
Clopidogrel	75 mg/day orally.
Doses of anticoagulation co-therapies	
With primary PCI	
Unfractionated heparin	70–100 U/kg i.v. bolus when no GP IIb/IIIa inhibitor is planned. 50–60 U/kg i.v. bolus with GP IIb/IIIa inhibitors.
Enoxaparin	0.5 mg/kg i.v. bolus.
Bivalirudin	0.75 mg/kg i.v. bolus followed by i.v infusion of 1.75 mg/kg/h for up to 4 h after the procedure as clinically warranted. After cessation of the 1.75 mg/kg/h infusion, a reduced infusion dose of 0.25 mg/kg/h may be continued for 4–12 h as clinically necessary.
With fibrinolytic therapy	
Unfractionated heparin	60 U/kg i.v. bolus with a maximum of 4000 U followed by an i.v. infusion of 12 U/kg with a maximum of 1000 U/h for 24–48 h. Target aPTT: 50–70 s or 1.5 to 2.0 times that of control to be monitored at 3, 6, 12 and 24 h.
Enoxaparin	In patients <75 years of age: 30 mg i.v. bolus followed 15 min later by 1 mg/kg s.c. every 12 h until hospital discharge for a maximum of 8 days. The first two doses should not exceed 100 mg. In patients >75 years of age: no i.v. bolus; start with first s.c. dose of 0.75 mg/kg with a maximum of 75 mg for the first two s.c. doses. In patients with creatinine clearance of <30 mL/min, regardless of age, the s.c. doses are given once every 24 h.

Doses of anticoagulation co-therapies (Cont.)	
With fibrinolytic therapy	
Fondaparinux	2.5 mg i.v. bolus followed by a s.c. dose of 2.5 mg once daily up to 8 days or hospital discharge.
Without reperfusion therapy	
Unfractionated heparin	Same dose as with fibrinolytic therapy.
Enoxaparin	Same dose as with fibrinolytic therapy.
Fondaparinux	Same dose as with fibrinolytic therapy.

Reference: Steg G et al. Eur Heart J. (2012);33:2569-619 [9]

STEMI TREATMENT (5)
DOSING OF OTHER DRUGS USED IN STEMI

Metoprolol: 5-25 mg BID, titrate as tolerated up to 200 mg QD
Bisoprolol: 1.25-5 mg QD, titrate as tolerated up to 10 mg QD
Carvedilol: 3.125-6.25 mg BID, titrate as tolerated up to 50 mg BID
Atenolol: 25-100 mg QD, titrate as tolerated up to 100 mg QD only if no LVSD or CHF

Ramipril: 1.25-5 mg QD, titrate as tolerated up to 10 mg QD
Lisinopril: 2.5 mg QD, titrate as tolerated up to 20 mg QD
Enalapril: 2.5-5 mg BID, titrate as tolerated up to 20 mg BID
Other ACEI are also optional

Valsartan: 80 mg QD, titrate as tolerated up to 320 mg QD
Candesartan: 8 mg QD, titrate as tolerated up to 32 mg QD
Losartan: 25-50 mg QD, titrate as tolerated up to 100 mg QD
Other ARBs are also optional

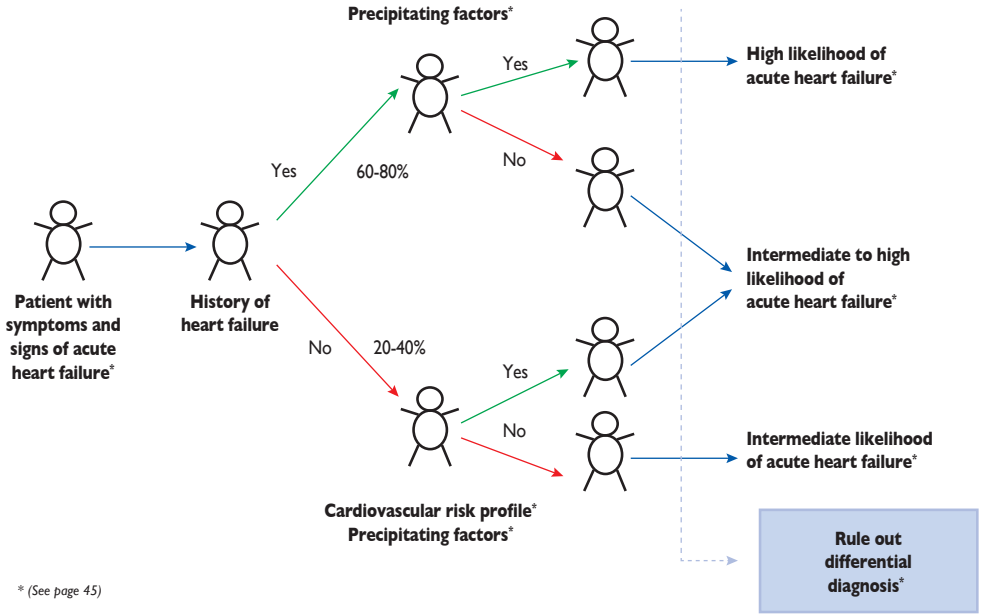
Spironolactone: 25 mg QD, titrate as needed and tolerated up to 100 mg QD
Eplerenone: 12.5-25 mg QD, titrate as tolerated up to 50 mg QD

Atorvastatin: 80 mg QD, down titrate if side effects
Other statins: Start with high doses and down titrate if side effects

Reference: Steg G et al. Eur Heart J. (2012);33:2569-619 [10]

Chapter 3
ACUTE HEART FAILURE

ACUTE HEART FAILURE: DIAGNOSIS AND CAUSES (1)



* (See page 45)

ACUTE HEART FAILURE: DIAGNOSIS AND CAUSES (2)

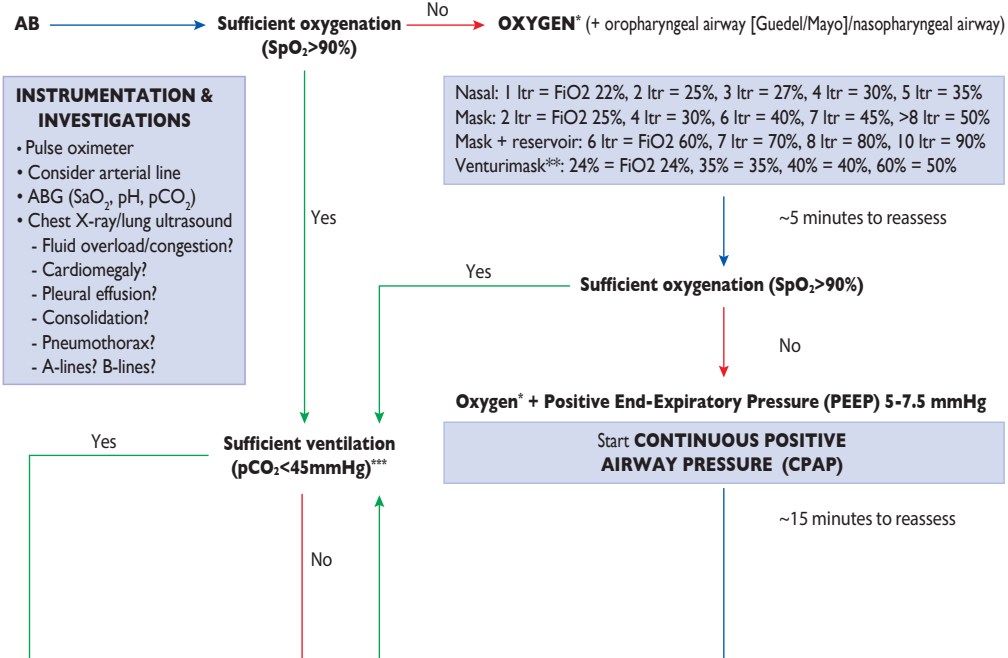
1• **Symptoms:** Dyspnea (on effort or at rest)/breathlessness, fatigue, orthopnea, cough, weight gain/ankle swelling
2• **Signs:** Tachypnea, tachycardia, low or normal blood pressure, raised jugular venous pressure, 3rd/4th heart sound, rales, edema;
3• **Cardiovascular risk profile:** Older age, HTN, diabetes, smoking, dyslipidemia, family history, history of CVD
4• **Precipitating factors:** Myocardial ischemia, rhythm disturbances, medication (NSAID, negative inotropic agents), infection, noncompliance
5• **Differential diagnosis:** Exacerbated pulmonary disease, pneumonia, pulmonary embolism, pneumothorax, acute lung injury, acute respiratory distress syndrome, (severe) anaemia, hyperventilation (acidosis), sepsis/septic shock, redistributive/hypovolemic shock
6• **Likelihood:** Clinical risk scores might be of additional value. They have high specificity but moderate sensitivity. They include predictors such as elevated BNP/NT-proBNP, interstitial edema on chest X-ray, orthopnea, lack of fever, diuretic use, age >75 years, rales.

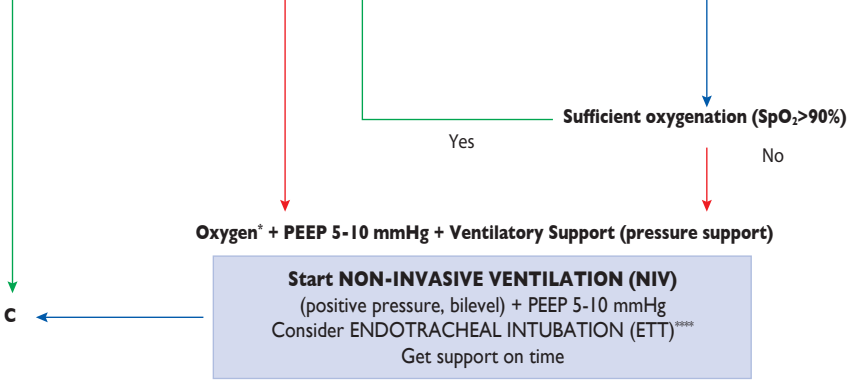
MAIN CAUSES OF ACUTE HEART FAILURE

- Coronary artery disease
- Hypertension
- Cardiomyopathy (familial, acquired)
- Valvular heart disease
- Peri-/endocardial disease
- Congenital heart disease
- Arrhythmia (tachy-, brady-)
- Conduction disorder (blocks)
- Volume overload (renal, iatrogenic)
- Tumor
- Pleural effusion
- Anxiety disorder
- Neurologic disease

Reference: McMurray JJ et al, Eur Herat J (2012) ;33(14):1787-847 (19)

ACUTE HEART FAILURE: INITIAL DIAGNOSIS AND TREATMENT
AIRWAY (A) & BREATHING (B)





* Goal SpO_2 94-98%
** Use the predefined liters of oxygen. When using higher flows the FiO_2 will drop
*** For a patient with COPD, a pCO_2 of 45-50 mmHg may be optimal. Aim for a normal pH
**** Consider if the above fails or when patient is fatigued

ACUTE HEART FAILURE: INITIAL DIAGNOSIS (CDE)

C – CIRCULATION* HR (bradycardia [<60/min], normal [60-100/min], tachycardia [>100/min]), rhythm (regular, irregular), SBP (very low [<85 mmHg], low, normal [110-140 mmHg], high [>140 mmHg]), and elevated jugular pressure should be checked

INSTRUMENTATION & INVESTIGATIONS:

Consider intravenous (central) & arterial line (BP monitoring)

Laboratory measures
- Cardiac markers (troponin, (BNP/NT-proBNP)
- Hb, electrolytes, creatinine, urea, glucose, inflammation, TSH

Standard 12-lead ECG
- Rhythm, rate, conduction times?
- Signs of ischemia/myocardial infarction? Hypertrophy?

Echocardiography
- Ventricular function (systolic and diastolic)?
- Presence of valve dysfunction (severe stenosis/insufficiency)?
- Pericardial effusion/tamponade?

ACTIONS:

Rule in/out diagnosis of acute heart failure as diagnosis for symptoms and signs

Establish cause of disease

Determine severity of disease

D – DISABILITY DUE TO NEUROLOGICAL DETERIORATION

Normal consiousness/altered mental status? Glasgow Coma Scale: EMV score <8 ⟶ Consider ETT

Anxiety, restlessness? ⟶ Consider morphine 2.0-5 mg i.v. bolus (diluted in normal saline), preceded by metoclopramide 10 mg i.v. PRN

E – EXPOSURE & EXAMINATION

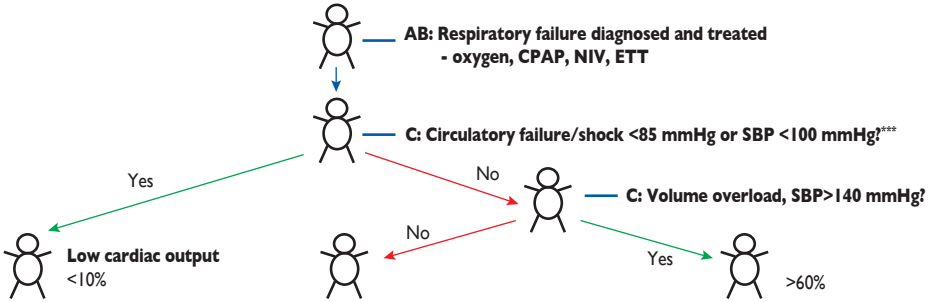
Temperature/fever : central and peripheral

Weight

Skin/extremities: circulation (e.g. capilary refill), color

Urinary output (<0.5ml/kg/hr) ⟶ Insert indwelling catheter

ACUTE HEART FAILURE: INITIAL TREATMENT (C) CLINICAL SCENARIOS*



1. Inotropic drugs - Dobutamine 2.5 µg/kg/min - Milrinone bolus 25 µg/kg in10-20 min, continuous 0.375 µg/kg/min **2. Vasopressor i.v.** - Norepinephrine 0.2 µg/kg/min **3. Diuretics i.v.** - Furosemide 20-40 mg bolus, continuous 100 mg/6 h **4. Consider hypertonic saline + diuretic** **5. Consider mechanical circulatory support**	**1. Diuretics i.v.** - Furosemide 20-40 mg bolus, continuous 100 mg/6 h**** **2. Inotropic drugs** - Dobutamine continuous 2.5 µg/kg/min - Milrinone bolus 25 µg/kg in 10-20 min, continuous 0.375 µg/kg/min - Levosimendan bolus 12 µg/kg in 10 min, continuous 0.1 µg/kg/min **3. Consider continuing beta-blockers, ACE-inhibitors at lower dose**	**1. Vasodilators** - Nitroglycerine spray 400 µg sublingual, repeat ~5-10 min - Nitroglycerine i.v. continuously ~10 µg/min, increase ~5 µg/min - Nitroprusside 0.3 µg/kg/min **2. Diuretics i.v.** - Furosemide 20-40 mg bolus, continuous 100 mg/6 h **3. Consider continuing beta-blockers, ACE inhibitors at lower dose**

* Clinical scenarios differ between patients. These scenarios describe the potential treatments schemes for often seen scenarios.

** Use the predefined liters of oxygen. When using higher flows the FiO2 will drop.

*** If caused by acute coronary syndrome/arrhythmias/valvular disease patients should be treated accordingly (see respective Toolkit chapters)

**** Use higher dose in patients on chonic diuretic treatment for HF (i.e. 2.5 times normal dose)

ACUTE HEART FAILURE: REASSESS ABCDE (CLINICAL SCENARIOS)

INITIAL TREATMENT

AB: Sufficient respiration and ventilation?*

ABC: Relieve of dyspnea/symptoms?

C: Normalisation of BP? HR? Urinary output?**

AB: SpO_2 94-98% and pCO_2 <45 mmHg - titrate oxygen, consider PEEP (CPAP, NIV, ETT)
C: Increase or add vasodilator if SBP >110 mmHg at reassessment, cave blood pressure drop >40 mmHg from initial assessment
C: Increase dosing of inotropic drugs if SBP <100 mmHg or low urinary output or altered mental state (organ perfusion), consider adding vasopressor therapy if necessary
C: Treat concomitant cardiac disease according guidelines, i.e. rhythm disturbances, myocardial ischemia
DE: Consider presence of co-morbity and treat accordingly, i.e. hyperglycemia, infection, electrolyte disturbances

Admit patient to the Intensive Care or Cardiac Care Unit for additional diagnostics and treatment***
Thrombosis prophylaxis should be started in patients not anticoagulated (enoxaparin 1 mg/Kg as first dose)
After stabilization (>24 hours) consider adding beta-blocker, ACEI/ARB, aldosterone antagonist, start low
Maintain an adequate nutritional status with a nutritional support of 20-25 kcal/kg/day within the first 48 hours

*Goals SpO_2 94-98%, pCO_2<45mmHg. **Goals SBP 100-120/60 mmHg, Frequency 60-100/min + regular rhythm, urinary output >0.5 ml/kg/hr.
*** Re-evaluate until patient is considered stable

ACUTE HEART FAILURE: PHARMACOLOGICAL THERAPY RECOMMENDATIONS AND DOSING (I)

- Based on additional investigation, patients can be categorised into an underlying cause and into HF with reduced and preserved ejection fraction
- After stabilisation and admission to the ICU, CCU or cardiology ward, heart failure treatment should be started or titrated
- Treatment for patients with reduced ejection fraction is better established

ACUTE HEART FAILURE: PHARMACOLOGICAL THERAPY RECOMMENDATIONS AND DOSING (I)

DRUG	STARTING DOSE	TARGET DOSE	CONSIDERATIONS	MAJOR CONTRAINDICATIONS
ACE inhibitors			Check renal function, electrolytes, drug interactions	History of angioedema Known bilateral renal artery stenosis Pregnancy (risk)
Captopril	6.25 mg TID	50 TID		
Enalapril	2.5 mg BID	10-20 BID		
Lisinopril	2.5-5.0 mg QD	20-35 QD		
Ramipril	2.5 mg QD	5 BID		
Trandolapril	0.5 QD	4 QD		
RB			If ACEI is not tolerated Check renal function, electrolytes, drug interactions	History of angioedema. Known bilateral renal artery stenosis Pregnancy (risk)
Candesartan	4-8 mg QD	32 QD		
Valsartan	40 mg BID	160 BID		
Losartan	50 QD	150 QD		
ß-blockers			Check 12- lead ECG	Asthma Second on third- degree AV block
Bisoprolol	1.25 QD	10 QD		
Carvedilol	3.125 BID	25-50 BID		
Metaprolol	12.5-25 QD	200 QD		
Nebivolol	1.25 QD	10 QD		
Aldosterone-antagonists			Check renal function, electrolytes, drug interactions	Eplenerone ___, strong CYP3A-4 inhibitors
Spironolactone	25 QD	25-50 QD		
Eplerenone	25 QD	50 QD		

Reference: McMurray JJ. Eur Heart J (2012) 33, 1787–1847

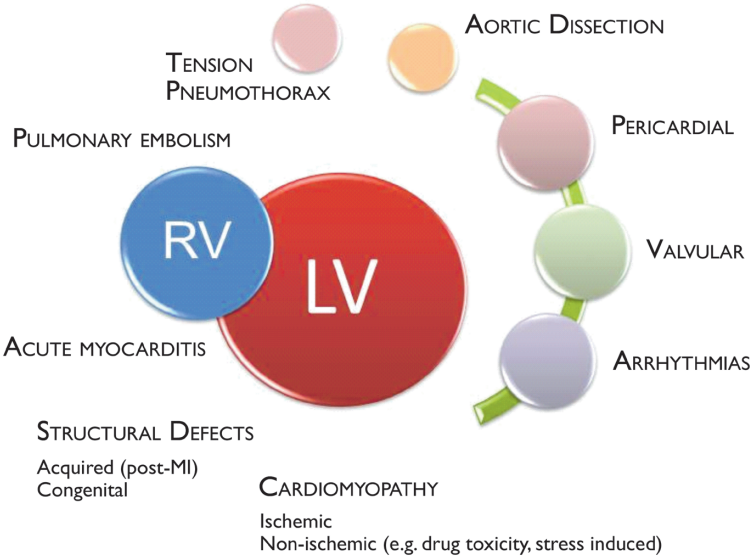
CARDIOGENIC SHOCK: DEFINITION

Clinical condition defined as the inability of the heart to deliver an adequate amount of blood to the tissues to meet resting metabolic demands as a result of impairment of its pumping function

Hemodynamic criteria to define cardiogenic shock
• Systolic blood pressure <80 to 90 mmHg or mean arterial pressure 30 mmHg lower than baseline
• Severe reduction in cardiac index: <1.8 L/min/m^2 without support or <2.0 to 2.2 L/min/m^2 with support
• Adequate or elevated filling pressure: Left ventricular end-diastolic pressure >18 mmHg or Right ventricular end-diastolic pressure >10 to 15 mmHg

CARDIOGENIC SHOCK: CAUSES

LV pump failure is the primary insult in most forms of CS, but other parts of the circulatory system contribute to shock with inadequate compensation or additional defects.



CARDIOGENIC SHOCK: INITIAL TRIAGE AND MANAGEMENT

This protocol should be initiated as soon as cardiogenic shock/end organ hypoperfusion is recognised and should not be delayed pending intensive care admission

EMERGENCY DEPARTMENT

0 min

5 min

15 min

60 min

CARDIAC INTENSIVE CARE UNIT

EARLY TRIAGE & MONITORING

Start high flow O_2

Establish i.v. access

- Age: 65–74, ≥75
- Heart rate >100 beats per minute
- Systolic blood pressure <100 mmHg
- Proportional pulse pressure ≤25 mmhg (CI <2.2l/min/m^2)
- Orthopnea (PCWP >22 mmHg)
- Tachypnea (>20/min), >30/min (!)
- Killip class II-IV
- Clinical symptoms of tissue hypoperfusion/hypoxia:
  - cool extremities,
  - decreased urine output (urine output <40 ml/h)
  - decreased capillary refill or mottling
  - alteration in mental status

INITIAL RESUSCITATION

- Arterial and a central venous catheterization with a catheter capable of measuring central venous oxygen saturation
- Standard transthoracic echocardiogram to assess left (and right) ventricular function and for the detection of potential mechanical complications following MI
- Early coronary angiography in specialized myocardial intervention center when signs and/or symptoms of ongoing myocardial ischemia (e.g. ST segment elevation myocardial infarction).

- CORRECT: hypoglycemia & hypocalcemia,
- TREAT: sustained arrhythmias: brady- or tachy-
- Isotonic saline-fluid challenge of 20 to 30 ml per kilogram of body weight over a 30-minute period to achieve a central venous pressure of 8 to 12 mmHg or until perfusion improves (with a maximum of 500 ml)
- CONSIDER NIVmechanical ventilation for comfort (fatigue, distress) or as needed:
  - To correct acidosis
  - To correct hypoxemia
- INOTROPIC SUPPORT (dobutamine and/or vasopressor support)

TREATMENT GOALS

- a mean arterial pressure of 60 mmHg or above,
- a mean pulmonary artery wedge pressure of 18 mmHg or below,
- a central venous pressure of 8 to 12 mmHg,
- a urinary ouput of 0,5 ml or more per hour per kilogram of body weight
- an arterial pH of 7.3 to 7.5
- a central venous saturation ($ScvO_2$) ≥70% (provided SpO_2 ≥93% and Hb level ≥9 g/dl)

In persistent drug-resistant cardiogenic shock, consider mechanical circulatory support

CARDIOGENIC SHOCK: PHARMACOLOGIC TREATMENT

DRUG TYPE		CLINICAL ACTION	DOSAGE
Levosimendan	Calcium sensitizer	Vasodilation, positive inotropic	0.05–0.2 µg/kg/min
Milrinone	Phosphodiesterase inhibitor	Vasodilation, positive inotropic	0.375 µg/kg/min, titrate to effect; range: 0.25 to 0,75 µg/kg/min
Isoprenaline	$ß_1$, $ß_2$ agonist	Positive chronotropic (pulmonary vasodilation)	0.5–5 µg/min (0.25–2,5 mL of a 1:250,000 dilution) i.v. infusion
Dobutamine	$ß_1$,$α_1$/$ß_2$ agonist	$ß_2$-mediated vasodilation, positive inotropic, chronotronic	2–20 µg/kg/min
Dopamine	ß, α, dopaminergic agonist	Peripheral vasodilation (e.g. splanchnic, renal) Positive chronotropic, positive inotropic Vasoconstriction at high doses	4 µg/kg/min 4–8 µg/kg/min >8 µg/kg/min
Noradrenaline	$α_1$, $ß_1$ agonist	Vasoconstriction, positive inotropic	0.05–0.2 µg/kg/min titrate to effect

Inotropes and vasopressors should be administered via a central venous catheter. All patients requiring (inotropes) and vasopressors should have an arterial line placed as soon as practical. Dopamine may influence the endocrine response via the hypothalamic-pituitary axis and may have immunosuppressive effects. Low-dose dopamine should not be used for renal protection.

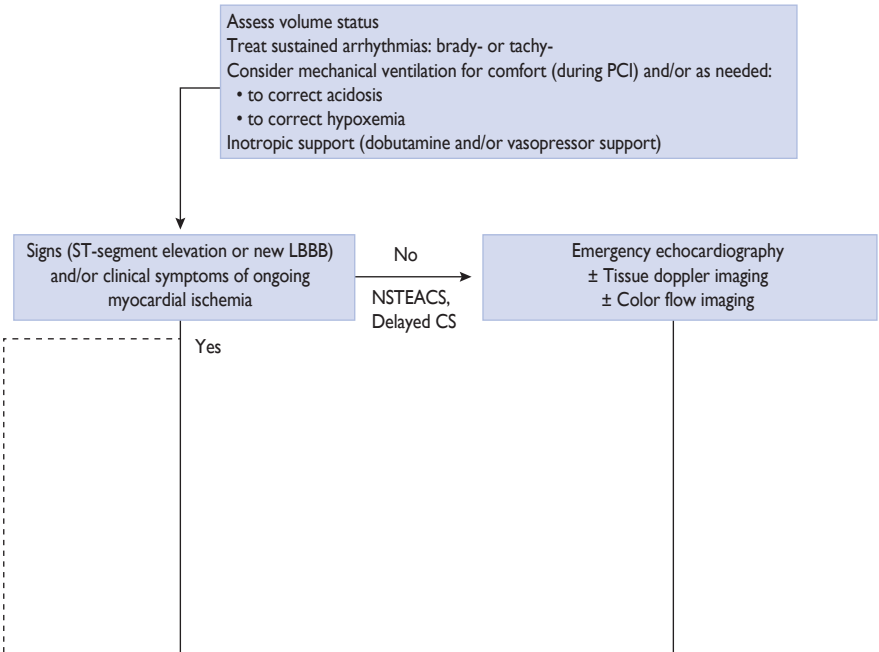
CARDIOGENIC SHOCK: VENTILATOR PROCEDURES

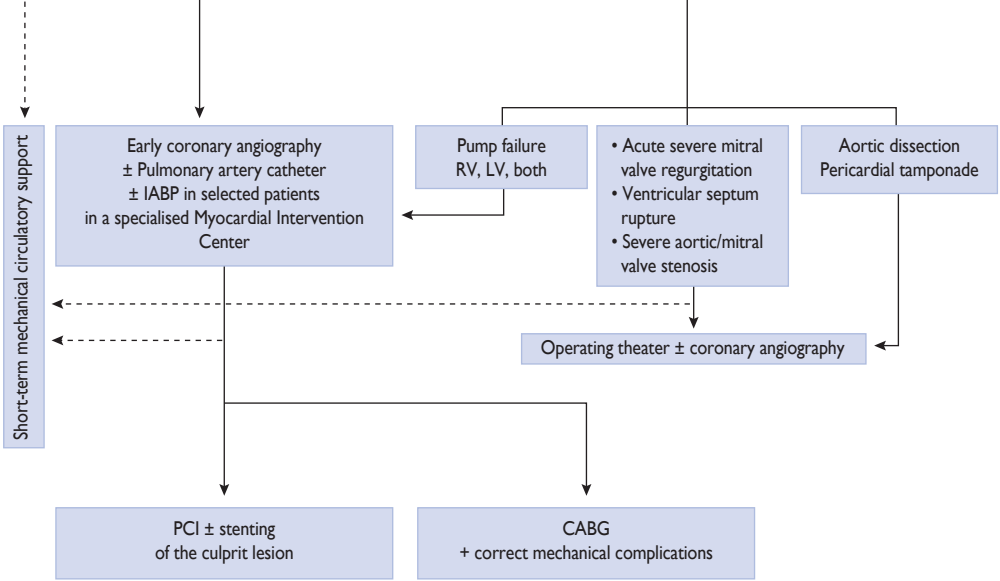
Ventilator mode	Pressure assist/control
Tidal Volume goal	Reduce tidal volume to 6-8 ml/kg lean body weight
Plateau Pressure goal	≤30 cm H_2O
Anticipated PEEP levels	5-10 cm H_2O
Ventilator rate and pH goal	12-20, adjusted to achieve a pH ≥7.30 if possible
Inspiration: Expiration time	1:1 to 1:2
Oxygenation goal:	
- PaO_2	50-80 mmHg
- SpO_2	>90%

Predicted body weight calculation:
- Male: 50 + 0.91 (height in cm - 152.4)
- Female: 45.5 + 0.91 (height in cm - 152.4)

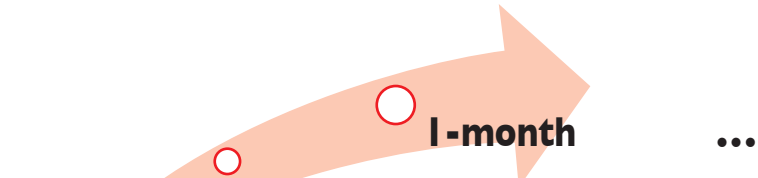
Some patients with CS will require increased PEEP to attain functional residual capacity and maintain oxygenation, and peak pressures above 30 cm H_2O to attain effective tidal volumes of 6-8ml/kg with adequate CO_2 removal.

CARDIOGENIC SHOCK: MANAGEMENT FOLLOWING STEMI




Short-term mechanical circulatory support
Early coronary angiography
± Pulmonary artery catheter
± IABP in selected patients
in a specialised Myocardial Intervention
Center
Pump failure
RV, LV, both
• Acute severe mitral valve regurgitation
• Ventricular septum rupture
• Severe aortic/mitral valve stenosis
Aortic dissection
Pericardial tamponade
Operating theater ± coronary angiography
PCI ± stenting
of the culprit lesion
CABG
+ correct mechanical complications

CARDIOGENIC SHOCK:
MECHANICAL CIRCULATORY SUPPORT, BASIC CHARACTERISTICS



IABP	Impella 2,5	Impella 5,0	Tandem-heart	Levitronix	ECMO	Implantable
Left ventricular support				BiVentricular support		
Partial support			Full support			
				Pulmonary support		

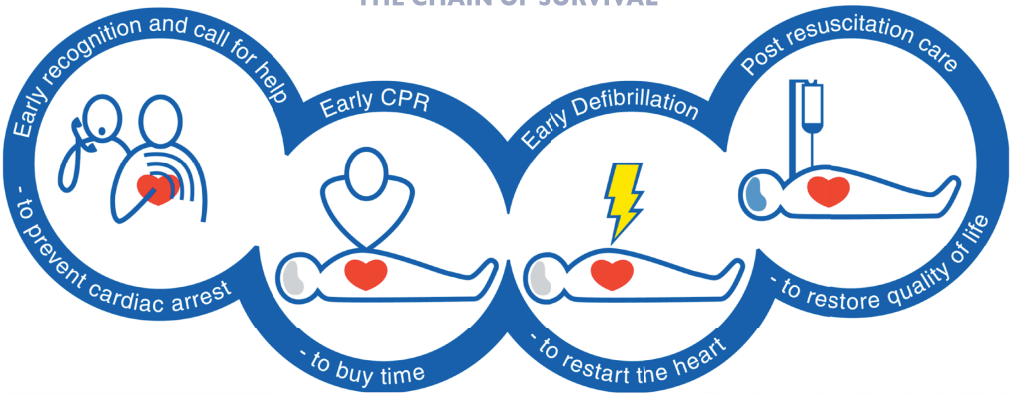
Level of support

	TYPE	SUPPORT		ACCESS
Intra-aortic balloon pump	Balloon counterpulsation	Pulsatile flow	<0.5 L	Arterial: 7.5 French
Impella Recover	Axial flow	Continuous flow		
LP 2.5			<2.5 L	Arterial: 12 French
CP			<4,0 L	Arterial: 14 French
LP 5.0			<5.0 L	Arterial: 21 French
Tandemheart	Centrifugal flow	Continuous flow	<5.0 L	Venous: 21 French Arterial: 15-17 French
Cardiohelp			<5.0 L	Venous: 15-29 French Arterial: 15-29 French

Different systems for mechanical circulatory support are available to the medical community. The available devices differ in terms of the insertion procedure, mechanical properties, and mode of action. A minimal flow rate of 70 ml/kg/min, representing a cardiac index of at least 2.5 L/m², is generally required to provide adequate organ perfusion. This flow is the sum of the mechanical circulatory support output and the remaining function of the heart

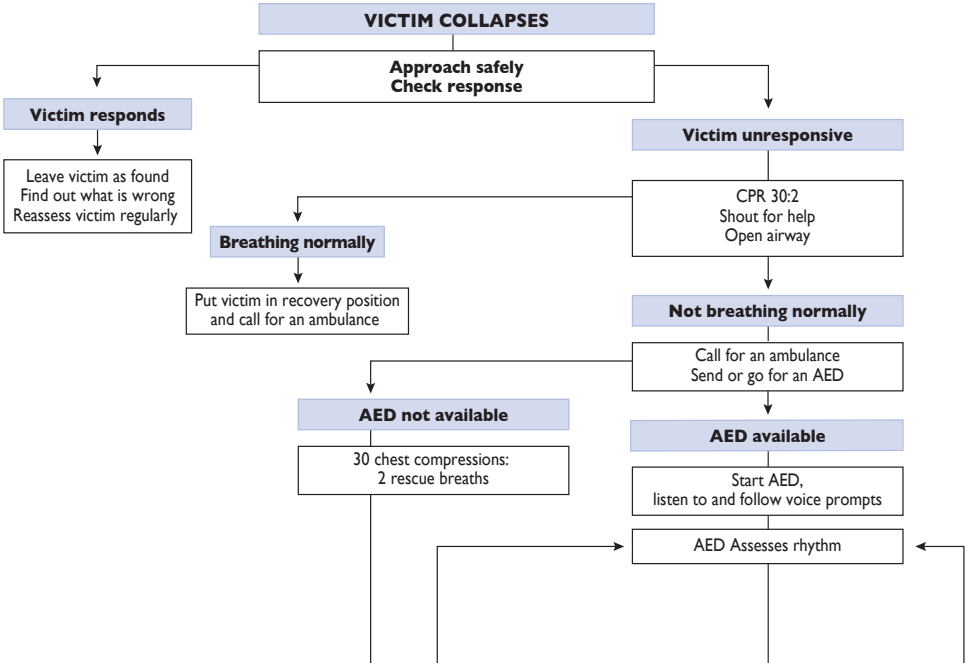
Chapter 4
CARDIAC ARREST AND CARDIOPULMONARY RESUSCITATION

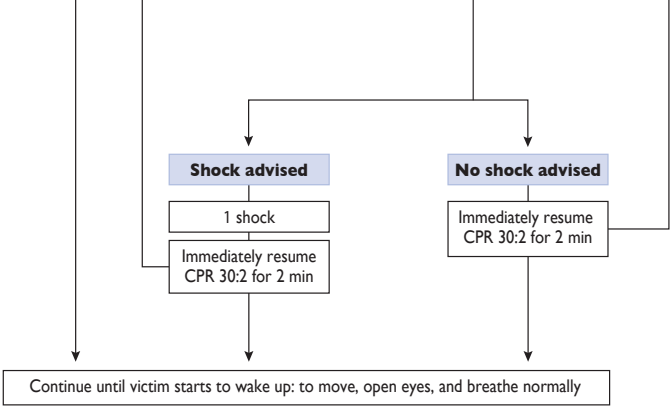
THE CHAIN OF SURVIVAL



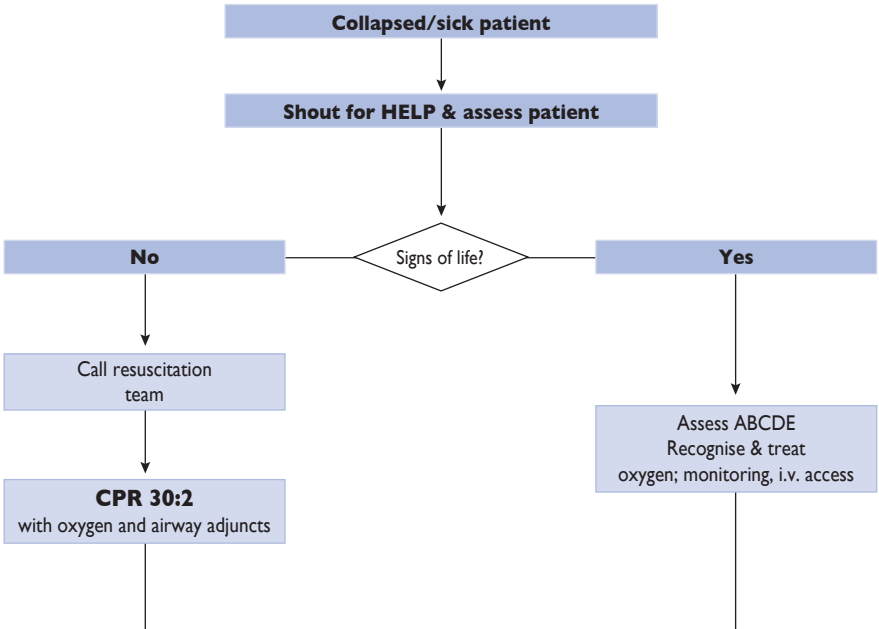
Adapted from Nolan JP, et al. European Resuscitation Council Guidelines for Resuscitation 2010. Executive Summary. Resuscitation 2010;81:1219-1276. © 2010 European Resuscitation Council www.erc.edu– 2013/029

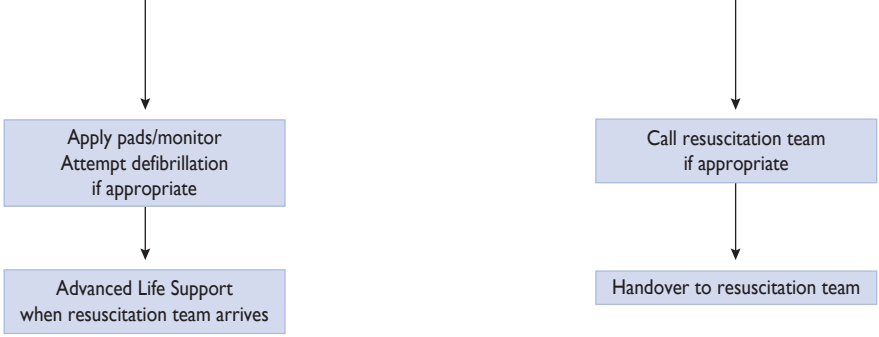
OUT OF HOSPITAL CARDIAC ARREST: ASSESSMENT OF A COLLAPSED VICTIM AND INITIAL TREATMENT



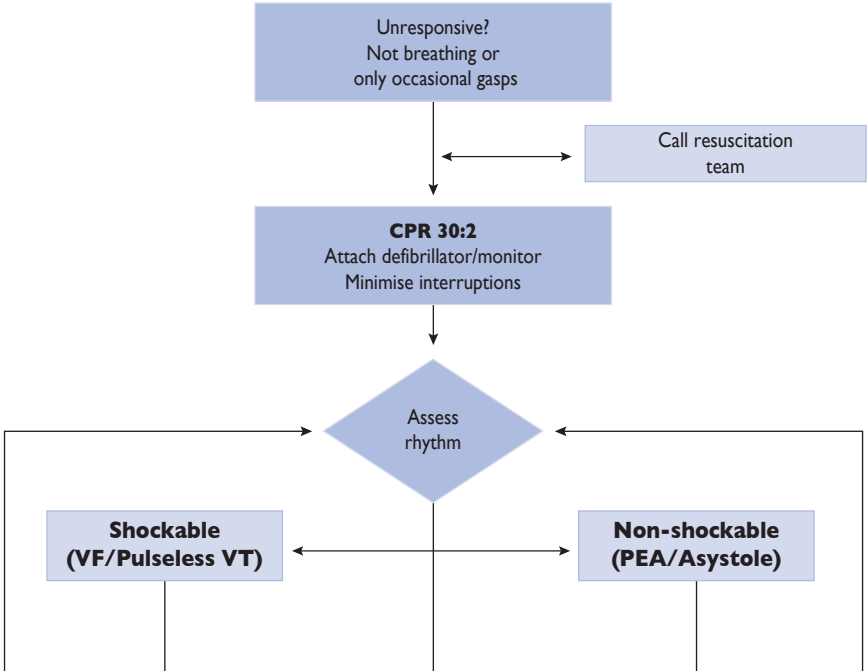

Shock advised
No shock advised
1 shock
Immediately resume CPR 30:2 for 2 min
Immediately resume CPR 30:2 for 2 min
Continue until victim starts to wake up: to move, open eyes, and breathe normally

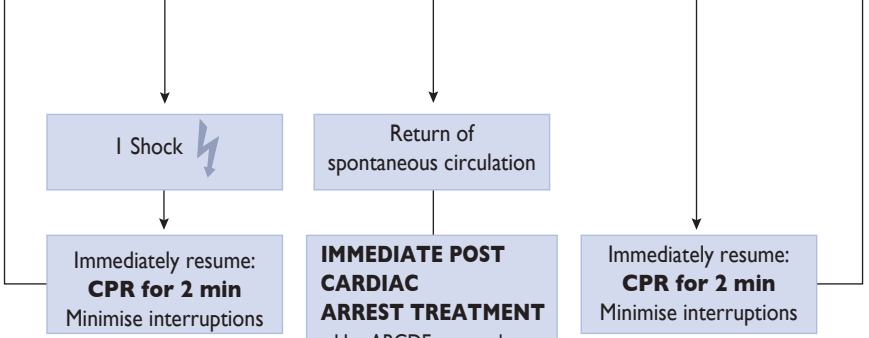
IN-HOSPITAL CARDIAC ARREST: ASSESSMENT OF A COLLAPSED VICTIM AND INITIAL TREATMENT




Apply pads/monitor
Attempt defibrillation
if appropriate
Advanced Life Support
when resuscitation team arrives
Call resuscitation team
if appropriate
Handover to resuscitation team

IN-HOSPITAL CARDIAC ARREST: ADVANCED LIFE SUPPORT





DURING CPR

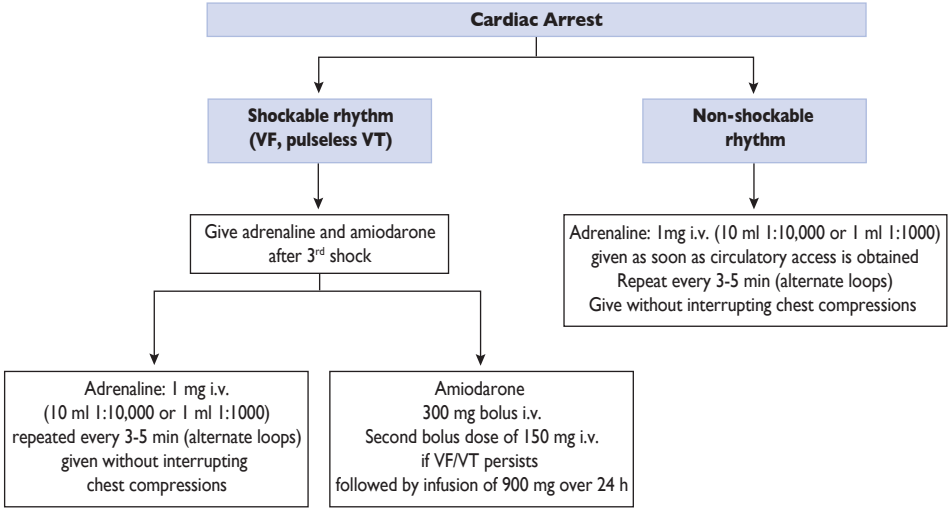
- Ensure high-quality CPR: rate, depth, recoil
- Plan actions before interrupting CPR
- Give oxygen
- Consider advanced airway and capnography
- Continuous chest compressions when advanced airway in place
- Vascular access (intravenous, intraosseous)
- Give adrenaline every 3-5 min
- Correct reversible causes

REVERSIBLE CAUSES

- Hypoxia
- Hypovolaemia
- Hypo-/hyperkalaemia/metabolic
- Hypothermia

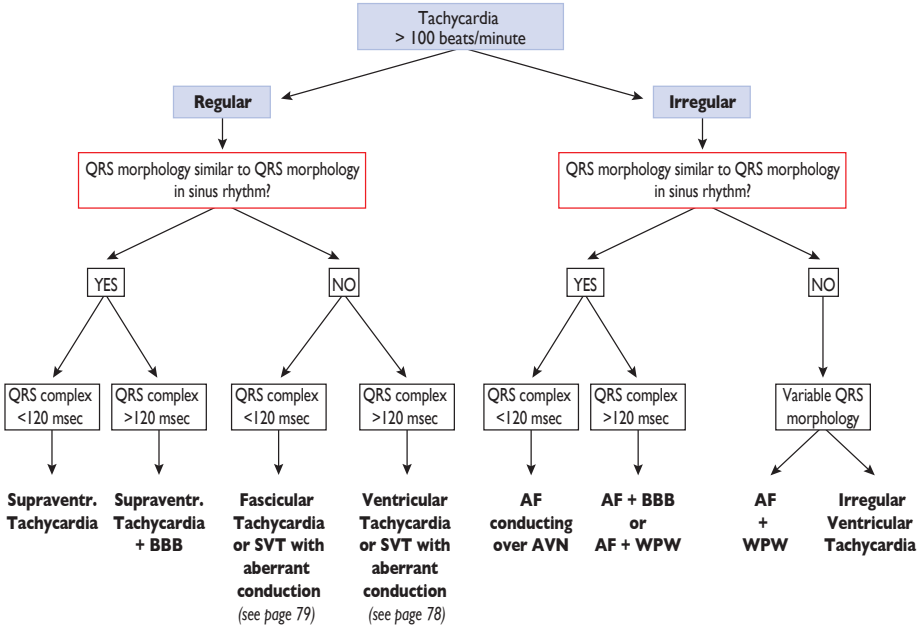
- Thrombosis
- Tamponade - cardiac
- Toxins
- Tension pneumothorax

IN-HOSPITAL CARDIAC ARREST: DRUG THERAPY DURING ADVANCED LIFE SUPPORT

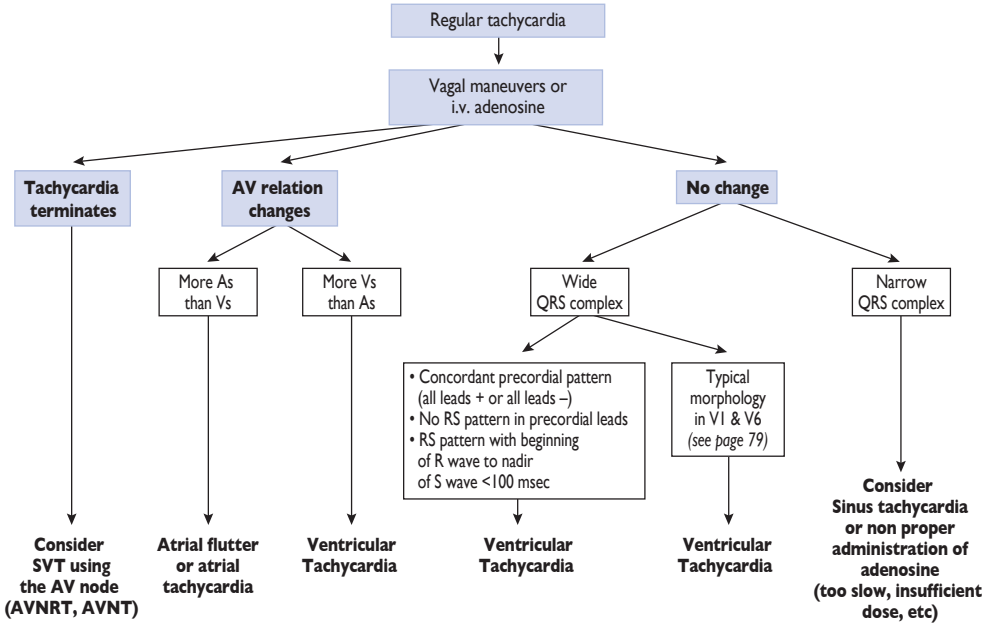


Chapter 5
RHYTHM DISTURBANCES

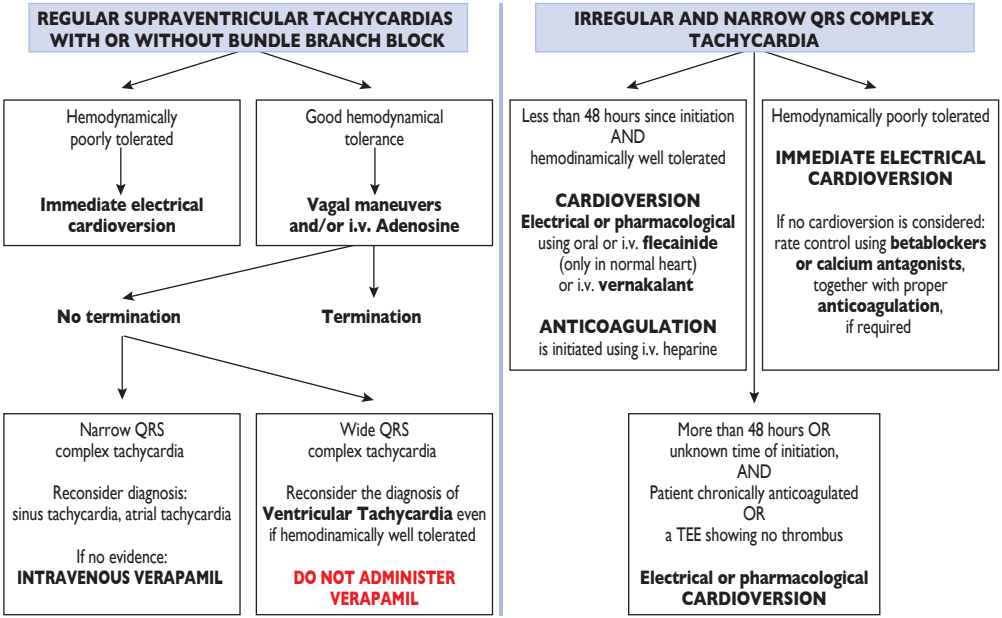
TACHYARRHYTHMIAS: DIAGNOSTIC CRITERIA



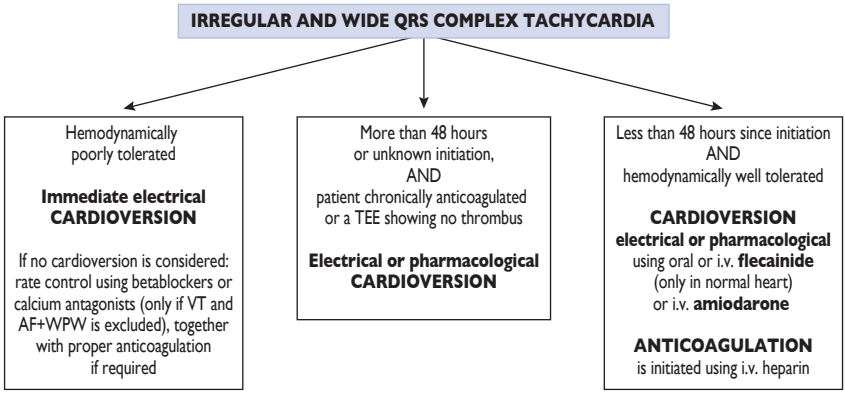
TACHYARRHYTHMIAS: DIAGNOSTIC MANEUVERS

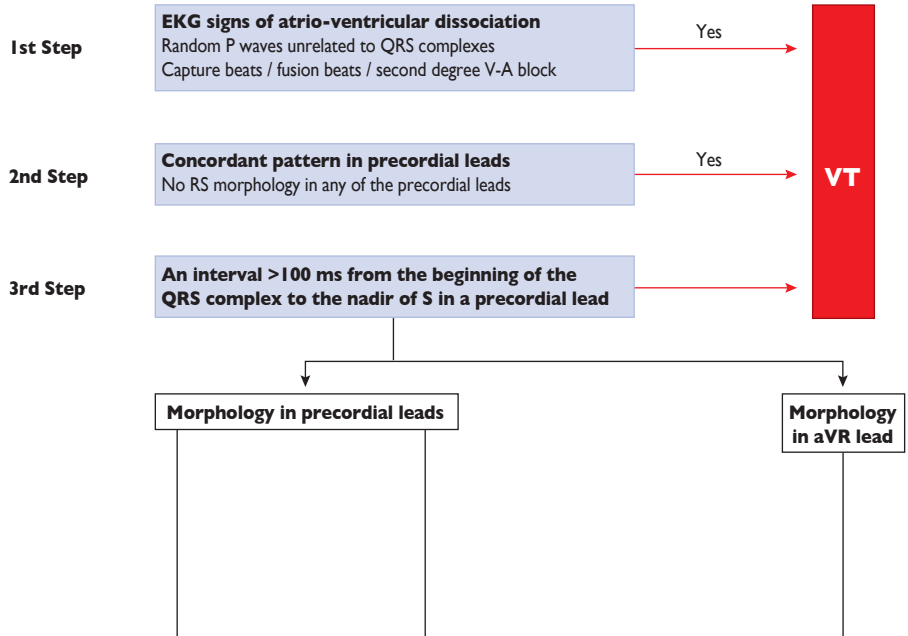


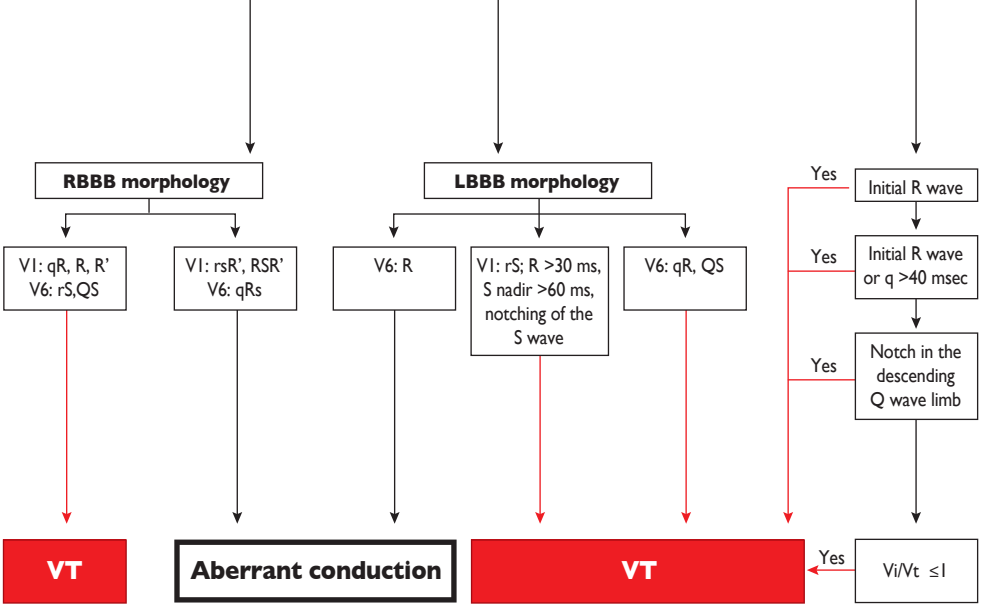
TACHYARRHYTHMIAS: THERAPEUTIC ALGORITHMS (I)



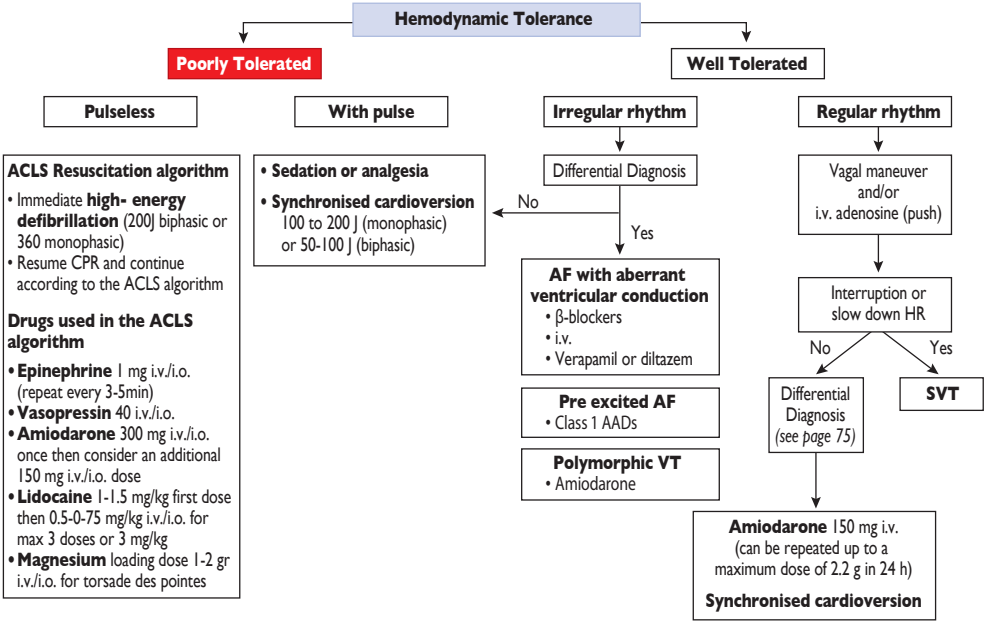
TACHYARRHYTHMIAS: THERAPEUTIC ALGORITHMS (2)




1st Step
EKG signs of atrio-ventricular dissociation
Random P waves unrelated to QRS complexes
Capture beats / fusion beats / second degree V-A block
Yes
2nd Step
Concordant pattern in precordial leads
No RS morphology in any of the precordial leads
Yes
VT
3rd Step
An interval >100 ms from the beginning of the QRS complex to the nadir of S in a precordial lead
Morphology in precordial leads
Morphology in aVR lead

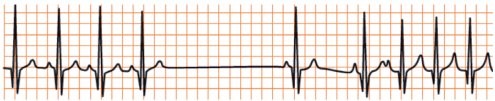

RBBB morphology
V1: qR, R, R'
V6: rS,QS
V1: rsR', RSR'
V6: qRs
LBBB morphology
V6: R
V1: rS; R >30 ms,
S nadir >60 ms,
notching of the
S wave
V6: qR, QS
Yes
Initial R wave
Yes
Initial R wave
or q >40 msec
Yes
Notch in the
descending
Q wave limb
VT
Aberrant conduction
VT
Yes
Vi/Vt ≤1

MANAGEMENT OF WIDE QRS TACHYCARDIAS



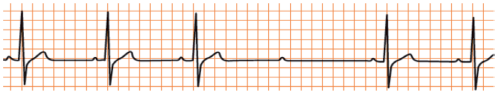
BRADYARRHYTHMIAS: DEFINITIONS AND DIAGNOSIS

SINUS NODE DYSFUNCTION



- Sinus bradycardia. It is a rhythm that originates from the sinus node and has a rate of under 60 beats per minute
- Sinoatrial exit block. The depolarisations that occur in the sinus node cannot leave the node towards the atria
- Sinus arrest. Sinus pause or arrest is defined as the transient absence of sinus P waves on the ECG

ATRIOVENTRICULAR (AV) BLOCKS



- First degree AV block. Atrioventricular impulse transmission is delayed, resulting in a PR interval longer than 200 msec
- Second degree AV block. Mobitz type I (Wenckebach block): Progressive PR interval prolongation, which precedes a nonconducted P wave
- Second degree AV block. Mobitz type II: PR interval remains unchanged prior to a P wave that suddenly fails to conduct to the ventricles
- Third degree (complete) AV block. No atrial impulses reach the ventricle

BRADYARRHYTHMIAS: TREATMENT (I)
ACUTE TREATMENT

- Rule out and treat any underlying causes of bradyarrhythmia
- Treat symptomatic patients only

PHARMACOLOGICAL TREATMENT

- **Atropine:** 0.5-1.0 mg, repeated up to 2 mg.
  Useful only if increased vagal tone, i.e. problem above AV node
- **Isoprenalin:** Bolus 20-40 µg i.v.
  Infusion 0.5 µg/min of 2 mg/100 ml normal saline
- **Adrenaline:** Infusion 2-10 µg/min.
  Useful when hypotension is an issue

TEMPORARY TRANSVENOUS PACING

Be Careful!

- Complications are common!
- Shall not be used routinely
- Use only as a last resource when chronotropic drugs are insufficient
- Every effort should be made to implant a permanent pacemaker as soon as possible, if the indications are established.

Indications limited to:

- High-degree AV block without escape rhythm
- Life threatening bradyarrhythmias, such as those that occur during interventional procedures, in acute settings such as acute myocardial infarction, drug toxicity.

BRADYARRHYTHMIAS: TREATMENT (2)
PACEMAKER THERAPIES IN SINUS NODE DYSFUNCTION

Permanent pacemaker is indicated in the following settings:

- Documented symptomatic bradycardia, including frequent sinus pauses that produce symptoms
- Symptomatic chronotropic incompetence
- Symptomatic sinus bradycardia that results from required drug therapy for medical conditions

Permanent pacemaker is not recommended in the following settings:

- Asymptomatic patients
- Patients for whom the symptoms suggestive of bradycardia have been clearly documented to occur in the absence of bradycardia
- Symptomatic bradycardia due to nonessential drug therapy

BRADYARRHYTHMIAS: TREATMENT (3)
PACEMAKER THERAPIES IN ATRIOVENTRICULAR BLOCKS

Permanent pacemaker therapy is indicated in the following settings regardless of associated symptoms:

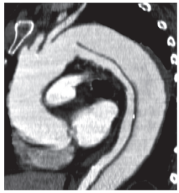
- Third-degree AV block
- Advanced second-degree AV block
- Symptomatic Mobitz I or Mobitz II second-degree AV block
- Mobitz II second-degree AV block with a wide QRS or chronic bifascicular block
- Exercise-induced second- or third-degree AV block
- Neuromuscular diseases with third- or second-degree AV block
- Third- or second-degree (Mobitz I or II) AV block after catheter ablation or valve surgery when block is not expected to resolve

Permanent pacemaker is not recommended in the following settings:

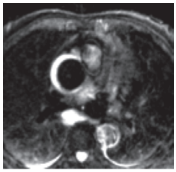
- Asymptomatic patients
- Patients for whom the symptoms suggestive of bradycardia have been clearly documented to occur in the absence of bradycardia
- Symptomatic bradycardia due to nonessential drug therapy

Chapter 6
ACUTE VASCULAR SYNDROMES

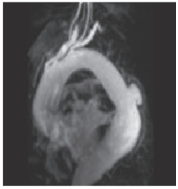
ACUTE AORTIC SYNDROMES: CONCEPT AND CLASSIFICATION (I)
TYPES OF PRESENTATION



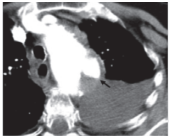
Classic aortic dissection
Separation of the aorta media with presence of extraluminal blood within the layers of the aortic wall. The intimal flap divides the aorta into two lumina, the true and the false



Intramural hematoma (IMH)
Aortic wall hematoma with no entry tear and no two-lumen flow



Penetrating aortic ulcer (PAU)
Atherosclerotic lesion penetrates the internal elastic lamina of the aorta wall



Aortic aneurysm rupture
(contained or not contained)

ACUTE AORTIC SYNDROMES: CONCEPT AND CLASSIFICATION (2)
ANATOMIC CLASSIFICATION AND TIME COURSE

DeBakey's Classification

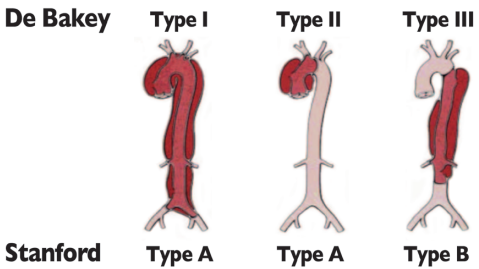
- Type I and type II dissections both originate in the ascending aorta
  - In type I, the dissection extends distally to the ascending aorta
  - In type II, it is confined to the ascending aorta
- Type III dissections originate in the descending aorta

Stanford Classification

- Type A includes all dissections involving the ascending aorta regardless of entry site location
- Type B dissections include all those distal to the brachiocephalic trunk, sparing the ascending aorta

Time course

- Acute: <2 weeks
- Subacute: 2-6 weeks
- Chronic: >6 weeks



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ACUTE AORTIC SYNDROME: CLINICAL SUSPICION AND DIFFERENTIAL DIAGNOSIS

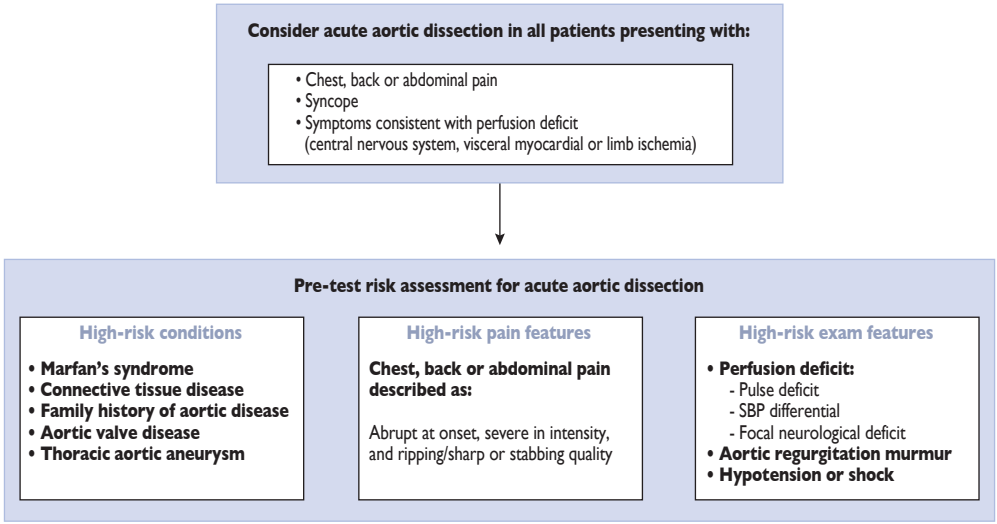
SYMPTOMS AND SIGNS SUGGESTIVE OF AAS

- Abrupt and severe chest/back pain with maximum intensity at onset
- Pulse/pressure deficit
  - Peripheral or visceral ischemia
  - Neurological deficit
- Widened mediastinum on chest X -ray
- Risk factors for dissection
- Other
  - Acute aortic regurgitation
  - Pericardial effusion
  - Hemomediastinum/hemothorax

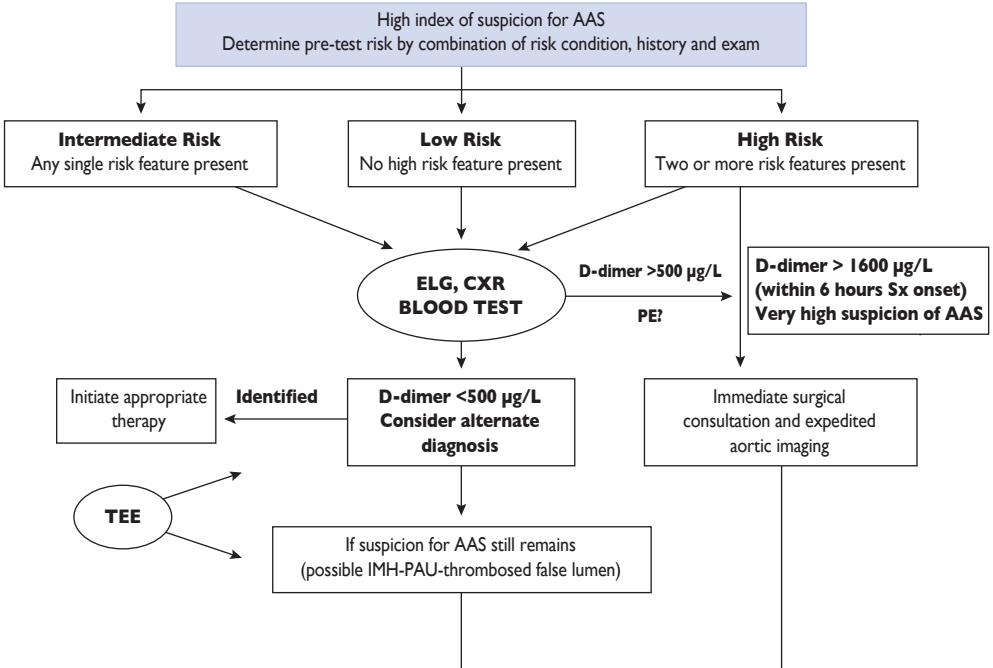
DIFFERENTIAL DIAGNOSIS

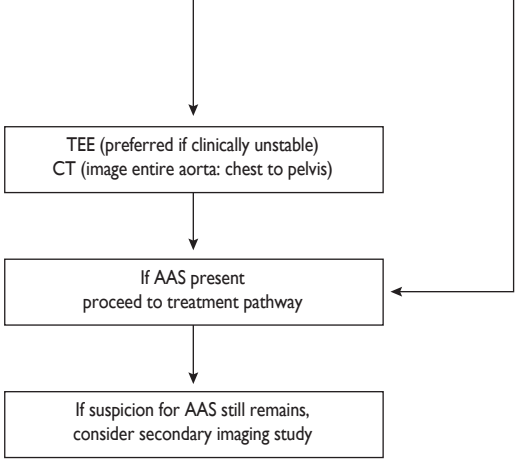
- Acute coronary syndrome (with/without ST-segment elevation)
- Aortic regurgitation without dissection
- Aortic aneurysms without dissection
- Musculoskeletal pain
- Pericarditis
- Pleuritis
- Mediastinal tumours
- Pulmonary embolism
- Cholecystitis
- Atherosclerosis or cholesterol embolism

GENERAL APPROACH TO THE PATIENT WITH SUSPECTED ACUTE AORTIC SYNDROME

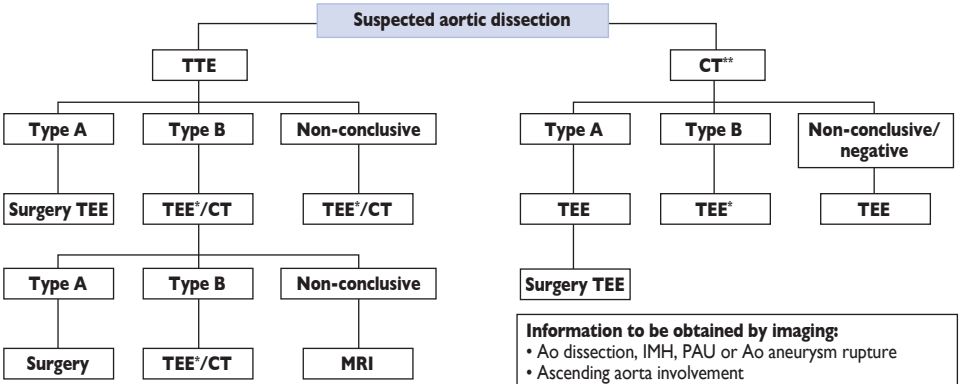


ACUTE AORTIC SYNDROMES: DIAGNOSIS





ACUTE AORTIC SYNDROME: IMAGING DIAGNOSTIC STRATEGY



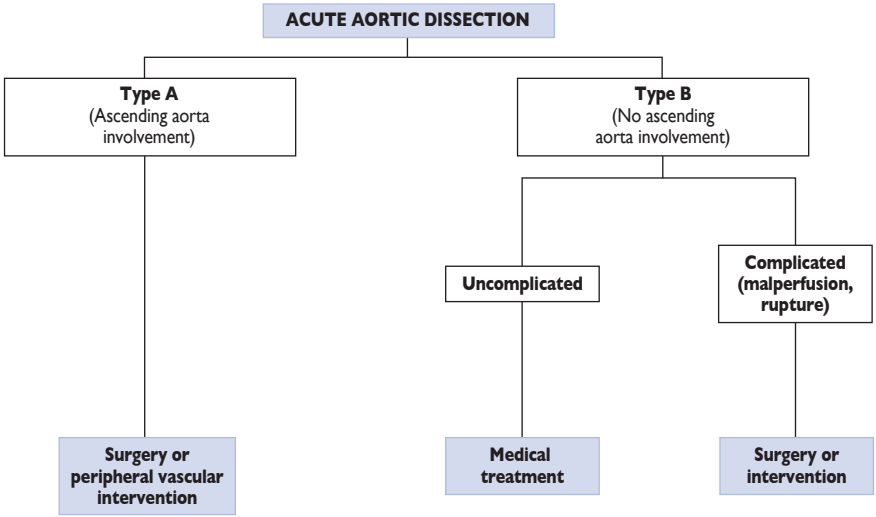
Information to be obtained by imaging:

- Ao dissection, IMH, PAU or Ao aneurysm rupture
- Ascending aorta involvement
- Impending rupture (hemomediastinum or hemothorax)
- Maximum aortic diameter
- Extent of the dissection
- Entry and re-entry sites
- True lumen compression
- Branch-vessel involvement
- Aortic regurgitation
- Pericardial effusion
- Coronary involvement

*Severe hemodynamic instability favours TEE
**Test availability and examiner experience favour CT scan

Reference: Evangelista A et al. Echocardiography in aortic diseases. EAE recommendations for clinical practice. Eur J Echocardiogr (2010);11:645–58.

ACUTE AORTIC SYNDROMES MANAGEMENT: GENERAL APPROACH



ACUTE AORTIC SYNDROMES: INITIAL MANAGEMENT

1 • Detailed **medical history** and complete **physical examination** (when possible)

2 • **Standard 12-lead ECG:** Rule-out ACS, documentation of myocardial ischemia

3 • **Intravenous line, blood sample** (CK, Tn, myoglobin, white blood count, D-dimer, hematocrit, LDH)

4 • **Monitoring:** HR and BP

5 • **Pain relief** (morphine sulphate) (see chapter 3)

6 • **Noninvasive imaging** (see previous page)

7 • **Transfer to ICU**

8 • **MEDICAL TREATMENT: Reduction in SBP**

- Intravenous **ß-blockers** (propranolol, metoprolol, esmolol or labetalol) alone, or in combination with **vasodilators** (sodium nitroprusside or angiotensin-converting enzyme inhibitors) in severe hypertensive states, titrated to achieve SBP between 100 and 120 mmHg

- Intravenous **verapamil or diltiazem** may also be used, particularly if ß-blockers are contraindicated.

9 • **Discuss in heart team** or with vascular surgeon

ACUTE AORTIC SYNDROMES: SURGICAL MANAGEMENT

TYPE A ACUTE AORTIC DISSECTION

URGENT SURGERY (<24h)

Graft replacement of ascending aorta +/- arch with/without aortic valve or aortic root replacement/repair (depending on aortic regurgitation and aortic root involvement)

Emergency Surgery

- Hemodynamic instability (hypotension/shock)
- Tamponade
- Severe acute aortic regurgitation
- Impending rupture
- Flap in aortic root
- Malperfusion syndrome

Elective/individualised Surgery

- Non-complicated intramural hematoma
- Comorbidities
- Age >80 years
- Proximal involvement in upper third of ascending aorta

TYPE B ACUTE AORTIC DISSECTION

Definitive diagnosis

by clinical presentation and imaging

COMPLICATED

defined as:

- Impending rupture
- Malperfusion
- Refractory HTN
- SBP <90 mmHg)
- Shock

UNCOMPLICATED

defined as:

No features of complicated dissection

MEDICAL MANAGEMENT and TEVAR

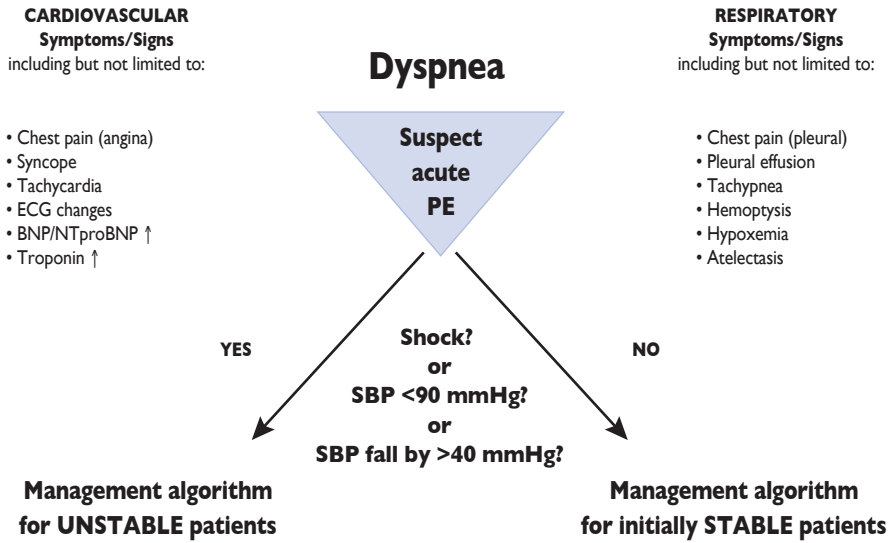
MEDICAL MANAGEMENT and OPEN SURGERY REPAIR

if TEVAR contraindicated

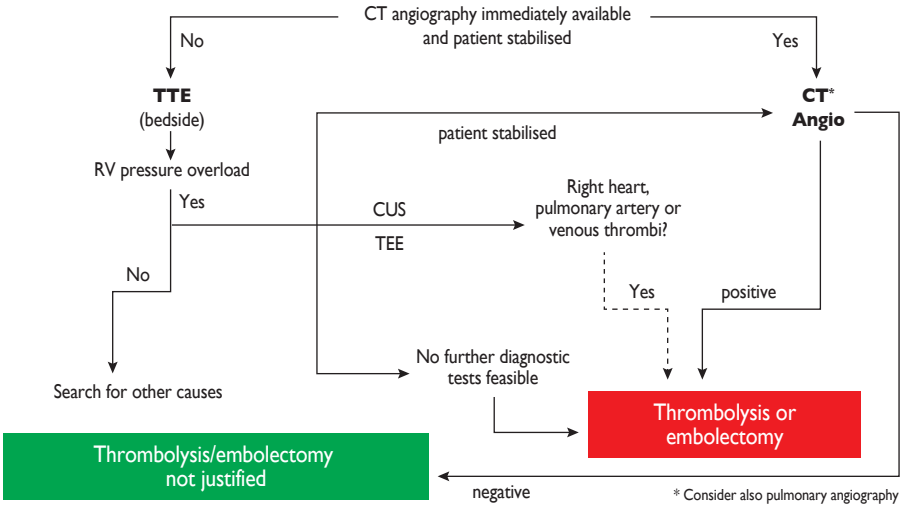
MEDICAL MANAGEMENT and imaging surveillance protocol

- On admission
- At 7 days
- At discharge
- Every 6 months thereafter

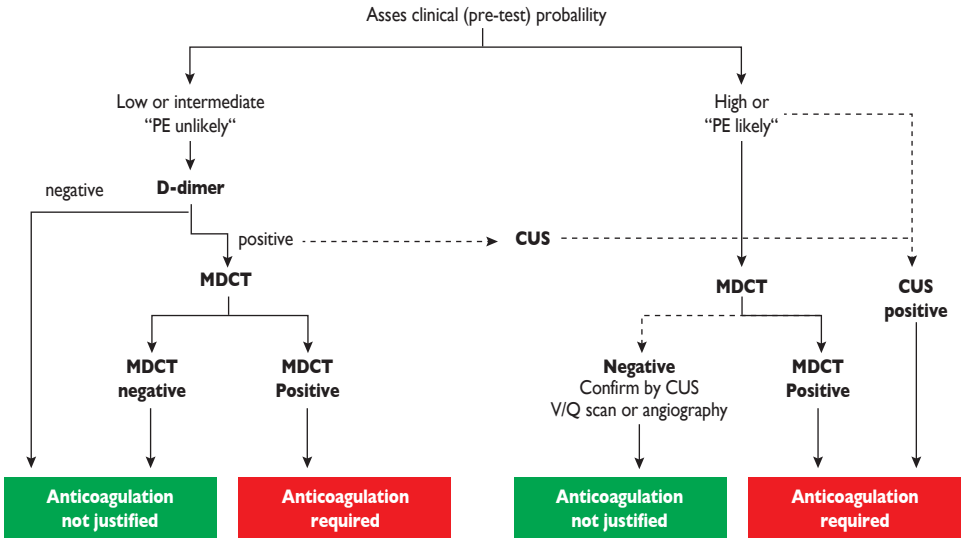
ACUTE PULMONARY EMBOLISM: DIAGNOSIS



MANAGEMENT ALGORITHM FOR UNSTABLE PATIENTS WITH SUSPECTED ACUTE PULMONARY EMBOLISM



MANAGEMENT ALGORITHM FOR INITIALLY STABLE PATIENTS WITH SUSPECTED ACUTE PULMONARY EMBOLISM



MANAGEMENT STRATEGY FOR INITIALLY STABLE PATIENTS WITH CONFIRMED NON-HIGH RISK PULMONARY EMBOLISM

Markers for myocardial injury	Positive*	Positive**	Negative
Markers for RV overload	Positive*	Positive**	Negative
Clinical risk assessment score	Positive*	Positive**	Negative
Preferred initial anticoagulation	**i.v. UFH/LMWH**	**LMWH/Fonda/NOAC**	**LMWH/Fonda/NOAC**

STRATEGY	**ICC monitoring Rescue thrombolysis*** **	**Hospitalisation telemonitoring**	**Early discharge**

* If all three positive
** if any of the three positive
*** Early thrombolysis may prevent hemodynamic decompensation but its use should be considered with great caution as it increases the risk of major hemorrhage and stroke

MANAGEMENT STRATEGY FOR INITIALLY UNSTABLE PATIENTS WITH CONFIRMED HIGH-RISK PULMONARY EMBOLISM

Shock or hypotension	**YES**			
Recent intracerebral hemorrhage	NO	YES		
Recent surgery or major bleed	NO		YES	
Right heart floating thrombus	NO or small and limited to right heart	NO	NO	YES, particularly if large or protruding via patent foramen ovale

STRATEGY	**Thrombolysis, surgical or percutaneous catheter embolectomy**	**Surgical or Percutaneous catheter embolectomy (availability/experience)**	**Surgical embolectomy**

i.v. UFH, STABILISE SYSTEMIC BLOOD PRESSURE, CORRECT HYPOXEMIA

Key drugs for initial treatment of patients with confirmed PE

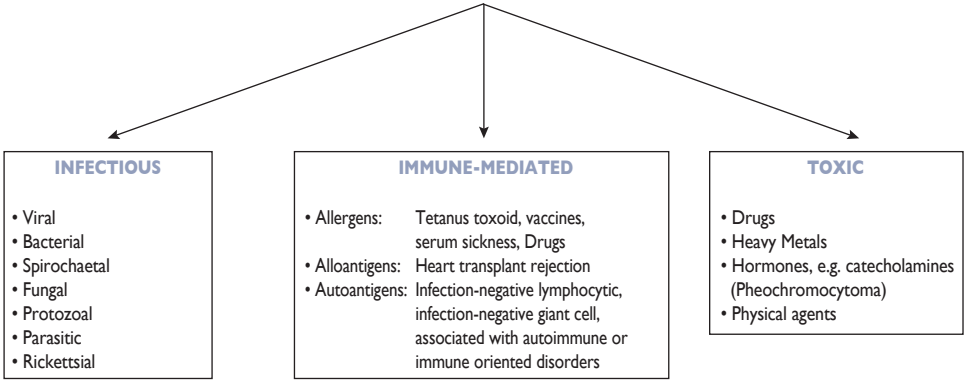
Unstable	Alteplase (rtPA) (intravenous)	100 mg/2 h or 0.6 mg/kg/15 min (max 50 mg)
	Urokinase (intravenous)	3 million IU over 2 h
	Streptokinase (intravenous)	1.5 million IU over 2 h
	Unfractionated heparin (intravenous)	80 IU/kg bolus + 18 IU/kg/h
Stable	Enoxaparine (subcutaneous)	1.0 mg/kg BID or 1.5 mg/kg QD
	Tinzaparin (subcutaneous)	175 U/kg QD
	Fondaparinux (subcutaneous)	7.5 mg (50-100 Kg of body weight) 5 mg for patients <50 kg, 10 mg for patients >100 kg
	Rivaroxaban (oral)	15 mg BID (for 3 weeks, then 20 mg QD)
	Other new oral anticoagulants	Pending approval for pulmonary embolism

Chapter 7
ACUTE MYOCARDIAL / PERICARDIAL SYNDROMES

ACUTE MYOCARDITIS: DEFINITION AND CAUSES

MYOCARDITIS (WHO /ISFC): Inflammatory disease of the myocardium diagnosed by established histological, immunological and immunohistochemical criteria.

CAUSES OF MYOCARDITIS



ACUTE MYOCARDITIS: DIAGNOSTIC CRITERIA (I)
DIAGNOSTIC CRITERIA FOR CLINICALLY SUSPECTED MYOCARDITIS

CLINICAL PRESENTATIONS
with or without ancillary findings

- Acute chest pain (pericarditic or pseudo-ischemic)
- New-onset (days up to 3 months) or worsening dyspnea or fatigue, with or without left/right heart failure signs
- Palpitation, unexplained arrhythmia symptoms, syncope, aborted sudden cardiac death
- Unexplained cardiogenic shock and/or pulmonary oedema

ANCILLARY FINDINGS
which support the clinical suspicion of myocarditis

- Fever ≥38.0°C within the preceding 30 days
- A respiratory or gastrointestinal infection
- Previous clinically suspected or biopsy proven myocarditis
- Peri-partum period
- Personal and/or family history of allergic asthma
- Other types of allergy
- Extra-cardiac autoimmune disease
- Toxic agents
- Family history of dilated cardiomyopathy, myocarditis

DIAGNOSTIC CRITERIA

I. **ECG/Holter/stress test features:** Newly abnormal ECG and/or Holter and/or stress testing, any of the following:

- I to III degree atrioventricular block, or bundle branch block, ST/T wave changes (ST elevation or non ST elevation, T wave inversion),
- Sinus arrest, ventricular tachycardia or fibrillation and asystole, atrial fibrillation, frequent premature beats, supraventricular tachycardia
- Reduced R wave height, intraventricular conduction delay (widened QRS complex), abnormal Q waves, low voltage

II. **Myocardiocytolysis markers:** Elevated TnT/TnI

III. **Functional/structural abnormalities on echocardiography**

- New, otherwise unexplained LV and/or RV structure and function abnormality (including incidental finding in apparently asymptomatic subjects): regional wall motion or global systolic or diastolic function abnormality, with or without ventricular dilatation, with or without increased wall thickness, with or without pericardial effusion, with or without endocavitary thrombi

IV. **Tissue characterisation by CMR:** Edema and/or LGE of classical myocarditic pattern

Reference: Caforio ALP et al. Eur Heart J. (2013) Jul 3 (15)

ACUTE MYOCARDITIS: DIAGNOSTIC CRITERIA (2)

Acute myocarditis should be clinically suspected in the presence of:

1 or more of the clinical presentations shown in the Diagnostic Criteria*
with or without Ancillary Features*
AND
1 or more Diagnostic Criteria from different categories (I to IV)*

OR

when the patient is asymptomatic, 2 or more diagnostic criteria from different categories (I to IV)*

in the absence of:
1) angiographically detectable coronary artery disease
2) known pre-existing cardiovascular disease or extra-cardiac causes that could explain the syndrome (e.g. valve disease, congenital heart disease, hyperthyroidism, etc.)

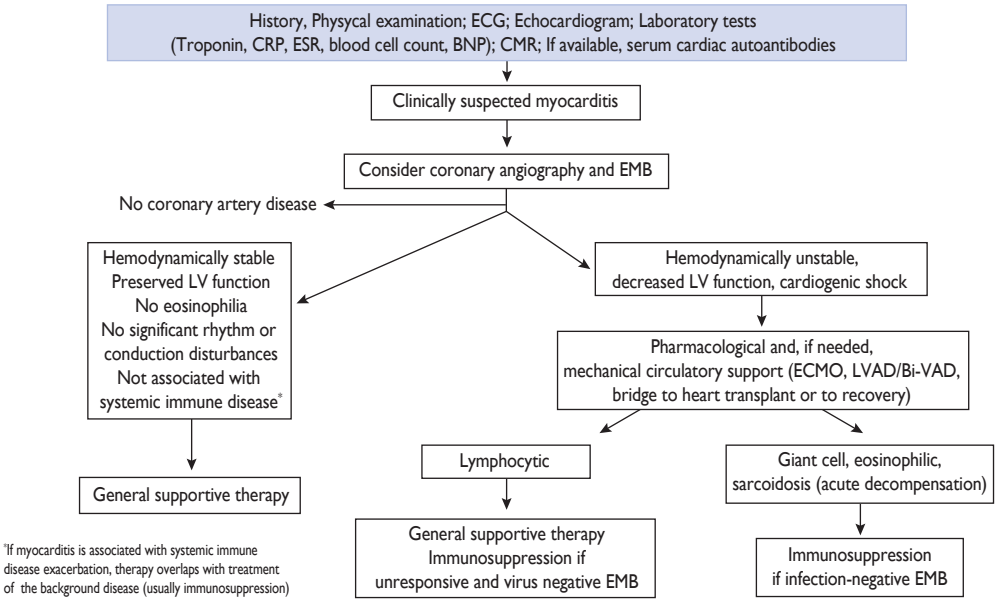
Suspicion is higher with higher number of fulfilled criteria

* See Diagnostic Criteria and Ancillary Features *(see page 105)*

Endomyocardial biopsy is necessary to: 1) confirm the diagnosis of clinically suspected myocarditis, 2) identify the type and aetiology of inflammation, and 3) provide the basis for safe immunosuppression (in virus negative cases).

Reference: Caforio ALP et al. Eur Heart J. (2013) Jul 3 (16)

ACUTE MYOCARDITIS: DIAGNOSTIC AND MANAGEMENT PROTOCOL



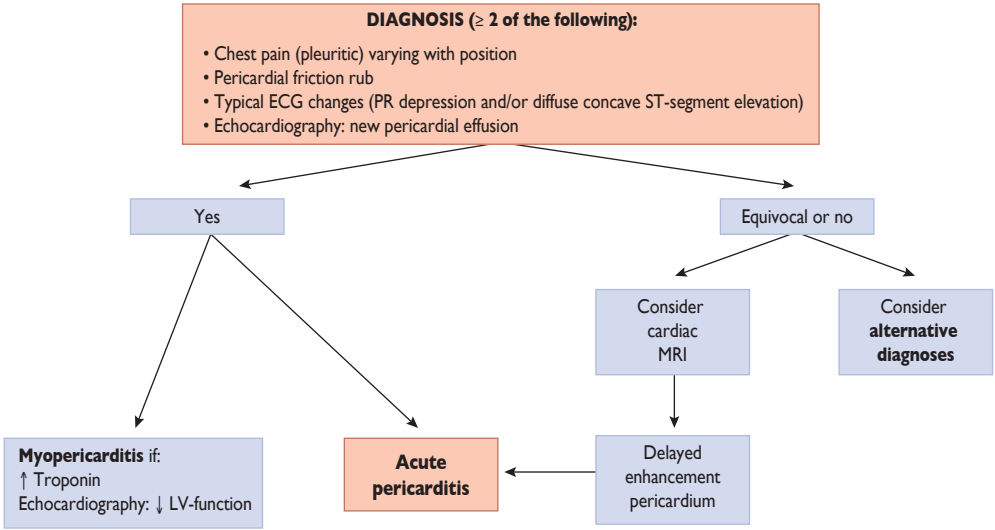
*If myocarditis is associated with systemic immune disease exacerbation, therapy overlaps with treatment of the background disease (usually immunosuppression)

MANAGEMENT OF PATIENTS WITH LIFE-THREATENING ACUTE MYOCARDITIS

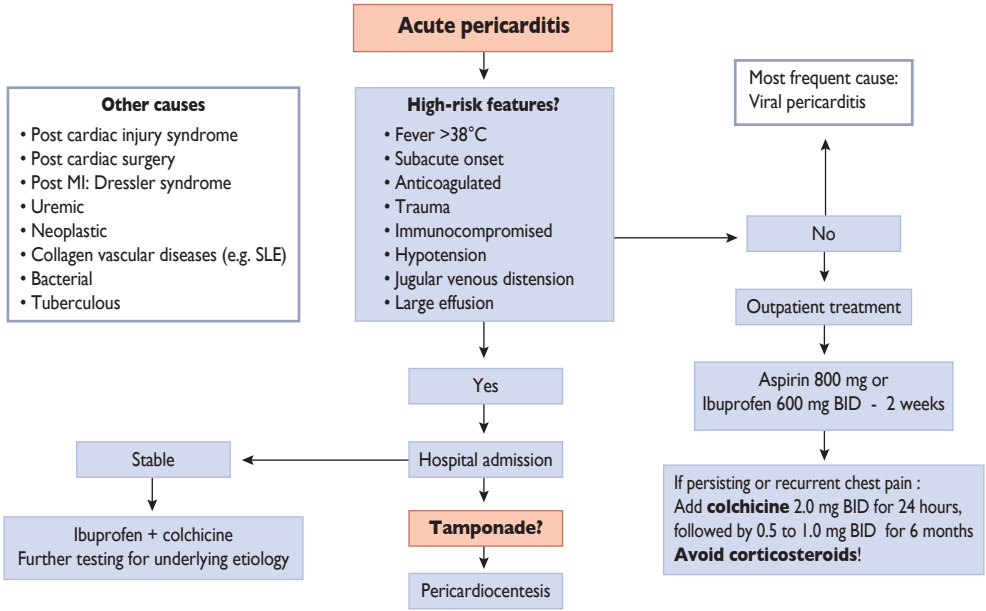
- Patients with a life-threatening presentation should be sent to specialised units with capability for hemodynamic monitoring, cardiac catheterisation and expertise in endomyocardial biopsy.
- In patients with hemodynamic instability a **mechanical cardio-pulmonary assist device** may be needed as a bridge to recovery or to heart transplantation.
- **Heart transplant** should be deferred in the acute phase, because recovery may occur, but can be considered for hemodynamically unstable myocarditis patients, including those with giant cell myocarditis, if optimal pharmacological support and mechanical assistance cannot stabilise the patient
- **ICD implantation** for complex arrhythmias should be deferred until resolution of the acute episode, with possible use of a lifevest during the recovery period.

Reference: Caforio ALP et al. Eur Heart J. 2013 Jul 3 (18)

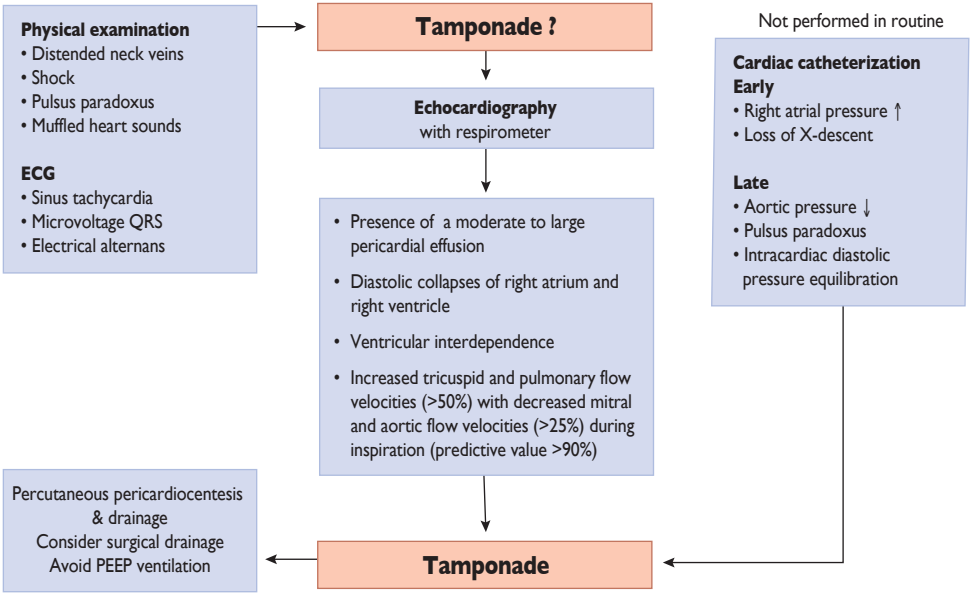
ACUTE PERICARDITIS: DIAGNOSIS



ACUTE PERICARDITIS: MANAGEMENT



CARDIAC TAMPONADE: DIAGNOSIS AND MANAGEMENT



Abbreviations

APTT = Activated partial thromboplastin time
AB = Airway and breathing
ABG = Arterial blood gas
AADs = Antiarrhythmic drugs
AAS = Acute aortic syndrome
ACEI = Angiotensin converting enzyme inhibitor
ACLS = Advanced cardiovascular life support
ACS = Acute coronary syndrome
AED = Automated external defibrillator
AF = Atrial fibrillation
Ao = Aortic
ARB = Angiotensin receptor blockers
AS = Aortic stenosis
AV = Atrioventricular
AVN = Atrioventricular node
AVNRT = Atrioventricular nodal re-entrant tachycardia
AVNT = Atrioventricular nodal tachycardia
BID = Twice a day
BBB = Bundle branch block
BLS = Basic life support
BNP = Brain natriuretic peptide
BP = Blood pressure
CABG = Coronary artery bypass grafting
CAD = Coronary artery disease
Cath Lab = Catheterisation laboratory
CCU = Coronary care unit
CHF = Congestive heart failure
CMR = Cardiovascular magnetic resonance
COPD = Chronic obstructive pulmonary disease
CPAP = Continuous positive airway pressure
CPR = Cardiopulmonary resuscitation
CS = Cardiogenic shock
CSM = Carotid sinus massage
CSNRT = Corrected sinus node recovery time
CSS = Carotid sinus syndrome
CT = Computed tomography
CT-angio = Computed tomography angiography
CUS = Compression venous ultrasound
CV = Cardiovascular
CXR = Chest X-ray
DD = Dyastolic dysfunction
DM = Diabetes mellitus
DVT = Deep venous thrombosis
ECG = Electrocardiogram
ED = Emergency department
EG = Electrograms

Abbreviations

EMB = Endomyocardial biopsy
EMS = Emergency medical services
EPS = Electrophysiological study
ERC = European Resuscitation Council
ESR = Erythrocyte sedimentation rate
ETT = Exercice treadmill testing
FMC = First medical contact
GER = Gastroesophageal reflux
GFR = Glomerular flow rate
GI = Gastrointestinal
GP = Glycoprotein
HTN = Hypertension
HR = Heart rate
hsTn = High-sensitive troponin
IABP = Intra-aortic balloon pump
ICC = Intensive cardiac care
ICCU = Intensive cardiac care unit
ICD = Implantable cardioverter defibrillator
IHD = Ischemic heart disease
IMH = Intramural hematoma
ISFC = International Society and Federation of Cardiology
i.o. = Intraosseous
IV = Invasive ventilation
i.v. = Intravenous
KD = Kidney disease
LBBB = Left bundle branch block
LD = Loading dose
LGE = Late gadolinium enhancement
LMWH = Low-molecular weight heparin
LOC = Loss of consciousness
LV = Left ventricular
LVEF = Left ventricular ejection fraction
LVH = Left ventricular hypertrophy
LVSD = Left ventricular systolic dysfunction
MCS = Mechanical circulatory support
MDCT = Computed tomography with >4 elements
MI = Myocardial infarction
MRI = Magnetic resonance imaging
Mvo = Microvascular obstruction
NIV = Non-invasive ventilation
NOAC = New oral anticoagulants
NSAID = Non-steroidal anti-inflammatory drugs
NSTEACS = Non-ST-elevation ACS
NSTEMI = Non ST-segment elevation myocardial infarction
NTG = Nitroglycerin
NT-proBNP = N-terminal pro brain natriuretic peptide

Abbreviations

NYHA = New York Heart Association
OH = Orthostatic hypotension
PAU = Penetrating aortic ulcer
PCI = Percutaneous coronary intervention
PCM = Physical counter-measures
PE = Pulmonary embolism
PEA = Pulmonary endarterectomy
PEEP = Positive end expiratory pressure
PR = Pulmonary regurgitation
ProCT = Procalcitonin
PRN = Pro re nata
QD = Once a day
rtPA = Recombinant tissue plasminogen activator
RV = Right ventricular
SBP = Systemic blood pressure
s.c = Subcutaneous
SLE = Systemic lupus erythematosus
SMU = Syncope management units
STEMI = ST-segment elevation myocardial infarction
SVT = Supraventricular tachycardia
SpO_2 = Oxygen saturation
TEE = Transesophageal echocardiography
TEVAR = Thoracic endovascular aortic aneurysm repair
TIA = Transient ischemic attack
TLOC = Transient loss of consciousness
Tn = Troponin
TSH = Thyroid-stimulating hormone
TTE = Transthoracic echocardiography
UFH = Unfractionated heparin
ULN = Upper limit of normal
VF = Ventricular fibrillation
VT = Ventricular tachycardia
VVS = Vasovagal syncope
WHO = World Health Organization
WPW = Wolff-Parkinson-White

NOTES

NOTES

ESC Guidelines Acknowledgements

(1) (2) (3) (4) Moya A, Sutton R, Ammirati F, Blanc JJ, Brignole M, Dahm JB, et al. Guidelines for the diagnosis and management of syncope (version 2009). The Task Force for the Diagnosis and Management of Syncope of the European Society of Cardiology (ESC) Eur Heart J (2009);30:2631-2671 doi:10.1093/eurheartj/ehp298. Reproduced with permission of Oxford University Press (UK) © European Society of Cardiology, www.escardio.org/guidelines

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Acute Cardiovascular Care Association
Clinical Decision-Making
Toolkit

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