Acute Cardiovascular Care Association
Clinical Decision-Making
Toolkit

www.escardio.org/ACCA
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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

1.1 Chest Pain
1.2 Dyspnea
1.3 Syncope
### FACTORS TO BE CONSIDERED IN THE EVALUATION AFTER THE FIRST CALL FOR CHEST PAIN

<table>
<thead>
<tr>
<th>FIRST CALL FOR CHEST PAIN</th>
<th>Higher risk / probability</th>
<th>Lower risk / probability</th>
</tr>
</thead>
</table>
| **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

Arguments for vital risk? (see page 2)

- Yes
- No

Emergency transport
with trained medical team

Emergency care:
Resuscitation, hemodynamic or
rhythm restoration (see chapter 4)

Origin of Chest Pain?

Acute Cardiac Disease

- High probability for ACS
  - ECG, decision for reperfusion,
antithrombotics, immediate
  transport to ED/cathlab
  (see chapter 2)

- Low probability for ACS
  - Emergency transport

No Acute Cardiac Disease

- Emergency transport

Hospital admission to the
Emergency Department

Cardiology
ward
Non-cardiology
ward
Discharge after
prolonged observation
FACTORS TO BE CONSIDERED IN THE EVALUATION DURING THE FIRST MEDICAL CONTACT FOR CHEST PAIN

<table>
<thead>
<tr>
<th>FIRST MEDICAL CONTACT</th>
<th>Higher risk / probability</th>
<th>Lower risk / probability</th>
</tr>
</thead>
</table>
| **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 SpO₂, 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** | • 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 |

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

Hemodynamic, respiratory or neurological distress? (see page 4)

Yes

Resuscitation, hemodynamic or respiratory support (see chapters 3 & 4)

No

ECG <10 min → ACS?

High probability

ST-segment elevation

Type of reperfusion (primary PCI or fibrinolysis)

Record times (onset, call, contact)

Start antiplatelet and anticoagulant treatment

Transfer to a center with cath-lab

Low probability

No ST-segment elevation but other ECG changes or persistent pain

Suspect ACS

Uncertain diagnosis

No antithrombotic treatment

Transfer to a proximity center (with or without cath-lab)

No non cardiovascular disease?

• Sepsis
• Acute respiratory distress
• GI disease, bleeding, others

Acute cardiovascular disease other than ACS?

• Acute aortic syndrome (see chapter 6)
• Pulmonary embolism (see chapter 6)
• Acute pericarditis (see chapter 7)
• Acute heart failure (see chapter 3)
Diagnosis of NSTEACS (see chapter 2)
Acute aortic syndrome (see chapter 6)
Acute pulmonary embolism (see chapter 6)
Acute pericarditis (see chapter 7)
Acute heart failure (see chapter 3)
Aortic stenosis, hyperthrophic cardiomyopathy
Acute gastro-oesophageal disease
Acute pleuro-pulmonary disease
Acute psychogenic disorders

Repeat clinical and ECG examination
Laboratory: Tn, renal function, Hb, D-dimers
Imaging: TTE, CT scan
Diagnostic coronary angiography
DYSPNEA: DIFFERENTIAL DIAGNOSIS

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

Basic measures
- BP, HR, respiratory rate, SpO2 & temperature
- Start oxygen to target SpO2 94-98%
- Start i.v. line & monitor patient

Criteria for transfer to ICU (despite treatment for 30 minutes)
- Respiratory rate >35/min
- SBP <90 mmHg
- SpO2 <85%
- HR >120 bpm

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

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

Acute heart failure
Acute coronary syndrome
Pneumonia
Exacerbated COPD or other chronic lung disease
Pulmonary embolism

* Defined as ≥1 criterion:
  - Respiratory rate ≥25/min
  - PaO2 ≤75 mmHg
  - SpO2 ≤92% in ambient air
  - PaCO2 ≥45 mmHg with arterial pH ≤7.35

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
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)
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- 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%
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  - 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

Unstable after 30 minutes
- CCU/ICU transfer

Stable after 30 minutes
- Ward transfer

DYSPNEA: ACUTE PULMONARY EMBOLISM (see chapter 6.2)


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

Right ventricular dysfunction

Hemodynamically stable

Wells criteria for PE:

- 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

Score
DYSPNEA: ACUTE PULMONARY EMBOLISM (see chapter 6.2)


Intermediate probability
Total score 2-6

High probability
Total score >6

Outpatient management possible?
→ Risk stratification
(see chapter 6.2)

Low probability
Total score <2

PE confirmed: Treatment
(see chapter 6.2)

Immediate TTE (if available)
PE confirmed: Treatment
(see chapter 6.2)

Result
inconclusive
→ CT-angio

Right ventricular dysfunction

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

ABG, ECG, chest X-ray plus clinical assessment of PE probability (risk factors) plus monitoring
**DYSPNEA: COPD EXACERBATION**

- Verify diagnosis (DD: PE, acute heart failure, pneumothorax)
- Oxygen administration → SpO₂ target 88-92% (Beware of carbonarcosis: ABC after 1 h)

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

- COPD classification (GOLD)
- Etiology

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

- Hospitalization indicated?
- Evaluate ICU criteria
- NIV indicated?

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

- 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

Copyright: Leuppi JD et al. JAMA. 2013 Jun 5;309(21):2223-31
DYSPNEA: COMMUNITY-ACQUIRED PNEUMONIA

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

Definition

- Chest X-ray
- Laboratory workup
- Sputum
- Blood cultures (2x2)
- Legionella antigen (urine)
- Pneumococcus antigen (urine)

if dyspnea & cough
clinical chemistry; BGA; procalcitonin
if patient admitted
if patient admitted
if Legionellosis suspected
if no other pathogen isolated

Risk stratification → manageable on an outpatient basis?
- Pneumonia Severity Index
- CURB-65

- Treatment; procalcitonin guided treatment
- Consider outpatient treatment where PSI I-III or CURB65 0 or 1
- Minimum 5-day course of treatment and afebrile for 48-72 h, 7-10 days, 14 days where intracellular organisms (e.g. Legionella) are present

Complications

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.

SYNCOPE: DIAGNOSTIC CRITERIA (1)
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.

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.

Patients with suspected syncope presenting to ED or clinic

"Uncertain" or unexplained syncope

High risk

Observation Unit
Home if stable,
Admit to hospital
if evidence of high risk

Hospital admission
Inpatient SMU

Intermediate risk

Low risk

Home
Outpatient SMU referral

Outpatient SMU
for diagnosis, treatment
and follow-up as appropriate

Certain diagnosis of syncope

Initiate therapy
Inpatient SMU, outpatient SMU or
personal physician as appropriate

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

<table>
<thead>
<tr>
<th>CAROTID SINUS MASSAGE</th>
<th>ORTHOSTATIC HYPOTENSION</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Indications</strong></td>
<td></td>
</tr>
<tr>
<td>• CSM is indicated in patients &gt;40 years with syncope of unknown aetiology after initial evaluation;</td>
<td></td>
</tr>
<tr>
<td>• 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).</td>
<td></td>
</tr>
<tr>
<td><strong>Diagnostic criteria</strong></td>
<td></td>
</tr>
<tr>
<td>• CSM is diagnostic if syncope is reproduced in presence of asystole longer than 6 s and/or a fall in systolic BP &gt;50 mmHg.</td>
<td></td>
</tr>
<tr>
<td><strong>Recommendations: Active standing</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Indications</strong></td>
<td></td>
</tr>
<tr>
<td>• Manual intermittent determination with sphygmomanometer of BP supine and, when OH is suspected, during active standing for 3 min is indicated as initial evaluation;</td>
<td></td>
</tr>
<tr>
<td>• Continuous beat-to-beat non-invasive pressure measurement may be helpful in cases of doubt.</td>
<td></td>
</tr>
<tr>
<td><strong>Diagnostic criteria</strong></td>
<td></td>
</tr>
<tr>
<td>• 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 &lt;90 mmHg;</td>
<td></td>
</tr>
<tr>
<td>• The test should be considered diagnostic when there is an asymptomatic fall in systolic BP from baseline value ≥20 mmHg or diastolic BP &gt;10 mmHg or a decrease in systolic BP to &lt;90 mmHg.</td>
<td></td>
</tr>
</tbody>
</table>

### TREATMENT ACCORDING TO TYPE OF SYNCOPE (I)

#### Treatment of reflex syncope

- 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

#### Treatment of orthostatic hypotension

- 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

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## 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

Chapter 2
ACUTE CORONARY SYNDROMES

2.1 General concepts

2.2 Non ST-segment elevation ACS

2.3 ST-segment elevation MI (STEMI)
ACUTE CORONARY SYNDROMES: DIAGNOSIS

CHEST PAIN
or symptoms suggestive of myocardial ischemia

ECG

ST elevation (persistent)

LBBB

ST/T abnormalities

Normal ECG

Pain resolves with nitroglycerin

1st hsTn

hsTn > ULN

hsTn < ULN

Pain onset < 6h

Pain onset > 6h

2nd hsTn (3 h)

↑ hsTn

No ↑ hsTn

Clinical diagnosis clear

Potential noncardiac causes for abnormal Tn

STEMI

NSTEMI

Unstable Angina

Consider No ACS

Consider STEMI
# Acute Coronary Syndromes: Differential Diagnosis

## Causes of Chest Pain Not Related to ACS

<table>
<thead>
<tr>
<th>PRIMARY CARDIOVASCULAR</th>
<th>PRIMARY CARDIOVASCULAR</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Acute pericarditis, pericardial effusion</td>
<td>• Acute myo(peri)carditis</td>
</tr>
<tr>
<td>• Acute myocarditis</td>
<td>• Severe hypertensive crisis</td>
</tr>
<tr>
<td>• Severe hypertensive crisis</td>
<td>• Pulmonary edema or severe congestive heart failure</td>
</tr>
<tr>
<td>• Stress cardiomyopathy (Tako-Tsubo syndrome)</td>
<td>• Stress cardiomyopathy (Tako-Tsubo syndrome)</td>
</tr>
<tr>
<td>• Hypertrophic cardiomyopathy, aortic stenosis</td>
<td>• Post-tachy- or bradyarrhythmias</td>
</tr>
<tr>
<td>• Severe acute heart failure</td>
<td>• Cardiac contusion, ablation, pacing, cardioversion, or endomyocardial biopsy</td>
</tr>
<tr>
<td>• Acute aortic syndrome (dissection, hematoma)</td>
<td>• Aortic dissection, aortic valve disease or hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td>• Pulmonary embolism, pulmonary infarction</td>
<td>• Pulmonary embolism, severe pulmonary hypertension</td>
</tr>
<tr>
<td>• Cardiac contusion</td>
<td></td>
</tr>
</tbody>
</table>

## Causes of Troponin Elevation Not Related to ACS

<table>
<thead>
<tr>
<th>PRIMARY NON-CARDIOVASCULAR</th>
<th>PRIMARY NON-CARDIOVASCULAR</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Oesophageal spasm, oesophagitis, GER</td>
<td>• Renal dysfunction (acute or chronic)</td>
</tr>
<tr>
<td>• Peptic ulcer disease, cholecystitis, pancreatitis</td>
<td>• Critical illness (sepsis, respiratory failure…)</td>
</tr>
<tr>
<td>• Pneumonia, bronchitis, asthma attack</td>
<td>• Acute neurological damage (i.e. stroke, subarachnoid hemorrhage)</td>
</tr>
<tr>
<td>• Pleuritis, pleural effusion, pneumothorax</td>
<td>• Severe burns (affecting &gt;30% of body surface area)</td>
</tr>
<tr>
<td>• Pulmonary embolism, severe pulmonary hypertension</td>
<td>• Rhabdomyolysis</td>
</tr>
<tr>
<td>• Thoracic trauma</td>
<td>• Drug toxicity (chemotherapy with adriamycin, 5-fluorouracil, herceptin, snake venoms…)</td>
</tr>
<tr>
<td>• Costochondritis, rib fracture</td>
<td>• Inflammatory or degenerative muscle diseases</td>
</tr>
<tr>
<td>• Cervical / thoracic vertebral or discal damage</td>
<td>• Hypothyroidism</td>
</tr>
<tr>
<td>• Herpes Zoster</td>
<td>• Infiltrative diseases (amyloidosis, hemochromatosis, sarcoidosis)</td>
</tr>
<tr>
<td></td>
<td>• Scleroderma</td>
</tr>
</tbody>
</table>
### ACUTE CORONARY SYNDROMES: DIFFERENTIAL DIAGNOSIS (2)
CAUSES OF REPOLARISATION ABNORMALITIES IN THE ECG NOT RELATED TO ACS

<table>
<thead>
<tr>
<th>ST-SEGMENT ELEVATION</th>
<th>NEGATIVE T WAVES</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fixed</strong></td>
<td></td>
</tr>
<tr>
<td>LV aneurysm</td>
<td>Normal variants, i.e. women (right precordial leads), children, teenagers</td>
</tr>
<tr>
<td>LBBB, WPW, hypertrophic cardiomyopathy, LVH</td>
<td>Evolutive changes post MI</td>
</tr>
<tr>
<td>Pacemaker stimulation</td>
<td>Chronic IHD</td>
</tr>
<tr>
<td>Early repolarisation (elevated J-point)</td>
<td>Acute (myo)pericarditis, cardiomyopathies</td>
</tr>
<tr>
<td><strong>Dynamic</strong></td>
<td></td>
</tr>
<tr>
<td>Acute (myo)pericarditis</td>
<td>BBB, LVH, WPW</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>Post-tachycardia or pacemaker stimulation</td>
</tr>
<tr>
<td>Electrolytic disturbances (hyperkaliemia)</td>
<td>Metabolic or ionic disturbances</td>
</tr>
<tr>
<td>Acute brain damage (stroke, subarachnoid haemorrhage)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ST-SEGMENT DEPRESSION</th>
<th>PROMINENT T WAVES</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fixed</strong></td>
<td></td>
</tr>
<tr>
<td>Abnormal QRS (LBBB, WPW, pacemaker stimulation...)</td>
<td>Normal variants, i.e. early repolarisation</td>
</tr>
<tr>
<td>LVH, hypertrophic cardiomyopathy</td>
<td>Metabolic or ionic disturbances (i.e. hyperkalemia)</td>
</tr>
<tr>
<td>Chronic IHD</td>
<td>Acute neurological damage (stroke, subarachnoid haemorrhage)</td>
</tr>
<tr>
<td><strong>Dynamic</strong></td>
<td></td>
</tr>
<tr>
<td>Acute (myo)pericarditis</td>
<td>Severe hypertensive crisis</td>
</tr>
<tr>
<td>Acute pulmonary hypertension</td>
<td>Drug effects (digoxin)</td>
</tr>
<tr>
<td>Electrolytic disturbances (hyperkalemia)</td>
<td>Shock, pancreatitis</td>
</tr>
<tr>
<td>Intermittent LBBB, WPW, pacing</td>
<td>Hyperventilation</td>
</tr>
<tr>
<td>Post-tachycardia / cardioversion</td>
<td></td>
</tr>
</tbody>
</table>
GENERAL APPROACH TO THE PATIENT WITH CHEST PAIN / SUSPECTED ACS

1. Clinical Evaluation
   - Quality of chest pain
   - Clinical context
   - Probability of CAD
   - Physical examination

2. ECG (<10 min)
   - STEMI: See chapter 2.3

3. Diagnosis / Risk assessment
   - NSTE ACS: See chapter 2.2
     - Clinical presentation (BP, HR)
     - ECG presentation
     - Past history
     - Ischemic risk (i.e. GRACE, TIMI scores)
     - Bleeding risk (i.e. CRUSADE score)
     - Additional information (labs, imaging...) optional

4. Medical Treatment
   - Thrombolysis
     - For STEMI if primary PCI not timely available
   - Anti-ischemic therapy
   - Antiplatelet therapy
   - Anticoagulation

5. Invasive Strategy
   - Primary PCI
   - Emergent <2 hours
   - Urgent 2-24 hours
   - Early 24-72 hours
   - No / Elective

* 3-12 hours after thrombolysis

## 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

![Probability of all-cause mortality from hospital discharge to 6 months (%)](image)

#### TIMI RISK SCORE

**Predictive Factors**
- Age
- 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

![Risk of 14-day events (%)](image)
# NON ST-SEGMENT ELEVATION ACS: RISK STRATIFICATION (Cont.)

## 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](http://www.crusadebleedingscore.org)

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**Copyrights**


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

**Initial treatment**
- Nitrates
- Morphine
- Oxygen (if SatO₂ < 95%)

**Pharmacological treatment**

**Antithrombotic therapy**

**Anticoagulation**
One of the following:
- Fondaparinux
- Enoxaparin
- UFH

**Antiplatelets**
Aspirin + one of:
- Ticagrelor
- Prasugrel
- Clopidogrel

**Anti ischemic treatment**
- Nitrates
- Beta-blockers
- Calcium antagonists

**Other preventive therapies**
- Statins
- ACE inh. (or ARB)
- Aldosteron inh.

**Myocardial revascularization**
- PCI
- CABG
## Non ST-Segment Elevation ACS: Treatment (2)
### Dosing and Recommendations for Pharmacological Therapies

<table>
<thead>
<tr>
<th>Drug</th>
<th>Loading dose</th>
<th>Maintenance dose</th>
<th>Considerations</th>
<th>Major contraindications (in addition to specific allergies)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>INITIAL TREATMENT</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nitrates</td>
<td>2–3 mg i.v.</td>
<td></td>
<td></td>
<td>Severe hypotension</td>
</tr>
<tr>
<td>Morphine</td>
<td>2–3 mg i.v.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oxygen</td>
<td>1–2 puffs s.l.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2–3 mg i.v.</td>
<td>_</td>
<td>If pain refractory to nitrates</td>
<td>Only if SpO₂ &lt;95%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FiO₂ needed for SpO₂ &gt;95%</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ANTITHROMBOTIC THERAPY</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>300 mg oral</td>
<td>75–150 mg QD</td>
<td>Preferred in moderate-high risk patients at diagnosis</td>
<td>Previous intracerebral hemorrhage</td>
</tr>
<tr>
<td>Ticagrelor</td>
<td>180 mg oral</td>
<td>90 mg BID</td>
<td>Preferred in clopi-naive with CAD and PCI planned</td>
<td>Previous stroke/TIA</td>
</tr>
<tr>
<td>Prasugrel</td>
<td>60 mg oral</td>
<td>10 mg QD</td>
<td>Preferred if no immediate cath</td>
<td>Weight &lt;60 kg, Age &gt;75 years*</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>300–600 mg oral</td>
<td>75 mg QD</td>
<td>If tica/prasu not available</td>
<td></td>
</tr>
<tr>
<td>Fondaparinux</td>
<td>2.5 mg s.c.</td>
<td>2.5 mg s.c. QD</td>
<td>Preferred if no immediate cath</td>
<td></td>
</tr>
<tr>
<td>Enoxaparin</td>
<td>30 mg i.v. +</td>
<td>1 mg/Kg/BID</td>
<td>If &gt;75 years, No LD and MD 0.75 mg/Kg/12 h</td>
<td></td>
</tr>
<tr>
<td>UFH</td>
<td>1 mg/kg s.c.</td>
<td>1000 IU/h</td>
<td>Consider if anticoagulation needed for other reasons</td>
<td></td>
</tr>
<tr>
<td>Bivalirudin</td>
<td>4000 IU i.v.</td>
<td>1.75 mg/kg/h ≤4 h</td>
<td>Consider only if immediate cath</td>
<td></td>
</tr>
</tbody>
</table>

*Unless dose reduced to 5 mg

Reference: Hamm CW et al. Eur Heart J (2011);32 (23): 2999-3054
### Non ST-segment Elevation ACS: Treatment (3)

**Dosing and Recommendations for Pharmacological Therapies (cont.)**

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

<table>
<thead>
<tr>
<th>Drug</th>
<th>Loading Dose</th>
<th>Maintenance Dose</th>
<th>Considerations</th>
<th>Major Contraindications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Statins</td>
<td>–</td>
<td>**</td>
<td>Use initially in all patients LVSD, HF, HTN. Consider in all others Same as ACEI (preferred if ACEI-related cough) In NSTEMI + LVEF &lt;40% and HF or diabetes</td>
<td>Hypotension</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>–</td>
<td>**</td>
<td></td>
<td>Severe KD</td>
</tr>
<tr>
<td>Angiotensin RB</td>
<td>–</td>
<td>**</td>
<td></td>
<td>Hyperkaliemia</td>
</tr>
<tr>
<td>Aldosterone RB</td>
<td>–</td>
<td>25 mg QD</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**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

<table>
<thead>
<tr>
<th>Clinical situation</th>
<th>Timing</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Severe clinical or electrical instability:</strong></td>
<td><strong>Within first 2 hours</strong></td>
</tr>
<tr>
<td>Cardiogenic shock, severe heart failure, acute mitral</td>
<td></td>
</tr>
<tr>
<td>regurgitation, refractory symptoms, ventricular</td>
<td></td>
</tr>
<tr>
<td>arrhythmias</td>
<td></td>
</tr>
<tr>
<td><strong>Significant troponin rise / fall</strong></td>
<td><strong>Within first 24 hours</strong></td>
</tr>
<tr>
<td><strong>ST changes in ECG</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Other risk markers</strong></td>
<td></td>
</tr>
<tr>
<td>• DM</td>
<td></td>
</tr>
<tr>
<td>• Renal insufficiency (eGFR &lt;60 ml/min/1.73 m²)</td>
<td></td>
</tr>
<tr>
<td>• Reduced LV function (LVEF &lt;40%)</td>
<td></td>
</tr>
<tr>
<td>• Early postinfarction angina</td>
<td></td>
</tr>
<tr>
<td>• Recent coronary revascularisation</td>
<td></td>
</tr>
<tr>
<td>• Intermediate-high GRACE risk score</td>
<td></td>
</tr>
<tr>
<td><strong>Other non low-risk patients</strong></td>
<td><strong>Within first 72 hours</strong></td>
</tr>
<tr>
<td><strong>Low risk patients</strong></td>
<td><strong>No invasive strategy</strong></td>
</tr>
<tr>
<td><strong>Non candidates for coronary revascularisation</strong></td>
<td></td>
</tr>
</tbody>
</table>

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 $\geq 0.2$ mV in men or $\geq 0.15$ mV in women in leads V$_2$-V$_3$ and/or $\geq 0.1$ 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$_3$R and V$_4$R), 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

STEMI diagnosis\(^a\)

Primary-PCI capable center

EMS or non primary-PCI capable center

PCI possible <120 min?

Immediate transfer to PCI center

Yes

No

Preferably ≤30 min

Primary-PCI

Rescue PCI

Preferably ≤90 min

(≤60 min in early presenters)

Immediate transfer to PCI center

Successful fibrinolysis?

Yes

No

Preferably 3–24 h

Coronary angiography

Immediate fibrinolysis

Immediate transfer to PCI center

\(^a\)The time point the diagnosis is confirmed with patient history and ECG ideally within 10 min from FMC.

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

<table>
<thead>
<tr>
<th>Prehospital</th>
<th>PCI</th>
<th>CCU/ICCU</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin 150-300 mg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heparin 70 IU/kg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ticagrelor 180 mg or Prasugrel 60 mg or Clopidogrel 600 mg</td>
<td>Bivalirudin or GPI: Eptifibatide Tirofiban Abciximab</td>
<td>Follow local in-lab instruction/dosing</td>
</tr>
<tr>
<td></td>
<td>Metoprolol 25 mg BID or Carvedilol 3,25 mg BID or Bisoprolol 2,5 mg QD</td>
<td></td>
</tr>
<tr>
<td></td>
<td>High dose potent statins, i.e.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Atorvastatin 80 mg QD</td>
<td></td>
</tr>
</tbody>
</table>

## Medication Titration Day 2-7

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

- **Metoprolol**: 200 mg QD
- **Carvedilol**: 25 mg BID
- **Bisoprolol**: 5 mg BID
- **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**

# Doses of fibrinolytic agents

<table>
<thead>
<tr>
<th></th>
<th><strong>Initial treatment</strong></th>
<th><strong>Specific contraindications</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Streptokinase (SK)</td>
<td>1.5 million units over 30–60 min i.v.</td>
<td>Prior SK or anistreplase</td>
</tr>
<tr>
<td>Alteplase (tPA)</td>
<td>15 mg i.v. bolus</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.75 mg/kg over 30 min (up to 50 mg) then</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.5 mg/kg over 60 min i.v. (up to 35 mg)</td>
<td></td>
</tr>
<tr>
<td>Reteplase (rt-PA)</td>
<td>10 units + 10 units i.v. bolus given 30 min apart</td>
<td></td>
</tr>
<tr>
<td>Tenecteplase (TNK–tPA)</td>
<td>Single i.v. bolus:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>30 mg if &lt;60 kg</td>
<td></td>
</tr>
<tr>
<td></td>
<td>35 mg if 60 to &lt;70 kg</td>
<td></td>
</tr>
<tr>
<td></td>
<td>40 mg if 70 to &lt;80 kg</td>
<td></td>
</tr>
<tr>
<td></td>
<td>45 mg if 80 to &lt;90 kg</td>
<td></td>
</tr>
<tr>
<td></td>
<td>50 mg if ≥90 kg</td>
<td></td>
</tr>
</tbody>
</table>

### Contraindications to fibrinolytic therapy

<table>
<thead>
<tr>
<th>Absolute</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous intracranial hemorrhage or stroke of unknown origin at any time</td>
</tr>
<tr>
<td>Ischemic stroke in the preceding 6 months</td>
</tr>
<tr>
<td>Central nervous system damage or neoplasms or arteriovenous malformation</td>
</tr>
<tr>
<td>Recent major trauma/surgery/head injury (within the preceding 3 weeks)</td>
</tr>
<tr>
<td>Gastrointestinal bleeding within the past month</td>
</tr>
<tr>
<td>Known bleeding disorder (excluding menses)</td>
</tr>
<tr>
<td>Aortic dissection</td>
</tr>
<tr>
<td>Non-compressible punctures in the past 24 h (e.g. liver biopsy, lumbar puncture)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Relative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischemic stroke more than 6 months ago</td>
</tr>
<tr>
<td>Transient ischemic attack in the preceding 6 months</td>
</tr>
<tr>
<td>Oral anticoagulant therapy</td>
</tr>
<tr>
<td>Pregnancy or within 1 week postpartum</td>
</tr>
<tr>
<td>Refractory hypertension (systolic blood pressure &gt;180 mmHg and/or diastolic blood pressure &gt;110 mmHg)</td>
</tr>
<tr>
<td>Advanced liver disease</td>
</tr>
<tr>
<td>Infective endocarditis</td>
</tr>
<tr>
<td>Active peptic ulcer</td>
</tr>
<tr>
<td>Prolonged or traumatic resuscitation</td>
</tr>
</tbody>
</table>

### Doses of antiplatelet co-therapies

#### With primary PCI

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>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.</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>Loading dose of 600 mg orally, followed by a maintenance dose of 75 mg QD.</td>
</tr>
<tr>
<td>Prasugrel</td>
<td>Loading dose of 60 mg orally, followed by a maintenance dose of 10 mg QD. In patients with body weight &lt;60 kg, a maintenance dose of 5 mg is recommended. In patients &gt;75 years, prasugrel is generally not recommended, but a dose of 5 mg should be used if treatment is deemed necessary.</td>
</tr>
<tr>
<td>Ticagrelor</td>
<td>Loading dose of 180 mg orally, followed by a maintenance dose of 90 mg BID.</td>
</tr>
<tr>
<td>Abciximab</td>
<td>Bolus of 0.25 mg/kg i.v. and 0.125 μg/kg/min infusion (maximum 10 μg/min) for 12 h.</td>
</tr>
<tr>
<td>Eptifibatide</td>
<td>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.</td>
</tr>
<tr>
<td>Tirofiban</td>
<td>25 μg/kg over 3 min i.v., followed by a maintenance infusion of 0.15 μg/kg/min for 18 h.</td>
</tr>
</tbody>
</table>

#### With fibrinolytic therapy

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>Starting dose 150–500 mg orally or i.v. dose of 250 mg if oral ingestion is not possible.</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>Loading dose of 300 mg orally if aged ≤75 years, followed by a maintenance dose of 75 mg QD.</td>
</tr>
</tbody>
</table>
### Doses of antiplatelet co-therapies (Cont.)

#### Without reperfusion therapy

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>Starting dose 150–500 mg orally.</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>75 mg/day orally.</td>
</tr>
</tbody>
</table>

#### Doses of anticoagulation co-therapies

### With primary PCI

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unfractionated heparin</td>
<td>70–100 U/kg i.v. bolus when no GP IIb/IIIa inhibitor is planned.</td>
</tr>
<tr>
<td></td>
<td>50–60 U/kg i.v. bolus with GP IIb/IIIa inhibitors.</td>
</tr>
<tr>
<td>Enoxaparin</td>
<td>0.5 mg/kg i.v. bolus.</td>
</tr>
<tr>
<td>Bivalirudin</td>
<td>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.</td>
</tr>
</tbody>
</table>

### With fibrinolytic therapy

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unfractionated heparin</td>
<td>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.</td>
</tr>
<tr>
<td>Enoxaparin</td>
<td>In patients &lt;75 years of age:</td>
</tr>
<tr>
<td></td>
<td>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.</td>
</tr>
<tr>
<td></td>
<td>In patients &gt;75 years of age:</td>
</tr>
<tr>
<td></td>
<td>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.</td>
</tr>
<tr>
<td></td>
<td>In patients with creatinine clearance of &lt;30 mL/min, regardless of age, the s.c. doses are given once every 24 h.</td>
</tr>
<tr>
<td>Doses of anticoagulation co-therapies (Cont.)</td>
<td></td>
</tr>
<tr>
<td>---------------------------------------------</td>
<td></td>
</tr>
<tr>
<td><strong>With fibrinolytic therapy</strong></td>
<td></td>
</tr>
<tr>
<td>Fondaparinux</td>
<td>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.</td>
</tr>
<tr>
<td><strong>Without reperfusion therapy</strong></td>
<td></td>
</tr>
<tr>
<td>Unfractionated heparin</td>
<td>Same dose as with fibrinolytic therapy.</td>
</tr>
<tr>
<td>Enoxaparin</td>
<td>Same dose as with fibrinolytic therapy.</td>
</tr>
<tr>
<td>Fondaparinux</td>
<td>Same dose as with fibrinolytic therapy.</td>
</tr>
</tbody>
</table>

Reference: Steg G et al. Eur Heart J. (2012);33:2569-619 (°)
STEMI TREATMENT (5)

DOISING 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

Chapter 3
ACUTE HEART FAILURE

3.1 Acute heart failure and pulmonary oedema

3.2 Cardiogenic shock
ACUTE HEART FAILURE: DIAGNOSIS AND CAUSES (1)

Patient with symptoms and signs of acute heart failure*

History of heart failure

Yes 60-80%

No 20-40%

Cardiovascular risk profile*

Precipitating factors*

Yes

High likelihood of acute heart failure*

Intermediate to high likelihood of acute heart failure*

Intermediate likelihood of acute heart failure*

Rule out differential diagnosis*

* (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, 3\textsuperscript{rd}/4\textsuperscript{th} 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

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

INSTRUMENTATION & INVESTIGATIONS
- Pulse oximeter
- Consider arterial line
- ABG (SaO₂, pH, pCO₂)
- Chest X-ray/lung ultrasound
  - Fluid overload/congestion?
  - Cardiomegaly?
  - Pleural effusion?
  - Consolidation?
  - Pneumothorax?
  - A-lines? B-lines?

INSTRUMENTATION & INVESTIGATIONS
- Pulse oximeter
- Consider arterial line
- ABG (SaO₂, pH, pCO₂)
- Chest X-ray/lung ultrasound
  - Fluid overload/congestion?
  - Cardiomegaly?
  - Pleural effusion?
  - Consolidation?
  - Pneumothorax?
  - A-lines? B-lines?

OXYGEN* (+ oropharyngeal airway [Guedel/Mayo]/nasopharyngeal airway)

Nasal: 1 ltr = FiO₂ 22%, 2 ltr = 25%, 3 ltr = 27%, 4 ltr = 30%, 5 ltr = 35%
Mask: 2 ltr = FiO₂ 25%, 4 ltr = 30%, 6 ltr = 40%, 7 ltr = 45%, >8 ltr = 50%
Mask + reservoir: 6 ltr = FiO₂ 60%, 7 ltr = 70%, 8 ltr = 80%, 10 ltr = 90%
Venturimask**: 24% = FiO₂ 24%, 35% = 35%, 40% = 40%, 60% = 50%

~5 minutes to reassess

Sufficient oxygenation (SpO₂>90%)

INSTRUMENTATION & INVESTIGATIONS
- Pulse oximeter
- Consider arterial line
- ABG (SaO₂, pH, pCO₂)
- Chest X-ray/lung ultrasound
  - Fluid overload/congestion?
  - Cardiomegaly?
  - Pleural effusion?
  - Consolidation?
  - Pneumothorax?
  - A-lines? B-lines?

Start CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP)

~15 minutes to reassess

Oxygen* + Positive End-Expiratory Pressure (PEEP) 5-7.5 mmHg

~5 minutes to reassess

Sufficient oxygenation (SpO₂>90%)

INSTRUMENTATION & INVESTIGATIONS
- Pulse oximeter
- Consider arterial line
- ABG (SaO₂, pH, pCO₂)
- Chest X-ray/lung ultrasound
  - Fluid overload/congestion?
  - Cardiomegaly?
  - Pleural effusion?
  - Consolidation?
  - Pneumothorax?
  - A-lines? B-lines?

Sufficient ventilation (pCO₂<45mmHg)***

INSTRUMENTATION & INVESTIGATIONS
- Pulse oximeter
- Consider arterial line
- ABG (SaO₂, pH, pCO₂)
- Chest X-ray/lung ultrasound
  - Fluid overload/congestion?
  - Cardiomegaly?
  - Pleural effusion?
  - Consolidation?
  - Pneumothorax?
  - A-lines? B-lines?

INSTRUMENTATION & INVESTIGATIONS
- Pulse oximeter
- Consider arterial line
- ABG (SaO₂, pH, pCO₂)
- Chest X-ray/lung ultrasound
  - Fluid overload/congestion?
  - Cardiomegaly?
  - Pleural effusion?
  - Consolidation?
  - Pneumothorax?
  - A-lines? B-lines?
Goal SpO₂ 94-98%

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

For a patient with COPD, a pCO₂ of 45-50 mmHg may be optimal. Aim for a normal pH.

Consider if the above fails or when patient is fatigued.

Sufficient oxygenation (SpO₂ > 90%)

Yes

No

Oxygen* + PEEP 5-10 mmHg + Ventilatory Support (pressure support)

Start NON-INVASIVE VENTILATION (NIV)
(positive pressure, bilevel) + PEEP 5-10 mmHg
Consider ENDOTRACHEAL INTUBATION (ETT)**
Get support on time

* Goal SpO₂ 94-98%
** Use the predefined liters of oxygen. When using higher flows the FiO₂ will drop.
*** For a patient with COPD, a pCO₂ 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 consciousness/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. capillary refill), color
Urinary output (<0.5 ml/kg/hr) ➔ Insert indwelling catheter
1. **Inotropic drugs**
   - Dobutamine 2.5 μg/kg/min
   - Milrinone bolus 25 μg/kg in 10-20 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**

---

**C: Circulatory failure/shock <85 mmHg or SBP <100 mmHg?**

---

**C: Volume overload, SBP >140 mmHg?**

---

**AB: Respiratory failure diagnosed and treated**
- oxygen, CPAP, NIV, ETT

---

**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 chronic 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:** SpO₂ 94-98% and pCO₂ <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-morbidity 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 SpO₂ 94-98%, pCO₂ <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
<table>
<thead>
<tr>
<th>DRUG</th>
<th>STARTING DOSE</th>
<th>TARGET DOSE</th>
<th>CONSIDERATIONS</th>
<th>MAJOR CONTRAINDICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ACE inhibitors</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Captopril</td>
<td>6.25 mg TID</td>
<td>50 TID</td>
<td>Check renal function, electrolytes, drug interactions</td>
<td>History of angioedema</td>
</tr>
<tr>
<td>Enalapril</td>
<td>2.5 mg BID</td>
<td>10-20 BID</td>
<td></td>
<td>Known bilateral renal artery stenosis</td>
</tr>
<tr>
<td>Lisinopril</td>
<td>2.5-5.0 mg QD</td>
<td>20-35 QD</td>
<td></td>
<td>Pregnancy (risk)</td>
</tr>
<tr>
<td>Ramipril</td>
<td>2.5 mg QD</td>
<td>5 BID</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trandolapril</td>
<td>0.5 QD</td>
<td>4 QD</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>RB</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Candesartan</td>
<td>4-8 mg QD</td>
<td>32 QD</td>
<td>If ACEI is not tolerated</td>
<td>History of angioedema.</td>
</tr>
<tr>
<td>Valsartan</td>
<td>40 mg BID</td>
<td>160 BID</td>
<td>Check renal function, electrolytes, drug interactions</td>
<td>Known bilateral renal artery stenosis</td>
</tr>
<tr>
<td>Losartan</td>
<td>50 QD</td>
<td>150 QD</td>
<td></td>
<td>Pregnancy (risk)</td>
</tr>
<tr>
<td><strong>ß-blockers</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bisoprolol</td>
<td>1.25 QD</td>
<td>10 QD</td>
<td>Check 12- lead ECG</td>
<td>Asthma</td>
</tr>
<tr>
<td>Carvedilol</td>
<td>3.125 BID</td>
<td>25-50 BID</td>
<td></td>
<td>Second on third- degree</td>
</tr>
<tr>
<td>Metaprolol</td>
<td>12.5-25 QD</td>
<td>200 QD</td>
<td></td>
<td>AV block</td>
</tr>
<tr>
<td>Nebivolol</td>
<td>1.25 QD</td>
<td>10 QD</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Aldosterone-antagonists</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spironolactone</td>
<td>25 QD</td>
<td>25-50 QD</td>
<td>Check renal function, electrolytes, drug interactions</td>
<td>Eplerenone —— strong</td>
</tr>
<tr>
<td>Eplerenone</td>
<td>25 QD</td>
<td>50 QD</td>
<td></td>
<td>CYP3A-4 inhibitors</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Hemodynamic criteria to define cardiogenic shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Systolic blood pressure &lt;80 to 90 mmHg or mean arterial pressure 30 mmHg lower than baseline</td>
</tr>
<tr>
<td>• Severe reduction in cardiac index:</td>
</tr>
<tr>
<td>&lt;1.8 L/min/m² without support or</td>
</tr>
<tr>
<td>&lt;2.0 to 2.2 L/min/m² with support</td>
</tr>
<tr>
<td>• Adequate or elevated filling pressure:</td>
</tr>
<tr>
<td>Left ventricular end-diastolic pressure &gt;18 mmHg or</td>
</tr>
<tr>
<td>Right ventricular end-diastolic pressure &gt;10 to 15 mmHg</td>
</tr>
</tbody>
</table>
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.

## 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 <2.2 l/min/m²)
- **Orthopnea (PCWP >22 mmHg)**
- **Tachypnea (>20/min, >30/min (!))**
- **Killip class II-IV**
- **Clinical symptoms of tissue hypoperfusion/hypoxia:**  
  - cool extremities
  - decreased capillary refill or mottling
  - decreased urine output (urine output <40 ml/h)
  - 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).**

## 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 output 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

<table>
<thead>
<tr>
<th>DRUG TYPE</th>
<th>CLINICAL ACTION</th>
<th>DOSAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Levosimendan</strong></td>
<td>Calcium sensitizer</td>
<td>Vasodilation, positive inotropic</td>
</tr>
<tr>
<td><strong>Milrinone</strong></td>
<td>Phosphodiesterase inhibitor</td>
<td>Vasodilation, positive inotropic</td>
</tr>
<tr>
<td><strong>Isoprenaline</strong></td>
<td>β₁, β₂ agonist</td>
<td>Positive chronotropic (pulmonary vasodilation)</td>
</tr>
<tr>
<td><strong>Dobutamine</strong></td>
<td>β₁,α₁/β₂ agonist</td>
<td>β₂-mediated vasodilation, positive inotropic, chronotronic</td>
</tr>
<tr>
<td><strong>Dopamine</strong></td>
<td>β, α, dopaminergic agonist</td>
<td>Peripheral vasodilation (e.g. splanchnic, renal)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Positive chronotropic, positive inotropic</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vasoconstriction at high doses</td>
</tr>
<tr>
<td><strong>Noradrenaline</strong></td>
<td>α₁, β₁ agonist</td>
<td>Vasoconstriction, positive inotropic</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Ventilator mode</th>
<th>Pressure assist/control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tidal Volume goal</td>
<td>Reduce tidal volume to 6-8 ml/kg lean body weight</td>
</tr>
<tr>
<td>Plateau Pressure goal</td>
<td>≤30 cm H\textsubscript{2}O</td>
</tr>
<tr>
<td>Anticipated PEEP levels</td>
<td>5-10 cm H\textsubscript{2}O</td>
</tr>
<tr>
<td>Ventilator rate and pH goal</td>
<td>12-20, adjusted to achieve a pH ≥7.30 if possible</td>
</tr>
<tr>
<td>Inspiration: Expiration time</td>
<td>1:1 to 1:2</td>
</tr>
<tr>
<td>Oxygenation goal:</td>
<td>50-80 mmHg</td>
</tr>
<tr>
<td>- ( \text{PaO}_2 )</td>
<td>&gt;90%</td>
</tr>
<tr>
<td>- ( \text{SpO}_2 )</td>
<td></td>
</tr>
</tbody>
</table>

Some patients with CS will require increased PEEP to attain functional residual capacity and maintain oxygenation, and peak pressures above 30 cm H\textsubscript{2}O to attain effective tidal volumes of 6-8ml/kg with adequate CO\textsubscript{2} removal.

Predicted body weight calculation:
- Male: 50 + 0.91 (height in cm - 152.4)
- Female: 45.5 + 0.91 (height in cm - 152.4)
CARDIOGENIC SHOCK: MANAGEMENT FOLLOWING STEMI

Assess volume status
Treat sustained arrhythmias: brady- or tachy-
Consider mechanical ventilation for comfort (during PCI) and/or as needed:
  • to correct acidosis
  • to correct hypoxemia
Inotropic support (dobutamine and/or vasopressor support)

Signs (ST-segment elevation or new LBBB) and/or clinical symptoms of ongoing myocardial ischemia

Yes

No

Emergency echocardiography ± Tissue doppler imaging ± Color flow imaging

NSTEACS, Delayed CS
CARDIOGENIC SHOCK: MANAGEMENT FOLLOWING STEMI

- Assess volume status
- Treat sustained arrhythmias: brady- or tachy-
- Consider mechanical ventilation for comfort (during PCI) and/or as needed:
  - to correct acidosis
  - to correct hypoxemia
- Inotropic support (dobutamine and/or vasopressor support)
- Signs (ST-segment elevation or new LBBB) and/or clinical symptoms of ongoing myocardial ischemia
- Early coronary angiography ± Pulmonary artery catheter ± IABP in selected patients in a specialised Myocardial Intervention Center
- PCI ± stenting of the culprit lesion ± correct mechanical complications
- CABG
- Operating theater ± coronary angiography
- Pump failure RV, LV, both
- Acute severe mitral valve regurgitation
- Ventricular septum rupture
- Severe aortic/mitral valve stenosis
- Aortic dissection
- Pericardial tamponade
- Emergency echocardiography ± Tissue doppler imaging ± Color flow imaging
- NSTEACS, Delayed CS
- Yes
- No
# Cardiogenic Shock: Mechanical Circulatory Support, Basic Characteristics

## Level of Support

<table>
<thead>
<tr>
<th>Time</th>
<th>Level</th>
<th>Support</th>
<th>Devices</th>
</tr>
</thead>
<tbody>
<tr>
<td>72-hrs</td>
<td></td>
<td>Left ventricular support</td>
<td>IABP, Impella 2.5, Impella 5.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>BiVentricular support</td>
<td>Tandem-heart, Levitronix, ECMO</td>
</tr>
<tr>
<td>2-weeks</td>
<td></td>
<td>Full support</td>
<td>Implantable</td>
</tr>
<tr>
<td>1-month</td>
<td></td>
<td>Pulmonary support</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
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.

<table>
<thead>
<tr>
<th>System</th>
<th>Type</th>
<th>Support</th>
<th>Min Flow Rate</th>
<th>Access</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intra-aortic balloon pump</td>
<td>Balloon counterpulsation</td>
<td>Pulsatile flow</td>
<td>&lt;0.5 L</td>
<td>Arterial: 7.5 French</td>
</tr>
<tr>
<td>Impella Recover</td>
<td>Axial flow</td>
<td>Continuous flow</td>
<td>&lt;2.5 L, &lt;4.0 L, &lt;5.0 L</td>
<td>Arterial: 12 French, Arterial: 14 French, Arterial: 21 French</td>
</tr>
<tr>
<td>Impella Recover LP 2.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Impella Recover CP</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Impella Recover LP 5.0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tandemheart</td>
<td>Centrifugal flow</td>
<td>Continuous flow</td>
<td>&lt;5.0 L</td>
<td>Venous: 21 French, Arterial: 15-17 French</td>
</tr>
<tr>
<td>Cardiohelp</td>
<td></td>
<td></td>
<td>&lt;5.0 L</td>
<td>Venous: 15-29 French, Arterial: 15-29 French</td>
</tr>
</tbody>
</table>
Chapter 4
CARDIAC ARREST
AND CARDIOPULMONARY RESUSCITATION

THE CHAIN OF SURVIVAL

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

VICTIM COLLAPSES

Approach safely
Check response

Victim responds
Leave victim as found
Find out what is wrong
Reassess victim regularly

Victim unresponsive

Breathing normally
Put victim in recovery position
and call for an ambulance

Not breathing normally

CPR 30:2
Shout for help
Open airway

Call for an ambulance
Send or go for an AED

AED not available

AED available

30 chest compressions:
2 rescue breaths

Start AED,
listen to and follow voice prompts

AED Assesses rhythm

Immediately resume
CPR 30:2 for 2 min

Shock advised
1

Immediately resume
CPR 30:2 for 2 min

No shock advised

Immediately resume
CPR 30:2 for 2 min

Put victim in recovery position
and call for an ambulance

Breathing normally

Approach safely
Check response
Victim collapses

Victim responds

Victim unresponsive

Leave victim as found

Find out what is wrong

Reassess victim regularly

CPR 30:2

Shout for help

Open airway

Not breathing normally

Call for an ambulance

Send or go for an AED

AED available

Start AED, listen to and follow voice prompts

AED Assesses rhythm

AED not available

30 chest compressions:

2 rescue breaths

Continue until victim starts to wake up: to move, open eyes, and breathe normally

Shock advised

1 shock

Immediately resume CPR 30:2 for 2 min

No shock advised

Immediately resume CPR 30:2 for 2 min
IN-HOSPITAL CARDIAC ARREST: ASSESSMENT OF A COLLAPSED VICTIM AND INITIAL TREATMENT

Collapsed/sick patient

Shout for HELP & assess patient

Yes
Assess ABCDE
Recognise & treat oxygen; monitoring, i.v. access

No
Signs of life?

Call resuscitation team

CPR 30:2
with oxygen and airway adjuncts
IN-HOSPITAL CARDIAC ARREST: ASSESSMENT OF A COLLAPSED VICTIM AND INITIAL TREATMENT

Collapsed/sick patient

Shout for HELP & assess patient

Assess ABCDE

Recognise & treat

- oxygen; monitoring, i.v. access

Call resuscitation team

CPR 30:2 with oxygen and airway adjuncts

Handover to resuscitation team when resuscitation team arrives

Apply pads/monitor if appropriate

Attempt defibrillation if appropriate

Advanced Life Support

Call resuscitation team if appropriate

Handover to resuscitation team
IN-HOSPITAL CARDIAC ARREST: ADVANCED LIFE SUPPORT

Unresponsive?
Not breathing or only occasional gasps

Call resuscitation team

CPR 30:2
Attach defibrillator/monitor
Minimise interruptions

Assess rhythm

Shockable (VF/Pulseless VT) → Non-shockable (PEA/Asystole)

REVERSIBLE CAUSES
Hypoxia
Hypovolaemia
Hypo-/hyperkalaemia/metabolic
Hypothermia
Thrombosis
Tamponade - cardiac
Toxins
Tension pneumothorax

IMMEDIATE POST CARDIAC ARREST TREATMENT
Immediately resume:
CPR for 2 min
Minimise interruptions

Add controlled oxygen and ventilation
12-lead ECG
Treat precipitating cause
Temperature control / Therapeutic hypothermia
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

IMMEDIATE POST CARDIAC ARREST TREATMENT
- Use ABCDE approach
- Controlled oxygenation and ventilation
- 12-lead ECG
- Treat precipitating cause
- Temperature control / Therapeutic hypothermia

REVERSIBLE CAUSES
- Hypoxia
- Hypovolaemia
- Hypo-/hyperkalaemia/metabolic
- Hypothermia
- Thrombosis
- Tamponade - cardiac
- Toxins
- Tension pneumothorax
IN-HOSPITAL CARDIAC ARREST: DRUG THERAPY DURING ADVANCED LIFE SUPPORT

Cardiac Arrest

Shockable rhythm (VF, pulseless VT)

Give adrenaline and amiodarone after 3rd shock

Adrenaline: 1 mg i.v. (10 ml 1:10,000 or 1 ml 1:1000) repeated every 3-5 min (alternate loops) given without interrupting chest compressions

Amiodarone
300 mg bolus i.v.
Second bolus dose of 150 mg i.v. if VF/VT persists followed by infusion of 900 mg over 24 h

Non-shockable rhythm

Adrenaline: 1 mg i.v. (10 ml 1:10,000 or 1 ml 1:1000) given as soon as circulatory access is obtained Repeat every 3-5 min (alternate loops) Give without interrupting chest compressions
Chapter 5
RHYTHM DISTURBANCES

5.1 Supraventricular tachycardias and atrial fibrillation

5.2 Ventricular tachycardias

5.3 Bradyarrhythmias
TACHYARRHYTHMIAS: DIAGNOSTIC CRITERIA

Tachycardia > 100 beats/minute

Regular

QRS morphology similar to QRS morphology in sinus rhythm?

YES

QRS complex <120 msec

Supraventricular Tachycardia

QRS complex >120 msec

Supraventricular Tachycardia + BBB

NO

QRS complex <120 msec

Fascicular Tachycardia or SVT with aberrant conduction (see page 79)

QRS complex >120 msec

Ventricular Tachycardia or SVT with aberrant conduction (see page 78)

Irregular

QRS morphology similar to QRS morphology in sinus rhythm?

YES

QRS complex <120 msec

AF conducting over AVN

QRS complex >120 msec

AF + BBB or AF + WPW

NO

QRS complex <120 msec

AF + WPW

Variable QRS morphology

AF

Irregular Ventricular Tachycardia
TACHYARRHYTHMIAS: DIAGNOSTIC MANEUVERS

Regular tachycardia

Vagal maneuvers or i.v. adenosine

Tachycardia terminates

AV relation changes

More As than Vs

More Vs than As

Wide QRS complex

• Concordant precordial pattern (all leads + or all leads –)
• No RS pattern in precordial leads
• RS pattern with beginning of R wave to nadir of S wave <100 msec

No change

Narrow QRS complex

Typical morphology in V1 & V6 (see page 79)

Consider Sinus tachycardia or non proper administration of adenosine (too slow, insufficient dose, etc)

Consider SVT using the AV node (AVNRT, AVNT)

Atrial flutter or atrial tachycardia

Ventricular Tachycardia

Ventricular Tachycardia
TACHYARRHYTHMIAS: THERAPEUTIC ALGORITHMS (I)

REGULAR SUPRAVENTRICULAR TACHYCARDIAS WITH OR WITHOUT BUNDLE BRANCH BLOCK

- Hemodynamically poorly tolerated
  - Immediate electrical cardioversion
  - No termination
  - Narrow QRS complex tachycardia
    - Reconsider diagnosis: sinus tachycardia, atrial tachycardia
    - If no evidence: INTRAVENOUS VERAPAMIL

- Good hemodynamical tolerance
  - Vagal maneuvers and/or i.v. Adenosine
  - Termination
  - Wide QRS complex tachycardia
    - Reconsider the diagnosis of Ventricular Tachycardia even if hemodynamically well tolerated
    - DO NOT ADMINISTER VERAPAMIL

IRREGULAR AND NARROW QRS COMPLEX TACHYCARDIA

- Less than 48 hours since initiation AND hemodynamically well tolerated
  - CARDIOVERSION
    - Electrical or pharmacological using oral or i.v. flecainide (only in normal heart) or i.v. vernakalant
  - ANTICOAGULATION is initiated using i.v. heparine

- Hemodynamically poorly tolerated
  - IMMEDIATE ELECTRICAL CARDIOVERSION
  - If no cardioversion is considered:
    - rate control using beta blockers or calcium antagonists, together with proper anticoagulation, if required

- More than 48 hours OR unknown time of initiation, AND
  - Patient chronically anticoagulated OR a TEE showing no thrombus
  - Electrical or pharmacological CARDIOVERSION
Hemodynamically poorly tolerated

**Immediate electrical CARDIOVERSION**

If no cardioversion is considered: rate control using betablockers or calcium antagonists (only if VT and AF+WPW is excluded), together with proper anticoagulation if required

More than 48 hours or unknown initiation, AND patient chronically anticoagulated or a TEE showing no thrombus

**Electrical or pharmacological CARDIOVERSION**

Less than 48 hours since initiation AND hemodynamically well tolerated

**CARDIOVERSION**

electrical or pharmacological using oral or i.v. **flecainide** (only in normal heart) or i.v. **amiodarone**

**ANTICOAGULATION** is initiated using i.v. heparin
VENTRICULAR TACHYCARDIAS: DIFFERENTIAL DIAGNOSIS OF WIDE QRS TACHYCARDIA

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**

Yes

Morphology in precordial leads

Morphology in aVR lead
VENTRICULAR TACHYCARDIAS: Differential Diagnosis of Wide QRS TACHYCARDIA

**EKG signs of atrio-ventricular dissociation**
- Random P waves unrelated to QRS complexes
- Capture beats / fusion beats / second degree V-A block

**1st Step**
- Concordant pattern in precordial leads
- No RS morphology in any of the precordial leads
- An interval >100 ms from the beginning of the QRS complex to the nadir of S in a precordial lead

**Morphology in precordial leads**
- **RBBB morphology**
  - V1: qR, R, R'
  - V6: rS, QS
- **LBBB morphology**
  - V1: rsR', RSR'
  - V6: qRs
- **Morphology in aVR lead**
  - Initial R wave
  - or q >40 msec
  - V1: rsR', RSR'
  - V6: qRs

**Aberrant conduction**
- V1: qR, R, R'
- V6: rS, QS

**VT**

**Aberrant conduction**

**VT**
MANAGEMENT OF WIDE QRS TACHYCARDIAS

Hemodynamic Tolerance

**Poorly Tolerated**

**With pulse**
- Sedation or analgesia
- Synchronised cardioversion
  - 100 to 200 J (monophasic)
  - or 50-100 J (biphasic)

**Irregular rhythm**
- Differential Diagnosis
  - AF with aberrant ventricular conduction
    - β-blockers
    - i.v.
    - Verapamil or diltazem
  - Pre excited AF
    - Class 1 AADs
  - Polymorphic VT
    - Amiodarone

**Regular rhythm**
- Vagal maneuver and/or i.v. adenosine (push)
- Interrupt or slow down HR
- Differential Diagnosis (see page 75)

**SVT**

**Well Tolerated**

**Pulseless**

ACLS Resuscitation algorithm
- Immediate **high-energy defibrillation** (200J biphasic or 360 monophasic)
- Resume CPR and continue according to the ACLS algorithm

Drugs used in the ACLS algorithm
- Epinephrine 1 mg i.v./i.o.
  (repeat every 3-5min)
- Vasopressin 40 i.v./i.o.
- Amiodarone 300 mg i.v./i.o.
  once then consider an additional 150 mg i.v./i.o. dose
- Lidocaine 1-1.5 mg/kg first dose
  then 0.5-0.75 mg/kg i.v./i.o. for max 3 doses or 3 mg/kg
- Magnesium loading dose 1-2 gr
  i.v./i.o. for torsade des pointes

Amiodarone 150 mg i.v.
(can be repeated up to a maximum dose of 2.2 g in 24 h)
Synchronised cardioversion
**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
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

6.1 Acute aortic syndromes

6.2 Acute pulmonary embolism
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
Consider acute aortic dissection in all patients presenting with:

- Chest, back or abdominal pain
- Syncope
- Symptoms consistent with perfusion deficit (central nervous system, visceral myocardial or limb ischemia)

Pre-test risk assessment for acute aortic dissection

**High-risk conditions**
- Marfan’s syndrome
- Connective tissue disease
- Family history of aortic disease
- Aortic valve disease
- Thoracic aortic aneurysm

**High-risk pain features**
Chest, back or abdominal pain described as:
Abrupt at onset, severe in intensity, and ripping/sharp or stabbing quality

**High-risk exam features**
- Perfusion deficit:
  - Pulse deficit
  - SBP differential
  - Focal neurological deficit
- Aortic regurgitation murmur
- Hypotension or shock

ACUTE AORTIC SYNDROMES: DIAGNOSIS

High index of suspicion for AAS
Determine pre-test risk by combination of risk condition, history and exam

- **Intermediate Risk**
  - Any single risk feature present

- **Low Risk**
  - No high risk feature present

- **High Risk**
  - Two or more risk features present

**ELG, CXR**
**BLOOD TEST**

- D-dimer <500 µg/L
- PE?

**Initiate appropriate therapy**

**Identified**
**D-dimer <500 µg/L**
**Consider alternate diagnosis**

- TEE (preferred if clinically unstable)
- CT (image entire aorta: chest to pelvis)

If AAS present
Proceed to treatment pathway

If suspicion for AAS still remains,
consider secondary imaging study

**D-dimer <1600 µg/L**
**(within 6 hours Sx onset)**
**Very high suspicion of AAS**

**Immediate surgical consultation and expedited aortic imaging**

If suspicion for AAS still remains
(possible IMH-PAU-thrombosed false lumen)
ACUTE AORTIC SYNDROMES: DIAGNOSIS

High index of suspicion for AAS

Determine pre-test risk by combination of risk condition, history and exam

Low Risk
- No high risk feature present

Intermediate Risk
- Any single risk feature present

High Risk
- Two or more risk features present

D-dimer <500 µg/L
- Consider alternate diagnosis

D-dimer >500 µg/L
- PE?

Identified
- If suspicion for AAS still remains (possible IMH-PAU-thrombosed false lumen)

TEE (preferred if clinically unstable)
CT (image entire aorta: chest to pelvis)

If AAS present
- proceed to treatment pathway

If suspicion for AAS still remains,
- consider secondary imaging study

Initiate appropriate therapy
- Immediate surgical consultation and expedited aortic imaging

D-dimer <1600 µg/L
- (within 6 hours Sx onset)
- Very high suspicion of AAS

ELG, CXR

BLOOD TEST

TEE

ACUTE AORTIC SYNDROME: IMAGING DIAGNOSTIC STRATEGY

Suspected aortic dissection

TTE
- Type A
  - Surgery TEE
- Type B
- Non-conclusive
  - Surgery TEE

TEE*/CT
- Type A
  - Surgery
- Type B
  - TEE*/CT
- Non-conclusive
  - TEE*/CT

CT**
- Type A
  - Non-conclusive/ negative
- Type B
- Non-conclusive

Information to be obtained by imaging:
- Ao dissection, IMH, PAU or Ao aneurysm rupture
- Ascending aorta involvement
- Impending rupture (hemomediatinum 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

ACUTE AORTIC SYNDROMES MANAGEMENT: GENERAL APPROACH

ACUTE AORTIC DISSECTION

Type A
(Ascending aorta involvement)

- Surgery or peripheral vascular intervention

Type B
(No ascending aorta involvement)

- Uncomplicated
  - Medical treatment
- Complicated
  - (malperfusion, rupture)

Surgery or intervention
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 β-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 β-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

UNCOMPPLICATED
defined as:
No features of complicated dissection

MEDICAL MANAGEMENT and imaging surveillance protocol
• On admission
• At 7 days
• At discharge
• Every 6 months thereafter

MEDICAL MANAGEMENT and TEVAR
MEDICAL MANAGEMENT and OPEN SURGERY REPAIR
if TEVAR contraindicated

ON ADMISSION
At 7 days
At discharge
Every 6 months thereafter
ACUTE PULMONARY EMBOLISM: DIAGNOSIS

**CARDIOVASCULAR Symptoms/Signs**
including but not limited to:

- Chest pain (angina)
- Syncope
- Tachycardia
- ECG changes
- BNP/NTproBNP ↑
- Troponin ↑

**RESPIRATORY Symptoms/Signs**
including but not limited to:

- Chest pain (pleural)
- Pleural effusion
- Tachypnea
- Hemothysis
- Hypoxemia
- Atelectasis

**Dyspnea**

**Suspect acute PE**

**YES**

**Shock?**
or
**SBP <90 mmHg?**

or
**SBP fall by >40 mmHg?**

**NO**

**Management algorithm for UNSTABLE patients**

**Management algorithm for initially STABLE patients**
MANAGEMENT ALGORITHM FOR UNSTABLE PATIENTS WITH SUSPECTED ACUTE PULMONARY EMBOLISM

CT angiography immediately available and patient stabilised

Thrombolysis or embolectomy

Thrombolysis/ embolectomy not justified

CT^*

Angio

TTE

RV pressure overload

CUS

TEE

Search for other causes

positive

No

Yes

No further diagnostic tests feasible

Patient stabilised

Right heart, pulmonary artery or venous thrombi?

Yes

negative

Thrombolysis or embolectomy

* Consider also pulmonary angiography if unstable patient in hemodynamic lab

Copyright: IACC Textbook (2011) chapter 64 Pulmonary embolism – Konstantinides S & Torbicki A - Page 661 - Figure 64.1
doi:10.1093/med/9780199584314.001.0001
MANAGEMENT ALGORITHM FOR INITIALLY STABLE PATIENTS WITH SUSPECTED ACUTE PULMONARY EMBOLISM

Asses clinical (pre-test) probability

Low or intermediate
“PE unlikely“

D-dimer

negative

MDCT

negative

Anticoagulation not justified

MDCT Positive

Anticoagulation required

High or
“PE likely“

MDCT

positive

CUS

positive

MDCT

Positive

Anticoagulation required

Negative
Confirm by CUS
V/Q scan or angiography

MDCT

to

CUS

not justified
**MANAGEMENT STRATEGY FOR INITIALLY STABLE PATIENTS WITH CONFIRMED NON-HIGH RISK PULMONARY EMBOLISM**

<table>
<thead>
<tr>
<th>Markers for myocardial injury</th>
<th>Positive*</th>
<th>Positive**</th>
<th>Negative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Markers for RV overload</td>
<td>Positive*</td>
<td>Positive**</td>
<td>Negative</td>
</tr>
<tr>
<td>Clinical risk assessment score</td>
<td>Positive*</td>
<td>Positive**</td>
<td>Negative</td>
</tr>
<tr>
<td>Preferred initial anticoagulation</td>
<td>i.v. UFH/LMWH</td>
<td>LMWH/Fonda/NOAC</td>
<td>LMWH/Fonda/NOAC</td>
</tr>
</tbody>
</table>

**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

<table>
<thead>
<tr>
<th>Shock or hypotension</th>
<th>YES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recent intracerebral hemorrhage</td>
<td>NO</td>
</tr>
<tr>
<td>Recent surgery or major bleed</td>
<td>NO</td>
</tr>
<tr>
<td>Right heart floating thrombus</td>
<td>NO or small and limited to right heart</td>
</tr>
</tbody>
</table>

### STRATEGY

<table>
<thead>
<tr>
<th>Thrombolysis, surgical or percutaneous catheter embolectomy</th>
<th>Surgical or Percutaneous catheter embolectomy (availability/experience)</th>
<th>Surgical embolectomy</th>
</tr>
</thead>
</table>

i.v. UFH, STABILISE SYSTEMIC BLOOD PRESSURE, CORRECT HYPOXEMIA
# PULMONARY EMBOLISM: PHARMACOLOGICAL TREATMENT

## Key drugs for initial treatment of patients with confirmed PE

<table>
<thead>
<tr>
<th>Status</th>
<th>Drug</th>
<th>Dose/Regimen</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Unstable</strong></td>
<td><strong>Alteplase (rtPA) (intravenous)</strong></td>
<td>100 mg/2 h or 0.6 mg/kg/15 min (max 50 mg)</td>
</tr>
<tr>
<td></td>
<td><strong>Urokinase (intravenous)</strong></td>
<td>3 million IU over 2 h</td>
</tr>
<tr>
<td></td>
<td><strong>Streptokinase (intravenous)</strong></td>
<td>1.5 million IU over 2 h</td>
</tr>
<tr>
<td></td>
<td><strong>Unfractionated heparin (intravenous)</strong></td>
<td>80 IU/kg bolus + 18 IU/kg/h</td>
</tr>
<tr>
<td><strong>Stable</strong></td>
<td><strong>Enoxaparine (subcutaneous)</strong></td>
<td>1.0 mg/kg BID or 1.5 mg/kg QD</td>
</tr>
<tr>
<td></td>
<td><strong>Tinzaparin (subcutaneous)</strong></td>
<td>175 U/kg QD</td>
</tr>
<tr>
<td></td>
<td><strong>Fondaparinux (subcutaneous)</strong></td>
<td>7.5 mg (50-100 Kg of body weight) 5 mg for patients &lt;50 kg, 10 mg for patients &gt;100 kg</td>
</tr>
<tr>
<td></td>
<td><strong>Rivaroxaban (oral)</strong></td>
<td>15 mg BID (for 3 weeks, then 20 mg QD)</td>
</tr>
<tr>
<td></td>
<td><strong>Other new oral anticoagulants</strong></td>
<td>Pending approval for pulmonary embolism</td>
</tr>
</tbody>
</table>
Chapter 7

ACUTE MYOCARDIAL / PERICARDIAL SYNDROMES

7.1 Acute myocarditis

7.2 Acute pericarditis and pericardial tamponade
**ACUTE MYOCARDITIS: DEFINITION AND CAUSES**

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

**CAUSES OF MYOCARDITIS**

**INFECTIOUS**
- Viral
- Bacterial
- Spirochaetal
- Fungal
- Protozoal
- Parasitic
- Rickettsial

**IMMUNE-MEDIATED**
- Allergens: Tetanus toxoid, vaccines, serum sickness, Drugs
- Alloantigens: Heart transplant rejection
- Autoantigens: Infection-negative lymphocytic, infection-negative giant cell, associated with autoimmune or immune oriented disorders

**TOXIC**
- Drugs
- Heavy Metals
- Hormones, e.g. catecholamines (Pheochromocytoma)
- Physical agents
**ACUTE MYOCARDITIS: DIAGNOSTIC CRITERIA (I)**

**DIAGNOSTIC CRITERIA FOR CLINICALLY SUSPECTED MYOCARDITIS**

<table>
<thead>
<tr>
<th>CLINICAL PRESENTATIONS with or without ancillary findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Acute chest pain (pericarditic or pseudo-ischemic)</td>
</tr>
<tr>
<td>• New-onset (days up to 3 months) or worsening dyspnea</td>
</tr>
<tr>
<td>or fatigue, with or without left/right heart failure</td>
</tr>
<tr>
<td>signs</td>
</tr>
<tr>
<td>• Palpitation, unexplained arrhythmia symptoms, syncope,</td>
</tr>
<tr>
<td>aborted sudden cardiac death</td>
</tr>
<tr>
<td>• Unexplained cardiogenic shock and/or pulmonary oedema</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ANCILLARY FINDINGS which support the clinical suspicion of myocarditis</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Fever ≥38.0°C within the preceding 30 days</td>
</tr>
<tr>
<td>• A respiratory or gastrointestinal infection</td>
</tr>
<tr>
<td>• Previous clinically suspected or biopsy proven myocarditis</td>
</tr>
<tr>
<td>• Peri-partum period</td>
</tr>
<tr>
<td>• Personal and/or family history of allergic asthma</td>
</tr>
<tr>
<td>• Other types of allergy</td>
</tr>
<tr>
<td>• Extra-cardiac autoimmune disease</td>
</tr>
<tr>
<td>• Toxic agents</td>
</tr>
<tr>
<td>• Family history of dilated cardiomyopathy, myocarditis</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>DIAGNOSTIC CRITERIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. ECG/Holter/stress test features: Newly abnormal ECG and/or Holter</td>
</tr>
<tr>
<td>and/or stress testing, any of the following:</td>
</tr>
<tr>
<td>• I to III degree atroventricular block, or bundle branch block,</td>
</tr>
<tr>
<td>ST/T wave changes (ST elevation or non ST elevation, T wave inversion),</td>
</tr>
<tr>
<td>• Sinus arrest, ventricular tachycardia or fibrillation and asystole,</td>
</tr>
<tr>
<td>atrial fibrillation, frequent premature beats, supraventricular tachycardia</td>
</tr>
<tr>
<td>• Reduced R wave height, intraventricular conduction delay (widened</td>
</tr>
<tr>
<td>QRS complex), abnormal Q waves, low voltage</td>
</tr>
<tr>
<td>II. Myocardiocytolysis markers: Elevated TnT/TnI</td>
</tr>
<tr>
<td>III. Functional/structural abnormalities on echocardiography</td>
</tr>
<tr>
<td>• New, otherwise unexplained LV and/or RV structure and function</td>
</tr>
<tr>
<td>abnormality (including incidental finding in apparently asymptomatic</td>
</tr>
<tr>
<td>subjects): regional wall motion or global systolic or diastolic</td>
</tr>
<tr>
<td>function abnormality, with or without ventricular dilatation, with or</td>
</tr>
<tr>
<td>without increased wall thickness, with or without pericardial effusion,</td>
</tr>
<tr>
<td>with or without endocavitary thrombi</td>
</tr>
<tr>
<td>IV. Tissue characterisation by CMR: Edema and/or LGE of classical</td>
</tr>
<tr>
<td>myocarditic pattern</td>
</tr>
</tbody>
</table>

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).

**ACUTE MYOCARDITIS: DIAGNOSTIC AND MANAGEMENT PROTOCOL**

History, Physical examination; ECG; Echocardiogram; Laboratory tests (Troponin, CRP, ESR, blood cell count, BNP); CMR; If available, serum cardiac autoantibodies

- Clinically suspected myocarditis
  - Consider coronary angiography and EMB
    - No coronary artery disease
      - Hemodynamically stable
        - Preserved LV function
        - No eosinophilia
        - No significant rhythm or conduction disturbances
        - Not associated with systemic immune disease*
          - General supportive therapy

  - Hemodynamically unstable, decreased LV function, cardiogenic shock
    - Pharmacological and, if needed, mechanical circulatory support (ECMO, LVAD/Bi-VAD, bridge to heart transplant or to recovery)

  - Lymphocytic
    - General supportive therapy
    - Immunosuppression if unresponsive and virus negative EMB

  - Giant cell, eosinophilic, sarcoidosis (acute decompensation)
    - Immunosuppression if infection-negative EMB

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

ACUTE PERICARDITIS: DIAGNOSIS

**DIAGNOSIS (≥ 2 of the following):**

- Chest pain (pleuritic) varying with position
- Pericardial friction rub
- Typical ECG changes (PR depression and/or diffuse concave ST-segment elevation)
- Echocardiography: new pericardial effusion

---

**Yes**

- **Myopericarditis if:**
  - ↑ Troponin
  - Echocardiography: ↓ LV-function

**Acute pericarditis**

- Delayed enhancement pericardium

---

**Equivocal or no**

- Consider cardiac MRI
- Consider alternative diagnoses
ACUTE PERICARDITIS: MANAGEMENT

**Acute pericarditis**

**High-risk features?**
- Fever >38°C
- Subacute onset
- Anticoagulated
- Trauma
- Immunocompromised
- Hypotension
- Jugular venous distension
- Large effusion

**Other causes**
- Post cardiac injury syndrome
- Post cardiac surgery
- Post MI: Dressler syndrome
- Uremic
- Neoplastic
- Collagen vascular diseases (e.g. SLE)
- Bacterial
- Tuberculous

**Yes**
- Hospital admission
- **Tamponade?**
  - **Yes**
  - Pericardiocentesis
  - **No**
  - Outpatient treatment

**No**
- Most frequent cause: Viral pericarditis

**Stable**
- Ibuprofen + colchicine
- Further testing for underlying etiology

**Hospital admission**
- Aspirin 800 mg or Ibuprofen 600 mg BID - 2 weeks
- If persisting or recurrent chest pain: Add **colchicine** 2.0 mg BID for 24 hours, followed by 0.5 to 1.0 mg BID for 6 months
  - **Avoid corticosteroids**!
CARDIAC TAMPOANDE: DIAGNOSIS AND MANAGEMENT

**Physical examination**
- Distended neck veins
- Shock
- Pulsus paradoxus
- Muffled heart sounds

**ECG**
- Sinus tachycardia
- Microvoltage QRS
- Electrical alternans

**Echocardiography with respirometer**
- Presence of a moderate to large pericardial effusion
- Diastolic collapses of right atrium and right ventricle
- Ventricular interdependence
- Increased tricuspid and pulmonary flow velocities (>50%) with decreased mitral and aortic flow velocities (>25%) during inspiration (predictive value >90%)

**Cardiac catheterization**
- Early
  - Right atrial pressure ↑
  - Loss of X-descent
- Late
  - Aortic pressure ↓
  - Pulsus paradoxus
  - Intracardiac diastolic pressure equilibration

**Percutaneous pericardiocentesis & drainage**
- Consider surgical drainage
- Avoid PEEP ventilation

**Not performed in routine**
## Abbreviations

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

EMB = Endomyocardial biopsy
EMS = Emergency medical services
EPS = Electrophysiological study
ERC = European Resuscitation Council
ESR = Erythrocyte sedimentation rate
ETT = Exercise 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
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
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

TIA = Transient ischemic attack
TLOC = Transient loss of consciousness
TTE = Transthoracic echocardiography
TTE = Transthoracic echocardiography
SpO$_2$ = Oxygen saturation
TEE = Transesophageal echocardiography
TEVAR = Thoracic endovascular aortic aneurysm repair
ESC Guidelines Acknowledgements


(19) McMurray JJ, Adamopoulos S, Anker SD, Auricchio A, Böhm M, Dickstein K et al. ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012: The Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2012 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association (HFA) of the ESC. Eur Heart J (2012);33(14):1787-847. doi: 10.1093/eurheartj/ehs104.Reproduced with permission of Oxford University Press (UK) © European Society of Cardiology, www.escardio.org/guidelines
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Acknowledgements
We are indebted to all the authors for their commitment and for the strong effort to synthesise their wide scientific knowledge and clinical experience into simple algorithms and schemes using the aim to help clinicians in everyday clinical practice in the easiest possible manner as the main driver of their work.

The support of this initiative by the ACCA board members was essential to launch this initiative as was the hard work of the ESC staff to make this project move forward.

The financial support of the sponsors, AstraZeneca and Novartis Pharma AG, made the development of the Toolkit easier. We appreciate the generous unrestricted educational grants and the independence to develop the Toolkit with no influence whatsoever in the selection of faculty, topics, clinical or scientific content.