The ACCA Clinical Decision-Making Toolkit is produced by the Acute Cardiovascular Care Association. Developed and distributed through an educational grant from AstraZeneca and Novartis Pharma AG. AstraZeneca and Novartis Pharma AG were not involved in the development of this publication and in no way influenced its contents.
Preface

The best care of patients with acute cardiovascular syndromes relies not only on specialists but also on systems of care that involve many non-cardiologists. Several of these syndromes require immediate diagnosis and decisions on treatment, some of them life-saving. Critical decisions must often be made quickly by professionals with different backgrounds and levels of expertise with limited resources. This poses a significant clinical challenge.

Against this background, the ACCA Clinical Decision-Making Toolkit was created as a comprehensive resource encompassing all aspects of acute cardiovascular care but structured as an easy-to-use instrument in environments where initial acute cardiovascular care is typically initiated. Comprehensive tables, clear diagrams and algorithms, based on the ESC clinical practice guidelines as well as in clinical experience should provide diagnostic and therapeutic guidance at a glance.

The Second Edition of the ACCA Toolkit has been updated with the 2014 and 2015 ESC Guidelines, and enriched with a new chapter with up-to-date coverage of drugs most frequently used in acute cardiovascular care. However, it does not replace textbooks and other sources of information that need to be consulted to reach an optimal management of these patients.

The ACCA Toolkit is available through different platforms:
Printed booklet, available at congresses where ESC-ACCA is represented
Web-based pdf file downloadable at www.escardio.org/ACCA
Mobile application for smartphones/tablets available in both Apple & Googleplay stores

Héctor Bueno, M.D., PhD., FESC, FAHA
Editor in Chief
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CHAPTER 1: KEY SYMPTOMS

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### Initial assessment of patients with CHEST PAIN

<table>
<thead>
<tr>
<th></th>
<th>Low Likelihood</th>
<th>High Likelihood</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Presentation</td>
<td>![Images]</td>
<td>![Image]</td>
</tr>
<tr>
<td>2. ECG</td>
<td>![Images]</td>
<td>![Images]</td>
</tr>
<tr>
<td>3. Troponin</td>
<td>![Images]</td>
<td>![Images]</td>
</tr>
<tr>
<td>4. Diagnosis</td>
<td>Noncardiac</td>
<td>UA</td>
</tr>
</tbody>
</table>

STEMI = ST-elevation myocardial infarction; NSTEMI = non-ST-elevation myocardial infarction; UA = unstable angina.

Reference: Roffi et Al. Eur Heart J 2015;eurheartj.ehv320
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>
<tbody>
<tr>
<td><strong>Arguments for vital risk</strong></td>
<td>• Cardiorespiratory arrest, syncope / loss of consciousness, neurological defect</td>
<td>• Normal consciousness</td>
</tr>
<tr>
<td></td>
<td>• Dyspnea</td>
<td>• Normal breathing (see chapter 1.1 page 9)</td>
</tr>
<tr>
<td></td>
<td>• Nausea – vomiting</td>
<td>• Normal heart rhythm</td>
</tr>
<tr>
<td></td>
<td>• Arrhythmias – tachycardia</td>
<td></td>
</tr>
<tr>
<td><strong>Context, CV risk</strong></td>
<td>Age &gt; 40 years, previous CV disease (MI, stroke, PE), modifiable CV risk factors</td>
<td>• Age &lt; 40 years,</td>
</tr>
<tr>
<td></td>
<td>(smoker, HTN, hypercholesterolemia, diabetes), chronic CV treatment</td>
<td>• No previous CV disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• No CV risk factors</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• No chronic treatment</td>
</tr>
<tr>
<td><strong>Chest Pain</strong></td>
<td>Medial / lateral thoracic pain, intense, with dyspnea</td>
<td>• Depends on position/ palpation/ movements</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Variable intensity, short duration (&lt;1 min)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Hyperthermia</td>
</tr>
<tr>
<td><strong>Cardiac Ischemic Pain</strong></td>
<td>Retro-sternal, constriction, jaw/cervical/arm/back irradiation, spontaneous, prolonged &gt; 20 min + dyspnea, sweating, lightheadedness, nausea</td>
<td>• Lateral, abdominal irradiation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• No neuro-vegetative symptoms</td>
</tr>
</tbody>
</table>
Approach after first call for out-of-hospital CHEST PAIN

Arguments for vital risk? (see chapter 1.1 page 3)

Emergency transport with trained medical team

Yes

No

Origin of Chest Pain?

Acute Cardiac Disease

High probability for ACS

Low probability for ACS

No Acute Cardiac Disease

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

Hospital admission to the Emergency Department

Emergency transport with trained medical team

Cardiology ward

Non-cardiology ward

Discharge after prolonged observation
**Acute Cardiac Disease**

**Emergency transport**
with trained medical team

**ECG, decision for reperfusion, antithrombotics, immediate transport to ED/cathlab**
*(see chapter 2)*

**No Acute Cardiac Disease**

**Emergency transport**

**Hospital admission to the Emergency Department**

- **Cardiology ward**
- **Non-cardiology ward**
- **Discharge after prolonged observation**

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

**Arguments for vital risk?** *(see chapter 1.1 page 3)*
# 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 chapter 2.1 page 24)* |
| **STEMI NSTEACS Uncertain diagnosis (see chapter 2.1 page 24)** | • ECG criteria for STEMI *(see chapter 2.3 page 35)*  
• ST depression or normal ECG  
• Normal ECG → Repeat 12-lead ECG recording | • Other ST-segment abnormalities not related to STEMI *(see chapter 2.3)* |
| **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** | | |
First medical contact in patients with CHEST PAIN (home-ambulance)

Hemodynamic, respiratory or neurological distress? (see chapter 1.1 page 8)

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

No ➔ ECG <10 min ➔ ACS?

High probability ➔ ST-segment elevation

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)

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)

Type of reperfusion (primary PCI or fibrinolysis)
Record times (onset, call, contact)

Start antiplatelet and anticoagulant treatment
Transfer to a center with cath-lab
Management of patients with CHEST PAIN (emergency room)

Hemodynamic, respiratory or neurological distress? (see chapter 1.1 page 6)

Yes → Resuscitation, hemodynamic or respiratory support (See chapters 3 & 4)

No → STEMI, NSTEACS with persistent pain, Hemodynamic distress

Direct transfer to cath-lab

STEMI (see chapter 2)

Other CVD or No ACS

No direct transfer to cath-lab → ED, Chest Pain Unit, cardiology ward, other wards

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

- 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
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
- SpO₂ <85%
- SBP <90 mmHg
- HR >120 bpm

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

**Acute heart failure**
**Acute coronary syndrome**

**Pneumonia**

**Exacerbated COPD**
or other chronic lung disease

**Pulmonary embolism**

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

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

## 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

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)
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• 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 → CT-angio

Result 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
DYSPNEA: ACUTE PULMONARY EMBOLISM
(see chapter 6.2)

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

Hemodynamically unstable
Hemodynamically stable
Intermediate probability
Total score 2-6
High probability
Total score >6
Low probability
Total score <2

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

PE confirmed: Treatment
(see chapter 6.2)

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

Result inconclusive
→ CT-angio

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

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 → \( \text{SpO}_2 \) 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)

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

- Hospitalisation indicated?
- Evaluate ICU criteria
- NIV indicated?

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

- Follow-up

DYSPNEA: Community-acquired pneumonia

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

Definition
- Chest X-ray if dyspnea & cough
- Laboratory workup clinical chemistry; BGA; procalcitonin
- Sputum if patient admitted
- Blood cultures (2x2) if patient admitted
- Legionella antigen (urine) if Legionellosis suspected
- Pneumococcus antigen (urine) 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 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 (I)**
**Diagnostic criteria with initial evaluation**

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

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

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

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

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

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

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

1. **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><strong>Recommendations: Active standing</strong></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><strong>Indications</strong></td>
</tr>
<tr>
<td><strong>Diagnostic criteria</strong></td>
<td><strong>Diagnostic criteria</strong></td>
</tr>
<tr>
<td>• CSM is diagnostic if syncope is reproduced in presence of asystole longer than 3 s and/or a fall in systolic BP &gt;50 mmHg</td>
<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>
</tr>
<tr>
<td></td>
<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>
</tr>
</tbody>
</table>

## Treatment according to type of SYNCPE (I)

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

### Treatment of arrhythmic syncope

<table>
<thead>
<tr>
<th>Cardiac Pacing</th>
<th>Catheter ablation</th>
</tr>
</thead>
<tbody>
<tr>
<td>• 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</td>
<td>• 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)</td>
</tr>
<tr>
<td>• Pacing is indicated in sinus node disease patients with syncope and abnormal CSNRT</td>
<td>• Catheter ablation may be indicated in patients with syncope due to the onset of rapid atrial fibrillation</td>
</tr>
<tr>
<td>• Pacing is indicated in sinus node disease patients with syncope and asymptomatic pauses &gt; 3 sec. (with possible exceptions of young trained persons, during sleep and in medicated patients)</td>
<td></td>
</tr>
<tr>
<td>• Pacing is indicated in patients with syncope and 2nd degree Mobitz II, advanced or complete AV block</td>
<td></td>
</tr>
<tr>
<td>• Pacing is indicated in patients with syncope, BBB and positive EPS</td>
<td></td>
</tr>
<tr>
<td>• Pacing should be considered in patients with unexplained syncope and BBB</td>
<td></td>
</tr>
<tr>
<td>• Pacing may be indicated in patients with unexplained syncope and sinus node disease with persistent sinus bradycardia itself asymptomatic</td>
<td></td>
</tr>
<tr>
<td>• Pacing is not indicated in patients with unexplained syncope without evidence of any conduction disturbance</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Antiarrhythmic drug therapy</th>
<th>Implantable Cardioverter Defibrillator (ICD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Antiarrhythmic drug therapy, including rate control drugs, is indicated in patients with syncope due to onset of rapid atrial fibrillation</td>
<td>• ICD is indicated in patients with documented VT and structural heart disease</td>
</tr>
<tr>
<td>• 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</td>
<td>• ICD is indicated when sustained monomorphic VT is induced at EPS in patients with previous myocardial infarction</td>
</tr>
<tr>
<td></td>
<td>• ICD should be considered in patients with documented VT and inherited cardiomyopathies or channelopathies</td>
</tr>
</tbody>
</table>

CHAPTER 2: ACUTE CORONARY SYNDROMES

2.1 GENERAL CONCEPTS ........................................................................................................ p.24
   H. Bueno

2.2 NON ST-SEGMENT ELEVATION ACS .............................................................................. p.29
   H. Bueno

2.3 ST-SEGMENT ELEVATION MI (STEMI) ........................................................................... p.35
   D. Zahger, P. Clemmensen
ACUTE CORONARY SYNDROMES: Diagnosis (1)

CHEST PAIN
or symptoms suggestive of myocardial ischemia

ECG

ST elevation
(persistent)

LBBB

ST/T abnormalities

Normal ECG

Pain resolves with nitroglycerin

Yes

1st hsTn

hs-cTn > ULN

hs-cTn < ULN

Pain onset <6h

Pain onset >6h

Re-test hs-cTn (3h later)
See next page for 1h rule-in & rule-out algorithm

hs-cTn > 5 ULN

or clinical diagnosis clear

Potential noncardiac causes for abnormal Tn

hs-cTn > x5 ULN

\( \Delta \) hs-cTn

(1 value > ULN)

hs-cTn no change

Work-up differential diagnoses

NSTEMI

Unstable Angina

STEMI

Consider STEMI

Reference: Roffi M. Eur Heart J 2015;eurheartj.ehv320
• NSTEMI can be ruled-out at presentation, if hs-cTn concentration is very low
• NSTEMI can be ruled out by the combination of low baseline levels and the lack of a relevant increase within 1 h
• NSTEMI is highly likely if initial hs-cTn concentration is at least moderately elevated or hs-cTn concentrations show a clear rise within the first hour

Reference: Roffi M. Eur Heart J 2015;eurheartj.ehv320
<table>
<thead>
<tr>
<th>Causes of chest pain</th>
<th>Causes of troponin elevation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Not related to ACS</strong></td>
<td><strong>Not related to ACS</strong></td>
</tr>
<tr>
<td><strong>Primary cardiovascular</strong></td>
<td><strong>Primary cardiovascular</strong></td>
</tr>
<tr>
<td>• Acute pericarditis, pericardial effusion</td>
<td>• Acute myo(per)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 or cardiac procedures (ablation, 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>
<tr>
<td><strong>Primary non-cardiovascular</strong></td>
<td><strong>Primary non-cardiovascular</strong></td>
</tr>
<tr>
<td>• Oesophageal spasm, oesophagitis, Gastro Esophageal Reflux (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 myocardial infarction</td>
</tr>
<tr>
<td>Pacemaker stimulation</td>
<td>Chronic ischemic heart disease</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>Electrolyte disturbances (hyperkalemia)</td>
<td>Metabolic or ionic disturbances</td>
</tr>
<tr>
<td>Acute brain damage (stroke, subarachnoid haemorrhage)</td>
<td></td>
</tr>
<tr>
<td>Tako Tsubo syndrome</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 ischemic heart disease</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>Electrolyte 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>Tako Tsubo syndrome</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)
   - ACS unclear (Rule out ACS)
   - No ACS
     - Chest Pain Unit (see chapter 1.1)
     - Rule out noncardiac causes

4. **Medical Treatment**
   - Anti-ischemic therapy
   - Antiplatelet therapy
   - Anticoagulation

5. **Invasive Strategy**
   - Primary PCI
   - Thrombolysis
     - For STEMI if primary PCI not timely available
   - 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 (1)

<table>
<thead>
<tr>
<th>Ischemic risk</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Grace risk score</strong></td>
<td><strong>Timi risk score</strong></td>
</tr>
<tr>
<td><strong>Predictive Factors</strong></td>
<td><strong>Predictive Factors</strong></td>
</tr>
<tr>
<td>- Age</td>
<td>- Age ≥ 65 years</td>
</tr>
<tr>
<td>- HR*</td>
<td>- At least 3 risk factors for CAD</td>
</tr>
<tr>
<td>- SBP*</td>
<td>- Significant (&gt;50%) coronary stenosis</td>
</tr>
<tr>
<td>- Creatinine (mg/dl)*</td>
<td>- ST deviation</td>
</tr>
<tr>
<td>- Killip class*</td>
<td>- Severe anginal symptoms (&gt;2 events in last 24 h)</td>
</tr>
<tr>
<td>- Cardiac arrest*</td>
<td>- Use of aspirin in last 7 days</td>
</tr>
<tr>
<td>- ST-segment deviation</td>
<td>- Elevated serum cardiac markers</td>
</tr>
<tr>
<td>- Elevated cardiac markers</td>
<td></td>
</tr>
</tbody>
</table>

**Outcomes**
- In-hospital, 6-month, 1-year and 3-year mortality
- 1-year death/MI

**Risk calculation**
- www.gracescore.org
- www.timi.org

*At admission.*
## NON ST-SEGMENT ELEVATION ACS: Risk stratification (2)

### 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\(^*\)

**Outcome**
In-hospital major bleeding

\(^*\) At admission.

---


NON ST-SEGMENT ELEVATION ACS: Treatment (1)

### General overview

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

- **Pharmacological treatment***

  - Antithrombotic therapy
  - Anti ischemic treatment
  - Other preventive therapies

#### Antithrombotic therapy
- One of the following:
  - Fondaparinux
  - Enoxaparin
  - UFH
  - Bivalirudin

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

#### Other preventive therapies
- Statins
- ACE inh. (or ARB)
- Aldosterone inhibitors

### For more information on individual drug doses and indications, see chapter 8: Use of drugs in Acute Cardiovascular Care.
NON ST-SEGMENT ELEVATION ACS: Treatment (2)
Antithrombotic strategies in patients with NSTE-ACS and non-valvular atrial fibrillation

Management strategy

NSSTE-ACS patients with non-valvular atrial fibrillation

PCI

Medically managed / CABG

Bleeding risk

Low to intermediate (e.g. HAS-BLED = 0–2)

High (e.g. HAS-BLED ≥ 3)

Triple therapy

Triple or dual therapy

Dual therapy

Dual therapy

Dual therapy

Monotherapy

CHA2DS2-VASc = Cardiac failure, Hypertension, Age ≥ 75 [2 points], Diabetes, Stroke [2 points] – Vascular disease, Age 65–74, Sex category.

a Dual therapy with oral anticoagulation and clopidogrel may be considered in selected patients (low ischaemic risk).

b Aspirin as an alternative to clopidogrel may be considered in patients on dual therapy (i.e., oral anticoagulation plus single antiplatelet); triple therapy may be considered up to 12 months in patients at very high risk for ischaemic events.

c Dual therapy with oral anticoagulation and one antiplatelet agent (aspirin or clopidogrel) beyond one year may be considered in patients at very high risk of coronary events.

d In patients undergoing coronary stenting, dual antiplatelet therapy may be an alternative to triple or a combination of anticoagulants and single antiplatelet therapy if the CHA2DS2-VASc score is 1 (males) or 2 (females).

Reference: Eur Heart J 2015;eurheartj.ehv320- Figure 5.
### Risk criteria mandating invasive strategy in NSTE-ACS

| Very-high-risk criteria | • Haemodynamic instability or cardiogenic shock  
| • Recurrent or ongoing chest pain refractory to medical treatment  
| • Life-threatening arrhythmias or cardiac arrest  
| • Mechanical complications of MI  
| • Acute heart failure  
| • Recurrent dynamic ST-T wave changes, particularly with intermittent ST-elevation |
| --- | --- |
| High-risk criteria | • Rise or fall in cardiac troponin compatible with MI  
| • Dynamic ST- or T-wave changes (symptomatic or silent)  
| • GRACE score >140 |
| Intermediate-risk criteria | • Diabetes mellitus  
| • Renal insufficienty (eGFR <60 mL/min/1.73 m$^2$)  
| • LVEF <40% or congestive heart failure  
| • Early post-infarction angina  
| • Prior PCI  
| • Prior CABG  
| • GRACE risk score >109 and <140 |
| Low-risk criteria | • Any characteristics not mentioned above |

Reference: Roffi M. Eur Heart J 2015;eurheartj.ehv320
NON ST-SEGMENT ELEVATION ACS: Treatment (4)
Timing and strategy for invasive management

Symptoms Onset

First medical contact → NSTE-ACS diagnosis

PCI center

EMS or Non–PCI center

Very high → Immediate transfer to PCI center

High → Same-day transfer

Intermediate → Transfer

Low → Transfer optional

Immediate invasive (<2 hr)

Early invasive (<24 hr)

Invasive (<72 hr)

Non-invasive testing if appropriate

Risk stratification

Therapeutic strategy

Reference: Eur Heart J 2015;eurheartj.ehv320 - Figure 6.
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 an overlooked left dominant circumflex using posterior leads in the fifth interspace
- ST elevation in right precordial leads ($V_3R$ and $V_4R$), using a cut-off point $> 0.05$ mV, and $> 0.1$ mV in men $< 30$ years
  → Capture suspected right ventricular infarction using right precordial leads

STEMI: Treatment (I)
General overview of initial management

STEMI diagnosis*

Primary-PCI capable center

Preferably <60 min

Primary-PCI

Preferably ≤90 min
(≤60 min in early presenters)

Rescue PCI

Immediately

No

Coronary angiography

Yes

EMS or non primary-PCI capable center

PCI possible <120 min?

Immediate transfer to PCI center

Yes

Immediate transfer to PCI center

No

Immediate fibrinolysis

Successful fibrinolysis?

Immediate transfer to PCI center

*The time point the diagnosis is confirmed with patient history and ECG ideally within 10 min from First Medical Contact (FMC). All delays are related to FMC.

For more information on individual drug doses and indications, see chapter 8: Use of drugs in Acute Cardiovascular Care.

<table>
<thead>
<tr>
<th>Pre hospital</th>
<th>PCI</th>
<th>CCU/ICCU</th>
<th>Medication Titration Day 2-7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetylicsalislyc Acid</td>
<td>300 mg</td>
<td>Bivalirudin or GPI: Eptifibatide, Tirofiban, Abxicimab</td>
<td>Acetylicsalislyc Acid 75 mg</td>
</tr>
<tr>
<td>Heparin</td>
<td>70 IU/kg</td>
<td>Follow local in-lab instruction / dosing</td>
<td>Ticagrelor 90 mg x 2</td>
</tr>
<tr>
<td>Ticagrelor</td>
<td>180 mg</td>
<td></td>
<td>or Prasugrel 10/5 mg x 1</td>
</tr>
<tr>
<td>or Prasugrel</td>
<td>60 mg</td>
<td></td>
<td>or Clopidogrel 75 mg x 1</td>
</tr>
<tr>
<td>or Clopidogrel</td>
<td>600 mg</td>
<td>Metoprolol 25 mg x 2</td>
<td>[Atorvastatin 80 mg x 1]</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>or Rosuvastatin 40 mg x 1</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Start ACE-i or ARB in DM, LVSD, CHF, or to control BP</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Aldosterone RB</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Start or continue anti-diabetic medication</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Pre-hospital management of patients with chest pain and/or dyspnoea of cardiac origin. A position paper of the Acute Cardiovascular Care Association (ACCA) of the ESC - European Heart Journal: Acute Cardiovascular Care August 27, 2015 2048872615604119.
CHAPTER 3: ACUTE HEART FAILURE

3.1 HEART FAILURE AND PULMONARY OEDEMA ......................... p.40
I.C.C. van der Horst, G. Filippatos

3.2 CARDIOGENIC SHOCK ........................................................................ p.52
P. Vranckx, U. Zeymer
ACUTE HEART FAILURE: Diagnosis and causes (I)

Rapid onset of, or worsening of symptoms and signs of heart failure*

History of heart failure

Cardiovascular risk profile*

Precipitating factors*

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 41).
ACUTE HEART FAILURE: Diagnosis and causes (2)

1• **Symptoms:** Dyspnea (on effort or at rest)/breathlessness, fatigue, orthopnea, cough, weight gain/ankle swelling

2• **Signs:** Tachypnea, tachycardia, low or normal blood pressure, raised jugular venous pressure, 3rd/4th heart sound, rales, oedema, intolerance of the supine position

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 respiratory distress syndrome, (severe) anaemia, hyperventilation (acidosis), sepsis/septic shock, redistributive/hypovolemic shock

6• **Likelihood:** Depending on the site of presentation the underlying cause of acute heart failure is likely to differ. Cardiologists see more often worsening heart failure and physicians at the Emergency Department more often see patients with preserved systolic left ventricular function

**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

**RESUSCITATION AREA/CCU/ICU**

To stabilize vital signs (echo if needed) and/or immediate non-invasive ventilation (see chapter 3.1 page 44).

**DIAGNOSTIC TESTS**
- ECG
- Laboratory tests (see chapter 3.1 page 44)
- Echo (lung, heart)
- Chest X-ray

**SEVERITY SCORE** (excluding shock)

- **Respiratory distress**
  - RR > 25/min,
  - SpO₂ < 90% on O₂,
  - or increased work of breathing

- **Haemodynamic instability**
  - Low or high blood pressure,
  - Severy arrhythmia,
  - HR < 40 or > 130/min

**SUSPECTED ACUTE HEART FAILURE**

- **SHOCK**
  - Yes → Ventilation support – Echocardiogram – ICU/CCU
  - No → High risk ACS
    - Yes → Cardiac catheterisation laboratory
    - No → INITIAL 30-60 MIN

**INITIAL 30-60 MIN**
- **Ventilation support**
- **Echocardiogram**
- **ICU/CCU**
- **Cardiac catheterisation laboratory**
RESUSCITATION AREA/CCU/ICU
To stabilize vital signs (echo if needed) and/or immediate non-invasive ventilation (see chapter 3.1 page 44)

DIAGNOSTIC TESTS
• ECG
• Laboratory tests (see chapter 3.1 page 44)
• Echo (lung, heart)
• Chest X-ray

IV THERAPY
• See chapter 3.1 page 47

ACUTE HEART FAILURE: Initial diagnosis and treatment
Airway (A) & Breathing (B)

AB → Sufficient oxygenation (SpO₂ > 90%) → No → OXYGEN* (+ oropharyngeal airway [Guedel/Mayo]/nasopharyngeal airway and upright position)

- 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%

Yes → ~5 minutes to reassess

Yes → Sufficient oxygenation (SpO₂ > 90%)

No → Oxygen* + Positive End-Expiratory Pressure (PEEP) 5-7.5 mmHg

Start CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP)

~15 minutes to reassess

Yes → Sufficient ventilation (pCO₂ < 45 mmHg)**

No → ~15 minutes to reassess
ACUTE HEART FAILURE: INITIAL DIAGNOSIS AND TREATMENT

AIRWAY (A) & BREATHING (B)

Sufficient oxygenation (SpO\textsubscript{2} > 90%)

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

Sufficient ventilation

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

OXYGEN*

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

Yes
No

~5 minutes to reassess
~15 minutes to reassess
No

Start CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP)

3.1 p.45

* Goal SpO\textsubscript{2} 94-98%.
** Use the predefined liters of oxygen. When using higher flows the FiO\textsubscript{2} will drop.
*** For a patient with COPD, a pCO\textsubscript{2} of 45-50 mmHg may be optimal. Aim for a normal pH.
**** Consider if the above fails or when patient is fatigued.
**C - CIRCULATION**

- HR (bradycardia [<60/min], normal [60-100/min], tachycardia [>100/min]), rhythm (regular, irregular), SBP (very low [<90 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, MR-proANP)
- Complete blood count, electrolytes, creatinine, urea, glucose, inflammation, TSH

**Standard 12-lead ECG**
- Venous blood gases, D-dimer (suspicion of acute pulmonary embolism)
- 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?

**D – DISABILITY DUE TO NEUROLOGICAL DETERIORATION**

- Normal consciousness/altered mental status? Measurement of mental state with AVPU (alert, visual, pain or unresponsive) 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 (<0.5ml/kg/hr) → Insert indwelling catheter; the benefits should outweigh the risks of infection and long-term complications

**ACUTE HEART FAILURE: Initial diagnosis (CDE)**

ACUTE HEART FAILURE: Initial treatment (C) IV therapy

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

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 to start ACE-I/ARB, beta-blocker, MRA.

*See Chapter 8: use of drugs in acute Cardiovascular Care.
(See table page 50-51)

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 increase to 5 microg/kg/min

2 Diuretics i.v.
- Furosemide 20-40 mg bolus, continuous 100 mg/6 h

3 Consider to start ACE-I/ARB, beta-blocker, MRA.

*See Chapter 8: use of drugs in acute Cardiovascular Care.
(See table page 50-51)

* Use higher dose in patients on chronic diuretic treatment for HF (i.e. 2.5 times normal dose).
MANAGEMENT OF ACUTE HEART FAILURE

DIAGNOSTIC TESTS

- No acute heart failure
- Confirmed acute heart failure

MONITORING
  - Dyspnea (VAS, RR), BP, SpO₂, HR and rhythm, urine output, peripheral perfusion

TREATMENT OBJECTIVES
to prevent organ aggravation:
  - Improve symptoms, maintain SBP >90 mmHg and peripheral perfusion, maintain SpO₂ >90% (see table page 50-51)

REASSESSMENT
  - Clinical, biological and psychosocial parameters by trained nurses

OBSERVATION UP TO 120 MIN
### ACUTE HEART FAILURE: Treatment (C) and preventive measures

**Management of oral therapy in AHF in the first 48 hours**

<table>
<thead>
<tr>
<th>Normotension/Hypertension</th>
<th>Hypotension</th>
<th>Low Heart rate</th>
<th>Potassium</th>
<th>Renal impairment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;100 mmHg</td>
<td>&lt;60 bpm</td>
<td>&lt;50 bpm</td>
<td>≤3.5 mg/dL</td>
</tr>
<tr>
<td></td>
<td>&gt;85 mmHg</td>
<td>≥50 bpm</td>
<td></td>
<td>&gt;5.5 mg/dL</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cr &lt; 2.5, eGFR &gt; 30</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cr &gt; 2.5, eGFR &lt; 30</td>
</tr>
<tr>
<td>ACE-I/ARB</td>
<td>Review/increase</td>
<td>Reduce/stop</td>
<td>No change</td>
<td>Review/increase</td>
</tr>
<tr>
<td>Beta-blocker</td>
<td>No change</td>
<td>Reduce/stop</td>
<td>Stop</td>
<td>No change</td>
</tr>
<tr>
<td>MRA</td>
<td>No change</td>
<td>No change</td>
<td>Stop</td>
<td>No change</td>
</tr>
<tr>
<td>Diuretics</td>
<td>Increase</td>
<td>Reduce</td>
<td>Stop</td>
<td>Review/increase</td>
</tr>
</tbody>
</table>

CCB, calcium channel blockers (mg/dL); Cr, creatinine blood level (mg/dL); eGFR, estimated glomerular filtration rate ml/min/1.73 m2; MRA, mineralocorticoid receptor antagonist; (*) amiodarone. - Depicted from Mebazaa A et al. Eur J Heart Fail. (2015);17(6):544-58.
Thrombosis prophylaxis should be started in patients not anticoagulated (enoxaparin 1 mg/kg as first dose)
Maintain an adequate nutritional status with a nutritional support of 20-25 kcal/kg/day within the first 48 hours

CCB, calcium channel blockers (mg/dL); Cr, creatinine blood level (mg/dL); eGFR, estimated glomerular filtration rate ml/min/1.73 m²; MRA, mineralocorticoid receptor antagonist; (*) amiodarone. - Depicted from Mebazaa A et al. Eur J Heart Fail. (2015);17(6):544-58.
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.
This protocol should be initiated as soon as cardiogenic shock/end organ hypoperfusion is recognised and should not be delayed pending intensive care admission.

### CARDIOGENIC SHOCK: Initial triage and management

<table>
<thead>
<tr>
<th>0 min</th>
<th><strong>EARLY TRIAGE &amp; MONITORING</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Start high flow O₂</td>
</tr>
<tr>
<td></td>
<td>Establish i.v. access</td>
</tr>
</tbody>
</table>

- Age: 65–74, ≥75
- Heart rate >100 beats per minute
- Systolic blood pressure <100 mmHg
- Proportional pulse pressure ≤25 mmHg (CI <2.2l/min/m²)
- Orthopnea (PCWP >22 mmHg)
- Tachypnea (>20/min), >30/min (!)
- Killip class II-IV
- Clinical symptoms of tissue hypoperfusion/hypoxia:
  - cool extremities, decreased urine output (urine output <40 ml/h)
  - decreased capillary refill or mottling, alteration in mental status

<table>
<thead>
<tr>
<th>5 min</th>
<th><strong>INITIAL RESUSCITATION</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Arterial and a central venous catheterization with a catheter capable of measuring central venous oxygen saturation</td>
</tr>
<tr>
<td></td>
<td>Standard transthoracic echocardiogram to assess left (and right) ventricular function and for the detection of potential mechanical complications following MI</td>
</tr>
<tr>
<td></td>
<td>Early coronary angiography in specialized myocardial intervention center when signs and/or symptoms of ongoing myocardial ischemia (e.g. ST segment elevation myocardial infarction).</td>
</tr>
</tbody>
</table>

| 15 min | **CORRECT**: hypoglycemia & hypocalcemia, **TREAT**: sustained arrhythmias: brady- or tachy- |
|        | Isotonic saline-fluid challenge of 20 to 30 ml per kilogram of body weight over a 30-minute period to achieve a central venous pressure of 8 to 12 mmHg or until perfusion improves (with a maximum of 500 ml) |
|        | CONSIDER NIV/mechanical ventilation for comfort (fatigue, distress) or as needed: - To correct acidosis - To correct hypoxemia |
|        | **INOTROPIC SUPPORT** (dobutamine and/or vasopressor support) |

<table>
<thead>
<tr>
<th>60 min</th>
<th><strong>TREATMENT GOALS</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>a mean arterial pressure of 60 mmHg or above,</td>
</tr>
<tr>
<td></td>
<td>a mean pulmonary artery wedge pressure of 18 mmHg or below,</td>
</tr>
<tr>
<td></td>
<td>a central venous pressure of 8 to 12 mmHg,</td>
</tr>
<tr>
<td></td>
<td>a urinary output of 0.5 ml or more per hour per kilogram of body weight</td>
</tr>
<tr>
<td></td>
<td>an arterial pH of 7.3 to 7.5</td>
</tr>
<tr>
<td></td>
<td>a central venous saturation (ScvO₂) ≥70% (provided SpO₂ ≥93% and Hb level ≥9 g/dl)</td>
</tr>
</tbody>
</table>

In persistent drug-resistant cardiogenic shock, consider mechanical circulatory support
Some patients with CS will require increased PEEP to attain functional residual capacity and maintain oxygenation, and peak pressures above 30 cm H2O to attain effective tidal volumes of 6-8ml/kg with adequate CO2 removal.

<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 H2O</td>
</tr>
<tr>
<td>Anticipated PEEP levels</td>
<td>5-10 cm H2O</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>• PaO₂</td>
<td>&gt; 90%</td>
</tr>
<tr>
<td>• SpO₂</td>
<td></td>
</tr>
</tbody>
</table>

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

- No
  - Emergency echocardiography ± Tissue doppler imaging ± Color flow imaging
- Yes
  - 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

Pump failure RV, LV, both

- Acute severe mitral valve regurgitation
- Ventricular septum rupture
- Severe aortic/mitral valve stenosis

Aortic dissection Pericardial tamponade

Operating theater ± coronary angiography

PCI ± stenting of the culprit lesion

CABG + correct mechanical complications
**CAR Diogenic SHOCK:**
Mechanical circulatory support, basic characteristics

<table>
<thead>
<tr>
<th>Time Period</th>
<th>Support Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>72-hrs</td>
<td>IABP, Impella 2,5, Impella 5,0, Tandem-heart, Levitronix, ECMO, Implantable</td>
</tr>
<tr>
<td>2-weeks</td>
<td>Partial support, Full support</td>
</tr>
<tr>
<td>1-month</td>
<td>BiVentricular support</td>
</tr>
</tbody>
</table>

**Level of support**
- **Left ventricular support**
- **BiVentricular support**
- **Partial support**
- **Full support**
- **Pulmonary support**
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.

The SAVE-score may be a tool to predict survival for patients receiving ECMO for refractory cardiogenic shock (www.save-score.com).
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**

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

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

- **Victim unresponsive**
  - Shout for help
  - Open airway
  - Assess breathing

- **Not breathing normally**
  - Call for an ambulance
  - Start CPR 30:2
  - Send or go for an AED

- **AED not available**
  - 30 chest compressions: 2 rescue breaths

- **As soon as AED arrives**
  - Start AED, listen to and follow voice prompts
  - AED Assesses rhythm

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

**Approach safely**

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

VICTIM COLLAPSES

Victim responds

Victim unresponsive

Leave victim as found

Find out what is wrong

Reassess victim regularly

Shout for help

Open airway

Assess breathing

Not breathing normally

Call for an ambulance

Start CPR 30:2

Send or go for an AED

As soon as AED arrives

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

No shock advised

Immediately resume CPR 30:2 for 2 min

Shock advised

1 shock

Immediately resume CPR 30:2 for 2 min

Immediately resume CPR 30:2 for 2 min

Breathing normally

Put victim in recovery position and call for an ambulance

Continue until victim starts to wake up: to move, open eyes, and breathe normally
IN-HOSPITAL CARDIAC ARREST: Assessment of a collapsed victim and initial treatment

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
Yes/No

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

Call resuscitation team
CPR 30:2 with oxygen and airway adjuncts

Apply pads/monitor
Attempt defibrillation if appropriate

Advanced Life Support when resuscitation team arrives

Call resuscitation team if appropriate

Handover to resuscitation team
IN-HOSPITAL CARDIAC ARREST: Advanced life support

Unresponsive and not breathing normally?

CPR 30:2
Attach defibrillator/monitor
Minimise interruptions

Assess rhythm

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

Call resuscitation team

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

Immediately resume:
- CPR for 2 min
- Minimise interruptions

- Use ABCDE approach
- Aim for SaO₂ 94-98%
- Aim for normal PaCO₂
- 12-lead ECG
- Treat precipitating cause
- Temperature control / Therapeutic hypothermia

CONSIDER
- Ultrasound imaging
- Mechanical chest compressions to facilitate transfer/treatment
- Coronary angiography and PCI
- Extracorporeal CPR
IN-HOSPITAL CARDIAC ARREST: ADVANCED LIFE SUPPORT

Unresponsive and not breathing normally?
Assess rhythm
CPR 30:2
Attach defibrillator/monitor

DURING CPR
- Ensure high-quality chest compressions
- Minimise interruptions to compressions
- Give Oxygen
- Use waveform capnography
- Continuous chest compressions when advanced airway in place
- Vascular access (intravenous, intraosseous)
- Give adrenaline every 3-5 min
- Give amiodarone after 3 shocks
- Correct reversible causes

IMMEDIATE POST CARDIAC ARREST TREATMENT
- Use ABCDE approach
- Aim for $\text{SaO}_2$ 94-98%
- Aim for normal $\text{PaCO}_2$
- 12-lead ECG
- Treat precipitating cause
- Temperature control / Therapeutic hypothermia

CONSIDER
- Ultrasound imaging
- Mechanical chest compressions to facilitate transfer/treatment
- Coronary angiography and PCI
- Extracorporeal CPR

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

IMMEDIATELY CALL RESUSCITATION TEAM

Shockable (VF/Pulseless VT)
1 Shock
Return of spontaneous circulation
Immediately resume: CPR for 2 min
Minimise interruptions

NON-SHOCKABLE (PEA/Asystole)
Immediately resume: CPR for 2 min
Minimise interruptions
- Use ABCDE approach
- Aim for $\text{SaO}_2$ 94-98%
- Aim for normal $\text{PaCO}_2$
- 12-lead ECG
- Treat precipitating cause
- Temperature control / Therapeutic hypothermia

CONSIDER
- Ultrasound imaging
- Mechanical chest compressions to facilitate transfer/treatment
- Coronary angiography and PCI
- Extracorporeal CPR

REFERENCES
- Thrombosis
- Tamponade - cardiac
- Toxins
- Tension pneumothorax
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.

IN-HOSPITAL CARDIAC ARREST: Drug therapy during advanced life support
CHAPTER 5: RHYTHM DISTURBANCES

5.1 SUPRAVENTRICULAR TACHYCARDIAS AND ATRIAL FIBRILLATION ................................................................. p.70
J. Brugada

5.2 VENTRICULAR TACHYCARDIAS ..................................................................................................................... p.74
M. Santini, C. Lavalle, S. Lanzara

5.3 BRADYARRHYTHMIAS ........................................................................................................................................ p.77
B. Gorenek
TACHYARRHYTHMIAS: Diagnostic criteria

Tachycardia > 100 beats/minute

Regular

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 chapter 5.3 page 77)

QRS complex >120 msec

Ventricular Tachycardia or SVT with aberrant conduction

(see chapter 5.2 page 76)

Irregular

YES

QRS morphology similar to QRS morphology in sinus rhythm?

QRS complex <120 msec

AF conducting over AVN

AF + BBB or AF + WPW

QRS complex >120 msec

Variable QRS morphology

AF + WPW

Irregular Ventricular Tachycardia

NO

QRS complex <120 msec

AF

QRS complex >120 msec

Irregular Ventricular Tachycardia

NO
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

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

Tachycardia terminates

Atrial flutter or atrial tachycardia

Ventricular Tachycardia

Ventricular Tachycardia

Ventricular Tachycardia

Consider SVT using the AV node (AVNRT, AVNT)

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

Typical morphology in V1 & V6 (see chapter 5.3 page 77)
TACHYARRHYTHMIAS: Therapeutic algorithms (1)

Regular Supraventricular Tachycardias
with or without bundle branch block

Hemodynamically non-stable
- Immediate electrical cardioversion

No termination

Narrow QRS complex tachycardia
- Reconsider diagnosis: sinus tachycardia, atrial tachycardia
- If no evidence: Intravenous verapamil

Hemodynamically stable
- Vagal maneuvers and/or i.v. Adenosine

Termination

Wide QRS complex tachycardia
- Reconsider the diagnosis of Ventricular Tachycardia even if hemodynamically stable
- Do not administer verapamil

Irregular and narrow QRS complex Tachycardia

Less than 48 hours since initiation AND hemodynamically stable

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 non-stable
- Immediate electrical Cardioversion

If no cardioversion is considered:
- rate control using betablockers 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
TACHYARRHYTHMIAS: Therapeutic algorithms (2)

Irregular and wide QRS complex Tachycardia

Hemodynamically non-stable

**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 stable

**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 TACHYSCARDIAS:
Differential diagnosis of wide QRS tachycardias

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**
  - **No**

2nd Step
- **Concordant pattern in precordial leads**
  - No RS morphology in any of the precordial leads
  - **Yes**
  - **No**

3rd Step
- **An interval >100 ms from the beginning of the QRS complex to the nadir of S in a precordial lead**
  - **Yes**
  - **No**

- **Morphology in precordial leads**
- **Morphology in aVR lead**

VT
VENTRICULAR TACHYCARDIAS: DIFERENTIAL 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
2nd Step
3rd 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
LBBB morphology
Morphology in aVR lead
Initial R wave or q >40 msec

V1: qR, R, R'
V6: rS,QS

V1: rsR', RSR'
V6: qRs

V1: rS; R >30 ms, S nadir >60 ms, notching of the S wave

V6: qR, QS

Aberrant conduction
VT
VT

Vi/Vt ≤ 1
Management of wide QRS TACHYCARDIAS

Hemodynamic Tolerance

Non-stable
- Pulseless
- With pulse

Stable
- Irregular rhythm
- Regular rhythm

ACLS Resuscitation algorithm
- Immediate high-energy defibrillation (200 J 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 3-5 min)
- 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

ACLs Resuscitation algorithm

• Sedation or analgesia
• Synchronised cardioversion 100 to 200 J (monophasic) or 50-100 J (biphasic)

Differential Diagnosis

No
- AF with aberrant ventricular conduction
  • β-blockers
  • i.v.
  • Verapamil or diltiazem
- Pre excited AF
  • Class 1 AADs
- Polymorphic VT
  • Amiodarone

Yes
- Vagal maneuver and/or i.v. adenosine (push)
- Interruption or slow down HR
- SVT

AF with aberrant ventricular conduction

Pre excited AF

Polymorphic VT

Differential Diagnosis
(see chapter 5.1 page 73)

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

Synchronised cardioversion
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: Definitions and diagnosis
BRADYARRHYTHMIAS: Treatment (I)

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

For more information on individual drug doses and indications, See chapter 8: Use of drugs in Acute Cardiovascular Care.

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.
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 ................................................................. p.82
A. Evangelista

6.2 ACUTE PULMONARY EMBOLISM ....................................................... p.92
A. Torbicki
ACUTE AORTIC SYNDROMES: Concept and classification (1)  
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)
**DeBakey’s Classification**
- Type I and type II dissections both originate in the ascending aorta
  - In type I, the dissection extends distally to the descending 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: < 14 days
- Subacute: 15-90 days
- Chronic: > 90 days

Adapted with permission from Nienaber CA, Eagle KA, Circulation 2003;108(6):772-778. All rights reserved.
ACUTE AORTIC SYNDROME: Clinical suspicion and differential diagnosis

SYMPTOMS AND SIGNS SUGGESTIVE OF AAS

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

DIFFERENTIAL DIAGNOSIS

- Acute coronary syndrome (with/without ST-segment elevation)
- Aortic regurgitation without dissection
- Aortic aneurysms without dissection
- Musculoskeletal pain
- Pericarditis
- Pleuritis
- Mediastinal tumours
- Pulmonary embolism
- Cholecystitis
- Atherosclerosis or cholesterol embolism
General approach to the patient with suspected ACUTE AORTIC SYNDROME

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

<table>
<thead>
<tr>
<th>High-risk conditions</th>
<th>High-risk pain features</th>
<th>High-risk exam features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marfan’s syndrome</td>
<td>Chest, back or abdominal pain described as:</td>
<td>Perfusion deficit:</td>
</tr>
<tr>
<td>Connective tissue disease</td>
<td>Abrupt at onset, severe in intensity, and ripping/sharp or stabbing quality</td>
<td>- Pulse deficit</td>
</tr>
<tr>
<td>Family history of aortic disease</td>
<td></td>
<td>- SBP differential</td>
</tr>
<tr>
<td>Aortic valve disease</td>
<td></td>
<td>- Focal neurological deficit</td>
</tr>
<tr>
<td>Thoracic aortic aneurysm</td>
<td></td>
<td>Aortic regurgitation murmur</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hypotension or shock</td>
</tr>
</tbody>
</table>

## Laboratory tests required for patients with ACUTE AORTIC dissection

<table>
<thead>
<tr>
<th>Laboratory tests</th>
<th>To detect signs of:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Red blood cell count</td>
<td>Blood loss, bleeding, anaemia</td>
</tr>
<tr>
<td>White blood cell count</td>
<td>Infection, inflammation (SIRS*)</td>
</tr>
<tr>
<td>C-reactive protein</td>
<td>Inflammatory response</td>
</tr>
<tr>
<td>ProCalcitonin</td>
<td>Differential diagnosis between SIRS* and sepsis</td>
</tr>
<tr>
<td>Creatine kinase</td>
<td>Reperfusion injury, rhabdomyolysis</td>
</tr>
<tr>
<td>Troponin I/LorT</td>
<td>Myocardial ischaemia, myocardial infarction</td>
</tr>
<tr>
<td>D-dimer</td>
<td>Aortic dissection, pulmonary embolism, thrombosis</td>
</tr>
<tr>
<td>Creatinine</td>
<td>Renal failure (existing or developing)</td>
</tr>
<tr>
<td>Aspartate transaminase/alanine aminotransferase</td>
<td>Liver ischaemia, liver disease</td>
</tr>
<tr>
<td>Lactate</td>
<td>Bowel ischaemia, metabolic disorder</td>
</tr>
<tr>
<td>Glucose</td>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>Blood gases</td>
<td>Metabolic disorder, oxygenation</td>
</tr>
</tbody>
</table>

*SIRS = systemic inflammatory response syndrome.

Reference: Eur Heart J 2014;eurheartj.ehu281.
ACUTE CHEST PAIN

Medical history + clinical examination + ECG → STEMI<sup>a</sup> : see ESC guidelines<sup>169</sup>

**UNSTABLE**

- TTE + TOE/CT<sup>o</sup>
  - AAS confirmed
  - AAS excluded

**STABLE**

- Low probability (score 0-1)
- High probability (score 2-3) or typical chest pain

**HAEMODYNAMIC STATE**

- D-dimers<sup>d,e</sup> + TTE + Chest X-ray
  - No argument for AD
  - Signs of AD
  - Widened mediastinum

- CT (MRI or TOE)<sup>b</sup>
  - AAS confirmed
  - Consider alternate diagnosis

- TTE
  - Inconclusive

- Definite Type A - AD<sup>c</sup>
  - Refer on emergency to surgical team and pre-operative TOE

- CT (or TOE)
  - AAS confirmed
  - Consider alternate diagnosis repeat CT if necessary

<sup>a</sup> STEMI can be associated with AAS in rare cases.

<sup>b</sup> Pending local availability, patient characteristics, and physician experience.

<sup>c</sup> Proof of type-A AD by the presence of flap, aortic regurgitation, and/or pericardial effusion.

<sup>d</sup> Preferably point-of-care, otherwise classical.

<sup>e</sup> Also troponin to detect non-ST-segment elevation myocardial infarction.

Flowchart for decision-making based on pre-test sensitivity of acute aortic syndrome.
Reference: Eur Heart J 2014;eurheartj.ehu281
### Details required from imaging in ACUTE AORTIC dissection

| Aortic dissection | Visualization of intimal flap  
|                  | Extent of the disease according to the aortic anatomic segmentation  
|                  | Identification of the false and true lumens (if present)  
|                  | Localization of entry and re-entry tears (if present)  
|                  | Identification of antegrade and/or retrograde aortic dissection  
|                  | Identification grading, and mechanism of aortic valve regurgitation  
|                  | Involvement of side branches  
|                  | Detection of malperfusion (low flow or no flow)  
|                  | Detection of organ ischaemia (brain, myocardium, bowels, kidneys, etc.)  
|                  | Detection of pericardial effusion and its severity  
|                  | Detection and extent of pleural effusion  
|                  | Detection of peri-aortic bleeding  
|                  | Signs of mediastinal bleeding  

| Intramural haematoma | Localization and extent of aortic wall thickening  
|                     | Co-existence of atheromatous disease (calcium shift)  
|                     | Presence of small intimal tears  

| Penetrating aortic ulcer | Localization of the lesion (length and depth)  
|                         | Co-existence of intramural haematoma  
|                         | Involvement of the peri-aortic tissue and bleeding  
|                         | Thickness of the residual wall  

| In all cases | Co-existence of other aortic lesions: aneurysms, plaques, signs of inflammatory disease, etc.  

ACUTE AORTIC SYNDROMES MANAGEMENT: General approach

ACUTE AORTIC DISSECTION

Type A
(Ascending aorta involvement)

- Open Surgery with/without Endovascular Therapy

Type B
(No ascending aorta involvement)

Uncomplicated
- Medical treatment

Complicated (malperfusion, rupture)
- Endovascular Therapy or Open Surgery (TEVAR*)

*TEVAR Thoracic Endovascular Aortic Repair.
ACUTE AORTIC SYNDROMES: Initial management

1. Detailed medical history and complete physical examination (when possible)
2. Standard 12-lead ECG: Rule-out ACS, documentation of myocardial ischemia
3. Intravenous line, blood sample (CK, Tn, myoglobin, white blood count, D-dimer, hematocrit, LDH)
4. Monitoring: HR and BP
5. Pain relief (morphine sulphate) (see chapter 3)
6. Noninvasive imaging (see previous page)
7. Transfer to ICU

For more information on individual drug doses and indications, See chapter 8: Use of drugs in Acute Cardiovascular Care.
**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**
- Haemodynamic 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

### 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**
- if TEVAR contraindicated

**MEDICAL MANAGEMENT and OPEN SURGERY REPAIR**
- if TEVAR contraindicated
Risk-adjusted management strategies in ACUTE PULMONARY EMBOLISM

Clinical suspicion

Yes

Shock / hypotension?

Diagnostic algorithm as for suspected not high-risk PE

PE confirmed

No

Diagnostic algorithm as for suspected not high-risk PE

Shock / hypotension?

PE confirmed

Intermediate risk

Consider further risk stratification

RV function (echo or CT)\textsuperscript{a}

Laboratory testing\textsuperscript{b}

Both positive

One positive or both negative

PESI Class III-IV or sPESI ≥ 1

PESI Class I-II or sPESI = 0
a If echocardiography has already been performed during diagnostic work-up for PE and detected RV dysfunction, or if the CT already performed for diagnostic work-up has shown RV enlargement (RV/LV (left ventricular) ratio ≥0.9, a cardiac troponin test should be performed except for cases in which primary reperfusion is not a therapeutic option (e.g. due to severe comorbidity or limited life expectancy of the patient).

b Markers of myocardial injury (e.g. elevated cardiac troponin I or T concentrations in plasma), or of heart failure as a result of (right) ventricular dysfunction (elevated natriuretic peptide concentrations in plasma). If a laboratory test for a cardiac biomarker has already been performed during initial diagnostic work-up (e.g. in the chest pain unit) and was positive, then an echocardiogram should be considered to assess RV function, or RV size should be (re)assessed on CT.

c Patients in the PESI Class I-II, or with sPESI of 0, and elevated cardiac biomarkers or signs of RV dysfunction on imaging tests, are also to be classified into the intermediate-low risk category. This might apply to situations in which imaging or biomarker results become available before calculation of the clinical severity index. These patients are probably no candidates for home treatment.

d Thrombolysis, if (and as soon as) clinical signs of haemodynamic decompensation appear; surgical pulmonary embolectomy or percutaneous catheter-directed treatment may be considered as alternative options to systemic thrombolysis, particularly if the bleeding risk is high.

e Monitoring should be considered for patients with confirmed PE and a positive troponin test, even if there is no evidence of RV dysfunction on echocardiography or CT.

f The simplified version of the PESI has not been validated in prospective home treatment trials; inclusion criteria other than the PESI were used in two single-armed (non-randomized) management studies.

ACUTE PULMONARY EMBOLISM: Diagnosis

**CARDIOVASCULAR Symptoms/Signs**
including but not limited to:
- Chest pain (angina)
- Syncope
- Tachycardia
- ECG changes
- NT-proBNP ↑
- Troponin ↑

**RESPIRATORY Symptoms/Signs**
including but not limited to:
- Chest pain (pleural)
- Pleural effusion
- Tachypnea
- Hemoptysis
- Hypoxemia
- Atelectasis

**Dyspnea**

- Suspect acute PE

**Management algorithm for UNSTABLE patients**

- **YES**
  - **NO**
  - **SBP <90 mmHg?**
  - **or**
  - **SBP fall by >40 mmHg?**
    - persisting > 15 min, otherwise unexplained

**Management algorithm for initially STABLE patients**

Management algorithm for unstable patients with suspected ACUTE PULMONARY EMBOLISM

CT angiography immediately available and patient stabilised

Primary PA reperfusion not justified

Echocardiography (bedside)

RV pressure overload

Yes

No

Search for other causes

CT angiography

No further diagnostic tests feasible

Primary PA reperfusion

No positive

Yes

CT* Angio

No

Yes

Right heart, pulmonary artery or venous thrombi?

CUS

TEE

Yes

negative

* Consider also pulmonary angiography if unstable patient in hemodynamic lab.

### ACUTE PE: 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><strong>Contraindications for thrombolysis</strong></td>
<td>No</td>
</tr>
<tr>
<td><strong>Primary PA reperfusion strategy</strong></td>
<td>Thrombolysis</td>
</tr>
<tr>
<td><strong>Supportive treatment</strong></td>
<td>i.v. UFH, STABILISE SYSTEMIC BLOOD PRESSURE, CORRECT HYPOXEMIA</td>
</tr>
</tbody>
</table>
Management algorithm for initially stable patients with suspected ACUTE PULMONARY EMBOLISM

Asses clinical (pre-test) probability

Low or intermediate
“PE unlikely”

D-dimer

negative

CT angiography

negative

Anticoagulation not justified

positive

Anticoagulation required

High or
“PE likely”

CT angiography

positive

Confirm by CUS, V/Q scan or angiography

Anticoagulation not justified

CUS

positive

Anticoagulation required

CUS positive

### Suggested management strategy for initially stable patients with (non-high risk) confirmed 6.2

#### Markers for myocardial injury
- Positive
- Positive
- Negative

#### Markers for RV overload
- Positive
- Positive
- Negative

#### Clinical risk assessment score (PESI)
- Positive (class III-V)
- Positive (class III-V)
- Negative (class I-II)

#### Suggested initial anticoagulation
- UFH i.v /LMWH s.c.
- LMWH/Fonda/ apixaban/ rivaroxaban
- apixaban/rivaroxaban

### STRATEGY

<table>
<thead>
<tr>
<th></th>
<th>Monitoring (ICU)* rescue thrombolysis</th>
<th>Hospitalisation** (telemonitoring)</th>
<th>Early discharge***</th>
</tr>
</thead>
<tbody>
<tr>
<td>* When all markers are positive.</td>
<td>** When at least one marker is positive.</td>
<td>*** When all markers are negative.</td>
<td></td>
</tr>
</tbody>
</table>

### Pharmacological treatment:

For more information on individual drug doses and indications, see chapter 8: Use of drugs in Acute Cardiovascular Care.
**PULMONARY EMBOLISM: Pharmacological treatment**

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

For more information on individual drug doses and indications, See chapter 8: Use of drugs in Acute Cardiovascular Care.

<table>
<thead>
<tr>
<th>Unstable/ Stable</th>
<th>Drug Description</th>
<th>Dose/ Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unstable</td>
<td>Alteplase (rtPA) (intravenous)</td>
<td>100 mg/2 h or 0.6 mg/kg/15 min (max 50 mg)</td>
</tr>
<tr>
<td></td>
<td>Urokinase (intravenous)</td>
<td>3 million IU over 2 h</td>
</tr>
<tr>
<td></td>
<td>Streptokinase (intravenous)</td>
<td>1.5 million IU over 2 h</td>
</tr>
<tr>
<td></td>
<td>Unfractionated heparin (intravenous)</td>
<td>80 IU/kg bolus + 18 IU/kg/h</td>
</tr>
<tr>
<td>Stable</td>
<td>Enoxaparine (subcutaneous)</td>
<td>1.0 mg/kg BID or 1.5 mg/kg QD</td>
</tr>
<tr>
<td></td>
<td>Tinzaparin (subcutaneous)</td>
<td>175 U/kg QD</td>
</tr>
<tr>
<td></td>
<td>Fondaparinux (subcutaneous)</td>
<td>7.5 mg (50-100 Kg of body weight)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5 mg for patients &lt;50 kg,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>10 mg for patients &gt;100 kg</td>
</tr>
<tr>
<td></td>
<td>Rivaroxaban (oral)</td>
<td>15 mg BID</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(for 3 weeks, then 20 mg QD)</td>
</tr>
<tr>
<td></td>
<td>Apixaban (oral)</td>
<td>10mg bid (for 7 days, than 5mg bid)</td>
</tr>
</tbody>
</table>
CHAPTER 7: ACUTE MYOCARDIAL / PERICARDIAL SYNDROMES

7.1 ACUTE MYOCARDITIS ........................................................................................................................................... p.102
   A. Keren, A. Caforio

7.2 ACUTE PERICARDITIS AND CARDIAC TAMPONADE ............................................................................... p.107
   C. Vrints, S. Price
**MYOCARDITIS (WHO /ISFC):** Inflammatory disease of the myocardium diagnosed by established histological, immunological and immunohistochemical criteria.

**CAUSES OF MYOCARDITIS**

- **INFECTIONOUS**
  - 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>
<th>Diagnostic criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Acute chest pain (pericarditic or pseudo-ischemic)</td>
<td>I. <strong>ECG/Holter/stress test features</strong>: Newly abnormal ECG and/or Holter and/or stress testing, any of the following:</td>
</tr>
<tr>
<td>• New-onset (days up to 3 months) or worsening dyspnea or fatigue, with or without left/right heart failure signs</td>
<td>• I to III degree atrioventricular block, or bundle branch block, ST/T wave changes (ST elevation or non ST elevation, T wave inversion),</td>
</tr>
<tr>
<td>• Palpitation, unexplained arrhythmia symptoms, syncope, aborted sudden cardiac death</td>
<td>• Sinus arrest, ventricular tachycardia or fibrillation and asystole, atrial fibrillation, frequent premature beats, supraventricular tachycardia</td>
</tr>
<tr>
<td>• Unexplained cardiogenic shock and/or pulmonary oedema</td>
<td>• Reduced R wave height, intraventricular conduction delay (widened QRS complex), abnormal Q waves, low voltage</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ancillary findings which support the clinical suspicion of myocarditis</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Fever ≥38.0°C within the preceding 30 days</td>
<td>II. <strong>Myocardiocytolysis markers</strong>: Elevated TnT/TnI</td>
</tr>
<tr>
<td>• A respiratory or gastrointestinal infection</td>
<td></td>
</tr>
<tr>
<td>• Previous clinically suspected or biopsy proven myocarditis</td>
<td>III. <strong>Functional/structural abnormalities on echocardiography</strong>:</td>
</tr>
<tr>
<td>• Peri-partum period</td>
<td>• New, otherwise unexplained LV and/or RV structure and function abnormality (including incidental finding in apparently asymptomatic subjects): regional wall motion or global systolic or diastolic function abnormality, with or without ventricular dilatation, with or without increased wall thickness, with or without pericardial effusion, with or without endocavitary thrombi</td>
</tr>
<tr>
<td>• Personal and/or family history of allergic asthma</td>
<td></td>
</tr>
<tr>
<td>• Other types of allergy</td>
<td>IV. <strong>Tissue characterisation by CMR</strong>:</td>
</tr>
<tr>
<td>• Extra-cardiac autoimmune disease</td>
<td>• Edema and/or LGE of classical myocarditic pattern</td>
</tr>
<tr>
<td>• Toxic agents</td>
<td></td>
</tr>
<tr>
<td>• Family history of dilated cardiomyopathy, myocarditis</td>
<td></td>
</tr>
</tbody>
</table>

**ACUTE MYOCARDITIS: Diagnostic criteria (2)**

Acute myocarditis should be clinically suspected in the presence of:

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

OR

when the patient is asymptomatic, two 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***

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

*See chapter 7.1 page 101.
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

Hemodynamically stable, decreased LV function, cardiogenic shock

Immunosuppression if infection-negative EMB

Hemodynamically stable
Preserved LV function
No eosinophilia
No significant rhythm or conduction disturbances
Not associated with systemic immune disease*

General supportive therapy

Lymphocytic

Pharmacological and, if needed, mechanical circulatory support (ECMO, LVAD/Bi-VAD, bridge to heart transplant or to recovery)

Giant cell, eosinophilic, sarcoidosis (acute decompensation)

General supportive therapy
Immunosuppression if unresponsive and virus negative EMB

No coronary artery disease

*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**

- **Equivocal or no**
  - Consider cardiac MRI
  - Consider alternative diagnoses

- Delayed enhancement pericardium
**Acute pericarditis**

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

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

**Yes**
- Hospital admission

**No**
- Outpatient treatment
  - 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!

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

**Tamponade?**
- Pericardiocentesis
CARDIAC TAMPONADE: 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

**Tamponade ?**

**Tamponade**

Not performed in routine

p.109
CHAPTER 8: DRUGS USED IN ACUTE CARDIOVASCULAR CARE

Ana de Lorenzo
# Oral antiplatelets

<table>
<thead>
<tr>
<th>Drug</th>
<th>Indications</th>
<th>Dose</th>
<th>Dose adjustments</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>Primary (not universally approved) and secondary cardiovascular disease prevention</td>
<td>LD (if ACS): 150-300 mg oral MD: 75-100 mg oral QD</td>
<td>-</td>
<td>Major contraindications: GI bleeding-active peptic ulcer</td>
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<tr>
<td>Ticagrelor</td>
<td>ACS (all patients at moderate-to-high risk of ischaemic events, e.g. elevated cardiac troponins)</td>
<td>LD: 180 mg oral MD: 90 mg oral BID</td>
<td>-</td>
<td>Major contraindications: previous intracerebral hemorrhage</td>
</tr>
<tr>
<td></td>
<td>Secondary prevention I-3 years post-MI</td>
<td>MD: 60 mg oral BID</td>
<td>-</td>
<td>Major contraindications: previous intracerebral hemorrhage</td>
</tr>
<tr>
<td>Prasugrel</td>
<td>ACS with planned PCI</td>
<td>LD: 60 mg oral MD: 10 mg oral QD</td>
<td>MD: 5 mg QD weight &lt; 60 kg</td>
<td>Contraindication: previous stroke/TIA Prasugrel is generally not recommended in elderly, and if positive benefit/risk 5 mg is recommended</td>
</tr>
<tr>
<td></td>
<td>ACS + PCI or medical management (patients who cannot receive ticagrelor or prasugrel) and in ACS patients at high bleeding risk (e.g. patients who require oral anticoagulation)</td>
<td>LD: 300-600 mg oral MD: 75 mg oral QD</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

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## Oral antiplatelets (Cont.)

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</thead>
<tbody>
<tr>
<td><strong>Clopidogrel</strong></td>
<td>STEMI + fibrinolysis &lt; 75 years</td>
<td>LD: 300 mg oral</td>
<td>-</td>
<td>Prasugrel and ticagrelor have not been studied as adjuncts to fibrinolysis and oral anticoagulants</td>
</tr>
<tr>
<td></td>
<td>STEMI + fibrinolysis ≥ 75 years</td>
<td>MD: 75 mg oral QD</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Secondary prevention &gt;12 months post coronary stenting</td>
<td>MD: 75 mg oral QD</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td><strong>Vorapaxar</strong></td>
<td>Co-administered with aspirin and, where appropriate, clopidogrel, in patients with a history of MI or peripheral artery disease</td>
<td>2.08 mg oral QD</td>
<td>-</td>
<td>Initiated at least 2 weeks after a MI and preferably within the first 12 months. Major contraindications: active pathologic bleeding or increased risk of bleeding, history of stroke / TIA or intracranial bleeding, severe hepatic dysfunction</td>
</tr>
</tbody>
</table>

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**Intravenous Antiplatelets**

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</thead>
<tbody>
<tr>
<td>Abciximab</td>
<td>Adjunct to PCI for bailout situations or thrombotic complications</td>
<td>LD: 0.25 mg/Kg i.v. MD: 0.125 μg/Kg/min i.v. (max: 10 μg/min) for 12h</td>
<td>-</td>
<td>Contraindications: Active internal bleeding - History of CVA within 2 years - Bleeding diathesis - Preexisting thrombocytopenia - Recent (within 2 months) intracranial or intraspinal surgery or trauma - Recent (within 2 months) major surgery - Intracranial neoplasm, arteriovenous malformation, or aneurysm - Severe uncontrolled hypertension - Presumed or documented history of vasculitis - Severe hepatic failure or severe renal failure requiring haemodialysis - Hypertensive retinopathy</td>
</tr>
<tr>
<td>Eptifibate</td>
<td>ACS treated medically or with PCI</td>
<td>LD: 180 μg/Kg i.v. (at a 10 min interval) If STEMI and PCI: add a second 180 mcg/kg i.v. bolus at 10 min MD: 2 μg/Kg/min i.v. infusion</td>
<td>Reduce infusion dose to 1 μg/kg/min if CrCl 30-50ml/min</td>
<td>Contraindications: Bleeding diathesis or bleeding within the previous 30 days - Severe uncontrolled hypertension - Major surgery within the preceding 6 weeks - Stroke within 30 days or any history of hemorrhagic stroke - Coadministration of another parenteral GP IIb/IIIa inhibitor - Dependency on renal dialysis - Known hypersensitivity to any component of the product</td>
</tr>
</tbody>
</table>
## Intravenous Antiplatelets (Cont.)

<table>
<thead>
<tr>
<th>Drug</th>
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<th>Dose adjustments</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tirofiban</td>
<td>ACS treated medically or with PCI</td>
<td>LD: 25 μg/Kg i.v. over 5 min</td>
<td>CrCl &lt; 30ml/min: decrease 50% bolus and infusion dose</td>
<td>Contraindications: Severe hypersensitivity reaction to tirofiban A history of thrombocytopenia following prior exposure Active internal bleeding or a history of bleeding diathesis, major surgical procedure or severe physical trauma within the previous month</td>
</tr>
<tr>
<td></td>
<td></td>
<td>MD: 0.15 μg/Kg/min i.v. infusion to 18 hour</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>CrCl &lt; 30ml/min: decrease 50% bolus and infusion dose</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cangrelor</td>
<td>All patients undergoing PCI (elective + ACS) immediate onset + rapid offset (platelet recovery in 60 min)</td>
<td>IV Bolus of 30 μg/Kg + IV infusion of 4 μg/kg/min For at least 2 hours from start of PCI</td>
<td>-</td>
<td>Major contraindications: significant active bleeding or stroke Transition to oral P2Y12 inhibitors variable according to type of agent</td>
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</tbody>
</table>
**Oral Anticoagulants**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Indications</th>
<th>Dose</th>
<th>Dose adjustments</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Warfarin Acenocoumarol</td>
<td><strong>Treatment and prophylaxis of thrombosis</strong></td>
<td>INR goal of 2-3</td>
<td>Assessing individual risks for thromboembolism and bleeding</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>(INR: 2.5-3.5 for mechanical mitral valve prostheses or double valve replacement)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dabigatran</td>
<td><strong>Prevention of stroke and systemic embolism in NVAF</strong></td>
<td>150 mg oral BID</td>
<td>110 mg BID (if age ≥ 80, increased bleeding risk or concomitant use of verapamil)</td>
<td>Contraindicated if CrCl &lt; 30ml/min or severe hepatic impairment</td>
</tr>
<tr>
<td></td>
<td><strong>Treatment of DVT and PE in patients who have been treated with a parenteral anticoagulant for 5-10 days and prevention of recurrent DVT and PE in patients who have been previously treated</strong></td>
<td>150 mg oral BID</td>
<td></td>
<td>Active pathological bleeding</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Idarucizumab: specific antidote (not yet available)</td>
</tr>
</tbody>
</table>

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# Oral Anticoagulants (Cont.)

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<th>Dose adjustments</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rivaroxaban</td>
<td>Prevention of stroke and systemic embolism in NVAF</td>
<td>20 mg oral QD</td>
<td>CrCl &lt; 50ml/min: 15 mg QD</td>
<td>Contraindicated if CrCl &lt; 15ml/min or hepatic disease associated with coagulopathy and clinically relevant bleeding risk</td>
</tr>
<tr>
<td></td>
<td>Treatment of DVT and PE and prevention of recurrent DVT and PE</td>
<td>15 mg oral BID for the first 3 weeks followed by 20 mg QD</td>
<td>Reduce the maintenance dose to 15 mg QD if bleeding risk outweighs the risk for recurrent DVT and PE (not formally approved)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Prevention of atherothrombotic events after an ACS</td>
<td>2.5 mg oral BID</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apixaban</td>
<td>Prevention of stroke and systemic embolism in NVAF</td>
<td>5 mg oral BID</td>
<td>2.5 mg oral BID 1) when at least 2 of the following characteristics: age ≥ 80, Cr &gt; 1.5 mg/dl or weight &lt; 60Kg 2) when CrCl 15-29 mL/min</td>
<td>Contraindicated if CrCl &lt; 15ml/min or severe hepatic impairment</td>
</tr>
<tr>
<td></td>
<td>Treatment of DVT and PE</td>
<td>10 mg oral BID for the first 7 days followed by 5 mg oral BID</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Prevention of recurrent DVT and PE</td>
<td>2.5 mg oral BID</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
**Intravenous/Subcutaneous Anticoagulants**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Indications</th>
<th>Dose</th>
<th>Dose adjustments</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>UFH</td>
<td>NSTEMI: Primary PCI: 70-100 IU/Kg i.v. when no GP-IIb/IIIa inhibitor is planned. 50-60 IU/Kg i.v. bolus with GP-IIb/IIIa inhibitors - Fibrinolysis/No reperfusion: 60 IU/kg i.v. bolus (max: 4000 IU) followed by an i.v. infusion of 12 IU/kg (max: 1000 IU/h) for 24-48h</td>
<td>Target aPTT: 50-70s or 1.5 to 2.0 times that of control to be monitored at 3, 6, 12 and 24h</td>
<td>Monitoring for heparin-induced thrombocytopenia (HIT) Dose-independent reaction</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Treatement of DVT and PE: 80 IU/Kg i.v. bolus followed by 18 IU/Kg/h</td>
<td>According to aPTT, thromboembolic and bleeding risk</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fondaparinux</td>
<td>NSTEMI: 2.5 mg QD s.c.</td>
<td>-</td>
<td>Severe hepatic impairment: caution advised</td>
<td></td>
</tr>
<tr>
<td></td>
<td>STEMI: Fibrinolysis/No reperfusion: 2.5 mg i.v. bolus followed by 2.5 mg QD s.c. up to 8 days or hospital discharge</td>
<td>-</td>
<td>Contraindicated if CrCl &lt; 20ml/min</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Treatment of DVT and PE: 5 mg QD s.c. (&lt; 50 kg); 7.5 mg QD s.c. (50-100 kg); 10 mg QD s.c. (&gt; 100 kg)</td>
<td>If &gt; 100Kg and CrCl 30-50ml/min: 10 mg followed by 7.5 mg/24h s.c.</td>
<td>Contraindicated for DVT/PE treatment</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Prevention of VTE: 2.5 mg QD s.c.</td>
<td>CrCl 20-50ml/min: 1.5 mg QD s.c.</td>
<td>if CrCl &lt; 30ml/min</td>
<td></td>
</tr>
</tbody>
</table>

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</tr>
</thead>
<tbody>
<tr>
<td>Bivalirudin</td>
<td>PCI for NSTE-ACS</td>
<td>0.75 mg/kg i.v. bolus followed immediately by 1.75 mg/kg/h infusion which may be continued for up to 4h post PCI as clinically warranted and further continued at a reduced infusion dose of 0.25 mg/kg/h for 4-12h as clinically necessary</td>
<td>Patients undergoing PCI with CrCl 30-50ml/min should receive a lower infusion rate of 1.4 mg/kg/h. No change for the bolus dose.</td>
<td>Contraindicated if CrCl &lt; 30ml/min</td>
</tr>
<tr>
<td></td>
<td>PCI for STEMI</td>
<td>0.75 mg/kg i.v. bolus followed immediately by 1.75 mg/kg/h infusion which should be continued for up to 4h after the procedure After cessation of the 1.75 mg/kg/h infusion, a reduced infusion dose of 0.25 mg/kg/h may be continued for 4-12h</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>PCI for elective cases</td>
<td>0.75 mg/kg i.v. bolus followed immediately by 1.75 mg/kg/h infusion which may be continued for up to 4h post PCI as clinically warranted</td>
<td></td>
<td></td>
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</tr>
</thead>
<tbody>
<tr>
<td>Enoxaparin</td>
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</tr>
<tr>
<td></td>
<td><strong>NSTE-ACS</strong></td>
<td>30 mg i.v. + 1 mg/kg s.c. BID</td>
<td>If &gt; 75 years: no LD and MD 0.75 mg/Kg BID s.c. CrCl &lt; 30ml/min: no LD and MD 1 mg/Kg QD s.c. If &gt; 75 years and CrCl &lt; 30ml/min: no LD and 0.75 mg/Kg QD s.c.</td>
<td>Monitoring for HIT - Anti Xa monitoring during treatment with LMWH might be helpful in pregnancy, extreme body weights and renal impairment.</td>
</tr>
<tr>
<td></td>
<td><strong>STEMI</strong></td>
<td>Primary PCI: 0.5 mg/Kg i.v. bolus Fibrinolysis/No reperfusion:</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>a) Age &lt; 75y: 30 mg i.v. bolus followed by 1 mg/Kg BID s.c. until hospital discharge for a max of 8 days - The first two doses should not exceed 100 mg</td>
<td>In patients with CrCl &lt; 30 ml/min: regardless of age, the s.c. doses are given once daily.</td>
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<tr>
<td></td>
<td>b) Age &gt; 75y: no bolus; 0.75 mg/Kg BID s.c. - The first two doses should not exceed 75 mg</td>
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<td></td>
</tr>
<tr>
<td></td>
<td><strong>Treatment of DVT and PE</strong></td>
<td>1 mg/Kg s.c. BID or 1.5 mg/Kg s.c. QD</td>
<td>CrCl &lt; 30ml/min: 1 mg/Kg/24h s.c.</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>Prevention of VTE</strong></td>
<td>40 mg s.c. QD</td>
<td>CrCl &lt; 30ml/min: 20 mg s.c. QD</td>
<td></td>
</tr>
</tbody>
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## Intravenous/Subcutaneous Anticoagulants (Cont.)

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<tbody>
<tr>
<td><strong>Tinzaparin</strong></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Prevention of VTE</td>
<td>3500 IU s.c. QD (moderate risk) 4500 IU s.c. QD (high risk)</td>
<td>-</td>
<td>Monitoring for HIT - Anti Xa monitoring during treatment with LMWH might be helpful in pregnancy, extreme body weights and renal impairment - Dalteparin: In cancer patients, dose of 200 IU/kg (max:18000 IU)/24h for 1 month, followed by 150 IU/kg/24h for 5 months - After this period, vitamin K antag or a LMWH should be continued indefinitely or until the cancer is considered cured.</td>
<td></td>
</tr>
<tr>
<td>Treatment of DVT and PE</td>
<td>175 IU/Kg s.c. QD</td>
<td>-</td>
<td></td>
<td></td>
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<tr>
<td><strong>Dalteparin</strong></td>
<td></td>
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</tr>
<tr>
<td>Prevention of VTE</td>
<td>2500 IU s.c. QD (moderate risk) 5000 IU s.c. QD (high risk)</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment of DVT and PE</td>
<td>200 IU/Kg QD or 100 IU/Kg BID s.c.</td>
<td>Anti Xa monitoring if renal impairment</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Argatroban</strong></td>
<td>Anticoagulant in patients with HIT</td>
<td>Initial i.v. infusion dose: 2 μg/kg/min (not to exceed 10 μg/kg/min) Patients undergoing PCI: 350 μg/kg i.v. followed by 25 μg/kg/min i.v.</td>
<td>Renal and hepatic impairment: caution advised</td>
<td>Monitored using aPTT goal: 1.5 to 3.0 times the initial baseline value PCI:ACT goal: 300-450s</td>
</tr>
</tbody>
</table>

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### Fibrinolytics

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<th>Dose adjustments</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Streptokinase (SK)</td>
<td>STEMI</td>
<td>1.5 million units over 30-60min i.v.</td>
<td>-</td>
<td>Absolute contraindications to fibrinolytics:</td>
</tr>
<tr>
<td></td>
<td>Treatment of PE</td>
<td>250000 IU as a LD over 30min,</td>
<td>-</td>
<td>Previous intracranial haemorrhage or stroke of unknown origin at any time</td>
</tr>
<tr>
<td></td>
<td></td>
<td>followed by 100000 IU/h over 12-24h</td>
<td></td>
<td>Ischaemic stroke in the preceding 6 months</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Central nervous system damage or neoplasms or atrioventricular malformation</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Recent major trauma/surgery/head injury (within the preceding 3 weeks)</td>
</tr>
<tr>
<td>Alteplase (tPA)</td>
<td>STEMI</td>
<td>15 mg i.v. bolus: 0.75 mg/kg over 30min</td>
<td>-</td>
<td>Gastrointestinal bleeding within the past month</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(up to 50 mg) then 0.5 mg/kg over 60min</td>
<td></td>
<td>Known bleeding disorder (excluding menses)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>i.v. (up to 35 mg)</td>
<td></td>
<td>Aortic dissection</td>
</tr>
<tr>
<td></td>
<td>Treatment of PE</td>
<td>Total dose of 100 mg: 10 mg i.v. bolus</td>
<td>If weight &lt; 65 Kg: max dose &lt; 1.5 mg/kg</td>
<td>Non-compressible punctures in the past 24h (e.g. liver biopsy, lumbar puncture)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>followed by 90 mg i.v. for 2h</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
**Drug** | **Indications** | **Dose** | **Dose adjustments** | **Comments**
---|---|---|---|---
Reteplase (rt-PA) | STEMI | 10 units + 10 units i.v. bolus given 30 min apart | Renal and hepatic impairment: caution advised | Absolute contraindications to fibrinolytics: Previous intracranial haemorrhage or stroke of unknown origin at any time Ischaemic stroke in the preceding 6 months Central nervous system damage or neoplasms or atrioventricular malformation Recent major trauma/surgery/head injury (within the preceding 3 weeks) Gastrointestinal bleeding within the past month Known bleeding disorder (excluding menses) Aortic dissection Non-compressible punctures in the past 24h (e.g. liver biopsy, lumbar puncture)
Tenecteplase (TNK-tPA) | STEMI | Over 10 seconds; Single i.v. bolus: 30 mg if < 60 kg 35 mg if 60 to < 70 kg 40 mg if 70 to < 80 kg 45 mg if 80 to < 90 kg 50 mg if ≥ 90 kg | - |
**Antiischemic drugs**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Indications</th>
<th>Dose</th>
<th>Dose adjustments</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><strong>Beta-blockers</strong>: Preferred over calcium channel blockers - Contraindicated if coronary spasm, severe bradycardia, AV block, severe bronchospasm</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atenolol</td>
<td>NSTE-ACS</td>
<td>LD: 25-100 mg oral</td>
<td>Elderly: start at a lower dose</td>
<td>Only if normal LVEF</td>
</tr>
<tr>
<td></td>
<td></td>
<td>MD: 25-100 mg QD</td>
<td>CrCl: 15-35 ml/min: max dose 50 mg/day; CrCl &lt; 15 ml/min: max dose 25 mg/day</td>
<td></td>
</tr>
<tr>
<td></td>
<td>STEMI</td>
<td>25-100 mg QD, titrate as tolerated up to 100 mg QD only if no LVSD or CHF</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carvedilol</td>
<td>NSTE-ACS</td>
<td>LD: 3.125-25 mg oral</td>
<td>Caution in elderly and hepatic impairment</td>
<td>Preferred if LVSD/HF</td>
</tr>
<tr>
<td></td>
<td></td>
<td>MD: 3.125-25 mg BID</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>STEMI</td>
<td>3.125-6.25 mg BID, titrated as tolerated up to 50 mg BID</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bisoprolol</td>
<td>NSTE-ACS</td>
<td>LD: 1.25-10 mg oral</td>
<td>Caution in renal or hepatic impairment</td>
<td>Preferred if LVSD/HF</td>
</tr>
<tr>
<td></td>
<td></td>
<td>MD: 1.25-10 mg QD</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>STEMI</td>
<td>1.25-5 mg QD, titrate as tolerated up to 10 mg QD</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metoprolol</td>
<td>NSTE-ACS</td>
<td>LD: 25-100 mg oral</td>
<td>Caution in hepatic impairment</td>
<td>Preferred if LVSD/HF</td>
</tr>
<tr>
<td></td>
<td></td>
<td>MD: 25-100 mg BID</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>STEMI</td>
<td>5-25 mg BID,</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>titrate as tolerated up to 200 mg QD</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Calcium antagonists:</strong> Consider if beta-blockers are contraindicated. First option in vasospastic angina</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Verapamil</td>
<td>ACS</td>
<td>LD: 80-120 mg oral</td>
<td>Caution in elderly, renal or hepatic impairment</td>
<td>Contraindicated if bradycardia, HF, LVSD</td>
</tr>
<tr>
<td></td>
<td></td>
<td>MD: 80-240 mg TID-QD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diltiazem</td>
<td>ACS</td>
<td>LD: 60-120 mg oral</td>
<td>Caution in elderly and hepatic impairment</td>
<td>Contraindicated if bradycardia, HF, LVSD</td>
</tr>
<tr>
<td></td>
<td></td>
<td>MD: 60-300 mg TID-QD</td>
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<td>Calcium antagonists:</td>
<td>Consider if beta-blockers are contraindicated. First option in vasospastic angina</td>
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<td></td>
</tr>
<tr>
<td><strong>Amlodipine</strong></td>
<td>ACS</td>
<td>LD: 5-10 mg oral, MD: 5-10 mg QD</td>
<td>Caution in hepatic impairment</td>
<td>Contraindicated if hypotension</td>
</tr>
<tr>
<td>Nitrates</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Nitroglycerin</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>i.v.</td>
<td>ACS</td>
<td>If intolerant or unresponsive to nitroglycerin s.l. 5 μg/min - Increase by 5 mcg/min q3-5min up to 20 μg/min - If 20 mcg/min is inadequate, increase by 10 to 20 μg/min every 3 to 5min - Max dose: 400 μg/min</td>
<td>-</td>
<td>Contraindicated if severe hypotension and co-administration with phosphodiesterase inhibitors. The most common adverse effects are headache and dizziness.</td>
</tr>
<tr>
<td>spray</td>
<td>Angina</td>
<td>1-2 puff s.l. every 5min as needed, up to 3 puff in 15min</td>
<td>-</td>
<td>Use glass bottles for nitroglycerin i.v. administration</td>
</tr>
<tr>
<td>sublingual tablet</td>
<td>Angina</td>
<td>0.3 to 0.6 mg s.l. or in the buccal pouch every 5min as needed, up to 3 doses in 15min</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

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### Antiischemic drugs (Cont.)

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</thead>
<tbody>
<tr>
<td>Isosorbide mononitrate</td>
<td>Angina</td>
<td>5-10 mg BID with the two doses given 7h apart (8am and 3pm) to decrease tolerance development - then titrate to 10 mg BID in first 2-3 days <strong>Extended release tablet:</strong> Initial: 30-60 mg given in the morning as a single dose Titrate upward as needed, giving at least 3 days between increases Max daily single dose: 240mg</td>
<td>-</td>
<td>Contraindicated if severe hypotension and co-administration with phosphodiesterase inhibitors The most common adverse effects are headache and dizziness</td>
</tr>
<tr>
<td>Isosorbide dinitrate</td>
<td>Angina</td>
<td>Initial dose: 5 to 20 mg orally 2 or 3 times/day MD: 10 to 40 mg orally 2 or 3 times a day Extended release: 40 to 160 mg/day orally</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Nitroglycerin transdermal patch</td>
<td>Angina</td>
<td>0.2 to 0.4 mg/h patch applied topically once a day for 12 to 14h per day; titrate as needed and tolerated up to 0.8 mg/h</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>
### Antiischemic drugs (Cont.)

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<th>Dose adjustments</th>
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</tr>
</thead>
<tbody>
<tr>
<td><strong>Ivabradine</strong></td>
<td>Stable angina</td>
<td>5-7.5 mg oral BID</td>
<td>Caution in elderly and CrCl &lt; 15ml/min</td>
<td>Contraindicated if severe hepatic impairment</td>
</tr>
</tbody>
</table>
| **Ranolazine**| Stable angina          | Initial dose: 375 mg oral BID  
After 2-4 weeks, the dose should be titrated to 500 mg BID and, according to the patient's response, further titrated to a recommended max dose of 750 mg BID | Use with caution in renal and hepatic impairment, CHF, elderly, low weight | Contraindicated if CrCl < 30ml/min, concomitant administration of potent CYP3A4 inhibitors, moderate or severe hepatic impairment |
| **Trimetazidine** | Stable angina         | Modified-release: 35 mg oral BID | Caution in elderly and 30 < CrCl < 60ml/min | Contraindicated in parkinson disease, parkinsonian symptoms, tremors, restless leg syndrome, movement disorders, severe renal impairment |

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**Hypolipidemic drugs**

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<th>Dose adjustments</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Statins: Secondary prevention of cardiovascular disease: start with high doses and down titrate if side effects</td>
<td>Target LDL-C levels &lt; 70 mg/dl initiated early after admission</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atorvastatin</td>
<td></td>
<td>LDL-C reduction</td>
<td></td>
<td>Contraindicated in patients with active liver disease or with unexplained elevation of liver function enzyme levels</td>
</tr>
<tr>
<td>Rosuvastatin</td>
<td></td>
<td>&lt;30%</td>
<td>30-40%</td>
<td>40-50%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Simva 10 mg</td>
<td>Simva 20-40 mg</td>
<td>Simva 40 mg</td>
</tr>
<tr>
<td>Pitavastatin</td>
<td></td>
<td>Lova 20 mg</td>
<td>Ator 10 mg</td>
<td>Ator 20-40 mg</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Prava 20-40 mg</td>
<td>Prava 40 mg</td>
<td>Rosu 10-20 mg</td>
</tr>
<tr>
<td>Simvastatin</td>
<td></td>
<td>Prava 40 mg</td>
<td>Rosu 10-20 mg</td>
<td></td>
</tr>
<tr>
<td>Fluvastatin</td>
<td></td>
<td>Fluva 40 mg</td>
<td>Fluva 80 mg</td>
<td>Pita 4 mg</td>
</tr>
<tr>
<td>Pravastatin</td>
<td></td>
<td>Pita 1 mg</td>
<td>Rosu 5 mg</td>
<td>Simva/ezet 20/10 mg</td>
</tr>
<tr>
<td>Lovastatin</td>
<td></td>
<td>Pita 2 mg</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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**Hypolipidemic drugs (Cont.)**

<table>
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<th>Dose</th>
<th>Dose adjustments</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Others</td>
<td>Hyperlipidemia</td>
<td>10 mg oral QD</td>
<td>Avoid use if moderate-severe hepatic impairment</td>
<td></td>
</tr>
<tr>
<td>Ezetimibe</td>
<td>Hyperlipidemia</td>
<td>48-160 mg oral QD</td>
<td>CrCl 50-90ml/min: start 48-54 mg QD</td>
<td>Contraindicated if CrCl &lt; 50ml/min or hepatic impairment</td>
</tr>
<tr>
<td>Fenofibrate</td>
<td>Hyperlipidemia</td>
<td>900-1200 mg/day oral</td>
<td></td>
<td>Contraindicated if severe renal impairment or hepatic dysfunction</td>
</tr>
<tr>
<td>Gemfibrozil</td>
<td>Hyperlipidemia</td>
<td>10 mg oral QD</td>
<td></td>
<td>Statins may increase muscle toxicity: avoid concomitant use</td>
</tr>
<tr>
<td>Evolocumab</td>
<td>PCSK9 inhibitor</td>
<td>900-1200 mg/day oral</td>
<td></td>
<td>Most common side effects: nasopharyngitis, upper respiratory tract infection, headache and back pain</td>
</tr>
</tbody>
</table>

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## Heart failure & hypertension

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<thead>
<tr>
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<th>Dose</th>
<th>Dose adjustments</th>
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</tr>
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<tbody>
<tr>
<td><strong>ACEI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Captopril</td>
<td>HF</td>
<td>Start: 6.25 mg oral TID</td>
<td>CrCl &gt; 50 ml/min: 75-100% of the normal dose</td>
<td>Check renal function, electrolytes, drug interactions</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Target dose: 50 mg TID</td>
<td>CrCl 10-50 ml/min: 25-50%</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>CrCl &lt; 10 ml/min: 12.5%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>HTN</td>
<td>Start: 12.5 mg oral BID</td>
<td></td>
<td>Major contraindications: History of angioedema, known bilateral renal artery stenosis, pregnancy (risk)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Target dose: 25-50 mg TID</td>
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<td></td>
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<tr>
<td></td>
<td></td>
<td>Max 450 mg/day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Enalapril</td>
<td>HF, HTN</td>
<td>Start: 2.5 mg oral BID</td>
<td>CrCl 30-80 ml/min: start 5 mg/day</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Target dose: 10-20 mg BID</td>
<td>CrCl 10-30 ml/min: start 2.5 mg/day</td>
<td></td>
</tr>
<tr>
<td>Lisinopril</td>
<td>HF</td>
<td>Start: 2.5-5.0 mg oral QD</td>
<td>CrCl 31-80 ml/min: start 5-10 mg/day</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Target dose: 20-35 mg QD</td>
<td>CrCl 10-30 ml/min: start 2.5-5 mg/day</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>CrCl &lt; 10 ml/min: start 2.5 mg/day</td>
<td></td>
</tr>
<tr>
<td></td>
<td>HTN</td>
<td>10-20 mg oral QD</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Max: 80 mg QD</td>
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</tbody>
</table>

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### Heart failure & hypertension (Cont.)

| Drug       | Indications | Dose                        | Dose adjustments                                                                 |
|------------|-------------|                            |                                                                               |
| **Perindopril** | HF          | Start: 2.5 mg oral QD       | CrCl > 60ml/min: start 5 mg/day                                                 |
|            |             | Max: 5mg QD                 | CrCl 31-60ml/min: start 2.5 mg/day                                              |
|            | HTN         | Start: 2.5-5 mg QD          | CrCl 15-30ml/min: start 2.5 mg alternate days                                   |
|            |             | Target dose: 10 mg QD       | CrCl < 15ml/min: start 2.5 mg/day on the day of dialysis                        |
| **Ramilpril** | HF, HTN     | Start: 2.5 mg oral QD       | CrCl < 40ml/min: start 1.25 mg QD, max 5 mg/day                                |
|            |             | Target dose: 5 mg BID       | Caution in elderly and hepatic impairment                                        |
| **Trandolapril** | HF          | Start: 0.5 mg oral QD       | CrCl < 30ml/min or severe hepatic impairment: start 0.5 mg                     |
|            |             | Target dose: 4 mg QD        |                                                                               |
|            | HTN         | 2-4 mg oral QD              | CrCl < 30ml/min or severe hepatic impairment: start 0.5 mg                     |

**Comments**

Check renal function, electrolytes, drug interactions.

Major contraindications: History of angioedema, known bilateral renal artery stenosis, pregnancy (risk).
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</tr>
</thead>
<tbody>
<tr>
<td>ARB</td>
<td>HF, HTN</td>
<td>Start: 4-8 mg oral QD&lt;br&gt;Target dose: 32 mg QD&lt;br&gt;if renal or hepatic impairment: start 4 mg/day</td>
<td>If ACEI is not tolerated. Check renal function, electrolytes, drug interactions&lt;br&gt;Major contraindications: History of angioedema, known bilateral renal artery stenosis, pregnancy (risk)</td>
<td></td>
</tr>
<tr>
<td>Candesartan</td>
<td>HF</td>
<td>Start: 50 mg oral QD&lt;br&gt;Target dose: 150 mg QD&lt;br&gt;CrCl &lt; 20ml/min: 25 mg QD&lt;br&gt;Caution if hepatic impairment</td>
<td>If renal or hepatic impairment: start 4 mg/day</td>
<td></td>
</tr>
<tr>
<td>Losartan</td>
<td>HF</td>
<td>Start: 50 mg oral QD&lt;br&gt;Target dose: 150 mg QD&lt;br&gt;CrCl &lt; 20ml/min: 25 mg QD&lt;br&gt;Caution if hepatic impairment</td>
<td>If ACEI is not tolerated. Check renal function, electrolytes, drug interactions&lt;br&gt;Major contraindications: History of angioedema, known bilateral renal artery stenosis, pregnancy (risk)</td>
<td></td>
</tr>
<tr>
<td>HTN</td>
<td>50-100 mg oral QD&lt;br&gt;CrCl &lt; 20ml/min: 25 mg QD&lt;br&gt;Caution if hepatic impairment</td>
<td>If ACEI is not tolerated. Check renal function, electrolytes, drug interactions&lt;br&gt;Major contraindications: History of angioedema, known bilateral renal artery stenosis, pregnancy (risk)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Valsartan</td>
<td>HF</td>
<td>Start: 40 mg oral BID&lt;br&gt;Target dose: 160 mg BID&lt;br&gt;CrCl &lt; 20ml/min: 25 mg QD&lt;br&gt;Caution if hepatic impairment</td>
<td>If ACEI is not tolerated. Check renal function, electrolytes, drug interactions&lt;br&gt;Major contraindications: History of angioedema, known bilateral renal artery stenosis, pregnancy (risk)</td>
<td></td>
</tr>
<tr>
<td>HTN</td>
<td>80-160 mg QD&lt;br&gt;CrCl &lt; 20ml/min: 25 mg QD&lt;br&gt;Caution if hepatic impairment</td>
<td>If mild-moderate hepatic impairment: max dose 80 mg/day</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td><strong>Beta-blockers:</strong> Check 12-lead ECG</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atenolol</td>
<td>HTN</td>
<td>Start: 25 mg oral QD</td>
<td>CrCl 10-50ml/min: decrease dose 50%</td>
<td>Major contraindications: asthma, 2nd or 3rd degree AV block</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Usual dose: 50-100 mg QD</td>
<td>CrCl &lt; 10ml/min: decrease dose 75%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>HF</td>
<td>Start: 1.25 mg oral QD</td>
<td>CrCl &lt; 20ml/min: max dose 10 mg QD</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Target dose: 10 mg QD</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>HTN</td>
<td>Start: 2.5-5 mg oral QD</td>
<td></td>
<td>Hepatic impairment: avoid use</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Usual dose: 5-10 mg QD</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Max dose: 20 mg QD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bisoprolol</td>
<td>HF</td>
<td>Start: 12.5-25 mg oral QD</td>
<td></td>
<td>Hepatic impairment: start with low doses and titrate gradually</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Target dose: 200 mg QD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metoprolol</td>
<td>HTN</td>
<td>100-400 mg QD</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Max dose: 400 mg QD</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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### Heart failure & hypertension (Cont.)

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<th>Dose adjustments</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Beta-blockers:</strong> Check 12-lead ECG</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Cardioselective</strong>&lt;br&gt; Nebivolol&lt;br&gt;HF&lt;br&gt;Start: 1.25 mg oral QD&lt;br&gt;Target dose: 10 mg QD&lt;br&gt;Renal impairment or elderly:&lt;br&gt;start dose 2.5 mg QD, titrate to 5 mg QD&lt;br&gt;Hepatic impairment: contraindicated</td>
<td></td>
<td></td>
<td>Major contraindications: asthma, 2nd or 3rd degree AV block</td>
<td></td>
</tr>
<tr>
<td><strong>Cardioselective</strong>&lt;br&gt; Nebivolol&lt;br&gt;HTN&lt;br&gt;Start: 2.5 mg oral QD&lt;br&gt;Usual dose: 5 mg QD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Non-cardioselective</strong>&lt;br&gt; Carvedilol&lt;br&gt;HF&lt;br&gt;Start: 3.125 mg oral BID&lt;br&gt;Target dose: 25-50 mg BID</td>
<td></td>
<td></td>
<td>Caution in elderly&lt;br&gt;Contraindicated if hepatic impairment</td>
<td></td>
</tr>
<tr>
<td><strong>Non-cardioselective</strong>&lt;br&gt; Carvedilol&lt;br&gt;HTN&lt;br&gt;Start: 12.5 mg oral QD&lt;br&gt;Usual dose: 25 mg QD&lt;br&gt;and max dose: 25 mg BID or 50 mg QD</td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
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## Heart failure & hypertension (Cont.)

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</tr>
</thead>
<tbody>
<tr>
<td><strong>Other vasodilators</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amlodipine</td>
<td>HTN</td>
<td>Start: 5 mg oral QD, increase after 1-2 weeks Max: 10 mg/day</td>
<td>Elderly or secondary agent: start 2.5 mg QD Hepatic impairment: start 2.5 mg QD</td>
<td>Contraindicated if cardiogenic shock, 2nd or 3rd degree AV block, severe hypotension</td>
</tr>
<tr>
<td>Nifedipine</td>
<td>HTN</td>
<td>Extended-release form: Start 20 mg oral BID or TID Max: 60 mg BID</td>
<td>Renal and hepatic impairment: caution advised</td>
<td></td>
</tr>
<tr>
<td>Clevidipine</td>
<td>HTN</td>
<td>Initiate the IV infusion at 4 ml/h (2 mg/h); the dose may be doubled every 90 seconds Uptitration until desired BP range is achieved Half life of 1-2min</td>
<td>The desired therapeutic response for most patients occurs at doses of 8-12 ml/h (4-6 mg/h) The max recommended dose is 64 ml/h (32 mg/h)</td>
<td>Hypersensitivity to soy, peanut, or egg products Critical Aortic stenosis, mitral stenosis, HOCM</td>
</tr>
</tbody>
</table>
### Other vasodilators

<table>
<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>Verapamil</td>
<td>HTN</td>
<td><strong>Immediate-release form:</strong></td>
<td>Start 40 mg oral TID in elderly or small stature patients</td>
<td>Contraindicated if bradycardia, HF, LVSD</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dose: 80-120 mg oral TID;</td>
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<td></td>
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<td>Start: 80 mg TID;</td>
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<td></td>
<td></td>
<td>Max: 480 mg/day</td>
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</table>

### Loop diuretics

<table>
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<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>Furosemide</td>
<td>HF</td>
<td>20-40 mg i.v. bolus, continuous 100 mg/6h</td>
<td>Anuria: contraindicated</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(adjust based on kidney function and clinical findings; monitor creatinine)</td>
<td>Cirrhosis/ascites: caution advised</td>
<td></td>
</tr>
<tr>
<td></td>
<td>HTN</td>
<td>10-40 mg oral BID</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>Torsemide</td>
<td>HF</td>
<td>10-20 mg oral or i.v. QD</td>
<td>Hepatic impairment: initial dose should be reduced by 50% and dosage adjustments made cautiously</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>HTN</td>
<td>5 mg oral or i.v. QD</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Max 10 mg QD</td>
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</tbody>
</table>
### Heart failure & hypertension (Cont.)

<table>
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<tr>
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</tr>
</thead>
<tbody>
<tr>
<td><strong>Thiazides</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chlorthalidone</td>
<td>HF</td>
<td>50-100 mg oral QD</td>
<td>Elderly: max dose 25 mg/day</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td></td>
<td>MD: 25-50 mg QD</td>
<td>CrCl &lt; 25 ml/min: avoid use</td>
<td></td>
</tr>
<tr>
<td></td>
<td>HTN</td>
<td>Start 12.5-25 mg oral QD;</td>
<td>Elderly: max dose 25 mg/day</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Max: 50 mg/day</td>
<td>CrCl &lt; 25 ml/min: avoid use</td>
<td></td>
</tr>
<tr>
<td>Hydrochlorothiazide</td>
<td>HF</td>
<td>25-200 mg oral/day</td>
<td>CrCl &lt; 25 ml/min: avoid use</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>HTN</td>
<td>Start 12.5-25 mg oral QD;</td>
<td>CrCl &lt; 25 ml/min: avoid use</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td></td>
<td>MD: may increase to 50 mg oral</td>
<td>Hepatic impairment: caution advised</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>as a single or 2 divided doses</td>
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<td></td>
</tr>
</tbody>
</table>

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**Heart failure & hypertension (Cont.)**

<table>
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</tr>
</thead>
<tbody>
<tr>
<td><strong>Thiazides</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indapamide</td>
<td>HTN</td>
<td>Start 1.25 mg PO QAM x4weeks, then increase dose if no response Max: 5 mg/day</td>
<td>CrCl &lt; 25 ml/min: avoid use Hepatic impairment: caution advised</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>HF</td>
<td>Start 25 mg oral QD Target dose: 25-50 mg QD</td>
<td>CrCl &lt; 10ml/min, anuria or acute renal impairment: contraindicated Severe hepatic impairment and elderly: caution advised</td>
<td>Check renal function, electrolytes, drug interactions Produces gynecomastia</td>
</tr>
<tr>
<td></td>
<td>HTN</td>
<td>50-100 mg/day oral</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Heart failure & hypertension (Cont.)

#### Aldosterone-antagonists

<table>
<thead>
<tr>
<th>Drug</th>
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<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eplerenone</td>
<td>HF</td>
<td>Start 25 mg oral QD</td>
<td>Elderly: caution advised</td>
<td>Check renal function, electrolytes, drug interactions Major contraindications: strong CYP3A4 inhibitors</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Target dose: 50 mg QD</td>
<td>CrCl &lt; 50ml/min: contraindicated</td>
<td></td>
</tr>
<tr>
<td></td>
<td>HTN</td>
<td>50 mg oral QD-BID</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Max: 100 mg/day</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>HTN</td>
<td>50 mg oral QD-BID</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Max: 100 mg/day</td>
<td></td>
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</tr>
</tbody>
</table>

#### Others

<table>
<thead>
<tr>
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<th>Dose</th>
<th>Dose adjustments</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ivabradine</td>
<td>HF</td>
<td>5-7.5 mg oral BID</td>
<td>Caution in elderly and CrCl &lt; 15ml/min</td>
<td>Contraindicated if severe hepatic impairment</td>
</tr>
</tbody>
</table>
### Inotropics & vasopressors

<table>
<thead>
<tr>
<th>Drug</th>
<th>Indications</th>
<th>Dose</th>
<th>Dose adjustments</th>
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</tr>
</thead>
<tbody>
<tr>
<td><strong>Levsimendan</strong></td>
<td>HF/cardiogenic shock</td>
<td>LD: 6 to 12 μg/kg i.v. over 10 min (given only if immediate effect is needed) followed by 0.05 to 0.2 μg/kg/min as a continuous infusion for 24h</td>
<td>Avoid use if CrCl &lt; 30ml/min or severe hepatic impairment</td>
<td>Calcium sensitizer and ATP-dependent potassium channel opener</td>
</tr>
<tr>
<td><strong>Milrinone</strong></td>
<td>HF/cardiogenic shock</td>
<td>50 μg/kg i.v. in 10-20 min, continuous 0.375-0.75 μg/kg/min</td>
<td>Renal: Same bolus. Adjust infusion: CrCl 50ml/min: start 0.43 μg/kg/min CrCl 40ml/min: start 0.38 μg/kg/min CrCl 30ml/min: start 0.33 μg/kg/min CrCl 20ml/min: start 0.28 μg/kg/min CrCl 10ml/min: start 0.23 μg/kg/min CrCl 5ml/min: start 0.20 μg/kg/min</td>
<td>Phosphodiesterase inhibitor Caution if atrial flutter Hypotensive drug</td>
</tr>
<tr>
<td><strong>Isoprenaline/Isoproterenol</strong></td>
<td>Cardiogenic shock</td>
<td>0.5-5 μg/min (0.25-2.5 ml of a 1:250,000 dilution) i.v. infusion</td>
<td>-</td>
<td>β1, β2 agonist. Contraindicated in patients with tachyarrhythmia, tachycardia or heart block caused by digitalis intoxication, ventricular arrhythmias which require inotropic therapy, angina pectoris, recent ACS, hyperthyroidism</td>
</tr>
<tr>
<td></td>
<td>Bradyarrhythmias</td>
<td>Bolus: 20-40 μg i.v. Infusion: 0.5 μg/min of 2 mg/100 ml normal saline</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

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<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dobutamine</td>
<td>Cardiogenic shock</td>
<td>2-20 μg/kg/min i.v.</td>
<td>-</td>
<td>β1, α1/β2 agonist. Increases contractility with little effect on heart</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>rate and blood pressure. Reduces pulmonary and systemic VR, PCP</td>
</tr>
<tr>
<td>Dopamine</td>
<td>Cardiogenic shock</td>
<td>Dopaminergic effect: 2-5 μg/Kg/min i.v.</td>
<td>-</td>
<td>β, α, dopaminergic agonist Increases BP, PAP, heart rate, cardiac output</td>
</tr>
<tr>
<td></td>
<td></td>
<td>β effect : 5-15 μg/Kg/min i.v.</td>
<td></td>
<td>and pulmonary and systemic VR</td>
</tr>
<tr>
<td></td>
<td></td>
<td>α effect : 15-40 μg/Kg/min i.v.</td>
<td></td>
<td>More arrhythmogenic than dobutamine and noradrenaline</td>
</tr>
<tr>
<td>Noradrenaline</td>
<td>Cardiogenic shock</td>
<td>0.05-0.2 μg/kg/min i.v. titrate to effect</td>
<td>-</td>
<td>α 1, β1 agonist Increases BP and PAP Little arrhythmogenic</td>
</tr>
</tbody>
</table>

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### Antiarrhythmics

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</tr>
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<tbody>
<tr>
<td><strong>Group I</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| **Procainamide i.v.** | AF (termination); stable VT (with a pulse) | 15-18 mg/kg i.v. over 60 min, followed by infusion of 1-4 mg/min | Reduce LD to 12 mg/kg in severe renal impairment  
Reduce MD by one-third in moderate renal impairment and by two-thirds in severe renal impairment  
Caution in elderly and asthma | Hypotension  
(negative inotropic agent)  
Lupus-like syndrome  
Contraindicated if myasthenia gravis, AV block, severe renal impairment |
|               |                              |                                           |                                                                                  |                                                                          |
| **Lidocaine i.v.** | Pulseless VT/VF               | 1-1.5 mg/kg i.v./i.o. bolus (can give additional 0.5-0.75 mg/kg i.v./i.o. push every 5-10 min if persistent VT/VF, max cumulative dose = 3 mg/kg), followed by infusion of 1-4 mg/min | 1-2 mg/min infusion if liver disease or HF | Contraindicated if advanced AV block, bradycardia, hypersensitivity to local anesthetics  
Caution in HF, renal impairment and elderly  
May cause seizures, psychosis.  
Stop if QRS widens > 50% |
|               | Stable VT (with a pulse)     | 1-1.5 mg/kg i.v. bolus (can give additional 0.5-0.75 mg/kg i.v. push every 5-10min if persistent VT, max cumulative dose = 3 mg/kg), followed by infusion of 1-4 mg/min | 1-2 mg/min infusion if liver disease or HF | Caution in HF, renal impairment and elderly  
May cause seizures, psychosis.  
Stop if QRS widens > 50% |

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<tbody>
<tr>
<td><strong>Group I</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flecaide i.v.</td>
<td>SVT, ventricular arrhythmias</td>
<td>2 mg/kg (max 150 mg) i.v. over 30min</td>
<td>Severe renal impairment: caution advised</td>
<td>Contraindicated if cardiogenic shock, recent MI, 2nd or 3rd degree AV block</td>
</tr>
<tr>
<td></td>
<td></td>
<td>This may be followed by an infusion at a rate of 1.5 mg/kg/h for 1 h, reduced to 0.1-0.25 mg/kg/h for up to 24h, max cumulative dose = 600 mg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Propafenone i.v.</td>
<td>PSVT, ventricular arrhythmias</td>
<td>LD: 0.5-2 mg/kg i.v. direct over a minimum of 3-5min</td>
<td>May need to reduce dose in renal or hepatic failure</td>
<td>Contraindicated if unstable HF, cardiogenic shock, AV block, bradycardia, myasthenia gravis severe hypotension, bronchospastic disorders, Brugada syndrome</td>
</tr>
<tr>
<td></td>
<td></td>
<td>MD: 0.5-2.5 mg/kg i.v. direct q8h</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>(max 560 mg/day) or continuous infusion up to 23 mg/h</td>
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</tbody>
</table>
## Antiarrhythmics (Cont.)

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<tbody>
<tr>
<td><strong>Group II</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Atenolol i.v.</td>
<td>Arrhythmias</td>
<td>2.5 mg i.v. over 2.5 min every 5 min (max 10 mg)</td>
<td>Caution in elderly and/or severe renal impairment</td>
<td>Contraindicated if cardiogenic shock, bradycardia and greater than first-degree block, unstable HF</td>
</tr>
<tr>
<td>Metoprolol i.v.</td>
<td>Arrhythmias</td>
<td>2.5-5 mg i.v. over 5 min, may repeat every 5 min (max 15 mg)</td>
<td>Caution if severe hepatic impairment</td>
<td>Contraindicated if cardiogenic shock, bradycardia and greater than first-degree block, unstable HF</td>
</tr>
<tr>
<td>Propranolol i.v.</td>
<td>Arrhythmias</td>
<td>Initially given as slow i.v. boluses of 1 mg, repeated at 2 min intervals (max: 10 mg in conscious patients and 5 mg if under anesthesia)</td>
<td>-</td>
<td>Contraindicated if cardiogenic shock, bradycardia and greater than first-degree block, asthma, unstable HF</td>
</tr>
</tbody>
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<tr>
<td><strong>Group III</strong></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Amiodarone i.v.</td>
<td>AF (termination)</td>
<td>5 mg/Kg i.v. over 30 min, followed by infusion of 1 mg/min for 6h, then 0.5 mg/min</td>
<td>-</td>
<td>Reduce infusion rate if bradycardia, AV block, hypotension</td>
</tr>
<tr>
<td></td>
<td>Stable VT (with a pulse)</td>
<td>150 mg i.v. over 10 min followed by infusion of 1 mg/min for 6h, then 0.5 mg/min</td>
<td>-</td>
<td>Bolus should be avoided if hypotension or severe LV dysfunction</td>
</tr>
<tr>
<td></td>
<td>Pulseless VT/VF</td>
<td>300 mg bolus i.v. (can give additional 150 mg i.v. bolus if VF/VT persists) followed by infusion of 900 mg over 24h</td>
<td>-</td>
<td>Highly vesicant agent</td>
</tr>
<tr>
<td>Dronedarone</td>
<td>Paroxysmal or persistent AF prevention</td>
<td>400 mg oral BID</td>
<td>-</td>
<td>Contraindicated if severe renal or liver dysfunction, LVSD, symptomatic HF, permanent AF, bradycardia… (multiple contraindications)</td>
</tr>
</tbody>
</table>

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**Antiarrhythmics (Cont.)**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Indications</th>
<th>Dose</th>
<th>Dose adjustments</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group IV</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Diltiazem</strong></td>
<td>PSVT; AF (rate control)</td>
<td>0.25 mg/kg i.v. over 2 min (may repeat with 0.35 mg/kg i.v. over 2 min), followed by infusion of 5-15 mg/h</td>
<td>Hepatic impairment: caution advised</td>
<td>-</td>
</tr>
<tr>
<td>i.v.</td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>
| **Verapamil**                         | PSVT; AF (rate control)                                | 2.5-5 mg i.v. over 2 min (may repeat up to max cumulative dose of 20 mg); can follow with infusion of 2.5-10 mg/h | -                                 | Contraindicated if AF+WPW, tachycardias QRS (except RVOT-VT), fascicular VT, bronchospasm, age>70 | Antidote: - LVD: Calcium gluconate, dobutamine  
- Bradycardia/AV block: Atropine, Isoproterenol |
| i.v.      |                                                         |                                                                      |                                   |                                                                          |
| **Adenosine**                         | Rapid conversion to a normal sinus rhythm of PSVT including those associated with accessory by-pass tracts (WPW syndrome) | Rapid i.v. boluses separated by 2 min: 6 mg → 6 mg → 12 mg            | -                                 | Contraindicated if sick sinus syndrome, second or third degree Atrio-Ventricular (AV) block (except in patients with a functioning artificial pacemaker), chronic obstructive lung disease with evidence of bronchospasm (e.g. asthma bronchiale), long QT syndrome, severe hypotension; decompensated states of heart failure - Adenosine can cause AF |
## Antiarrhythmics (Cont.)

<table>
<thead>
<tr>
<th>Drug</th>
<th>Indications</th>
<th>Dose</th>
<th>Dose adjustments</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Others</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Magnesium sulfate</td>
<td>VT-Torsades de Pointes</td>
<td>Bolus: 1-2 g i.v./i.o. over 5 min Perfusion: 5-20 mg/min i.v.</td>
<td>Caution if severe renal failure</td>
<td>Contraindicated if myasthenia gravis</td>
</tr>
<tr>
<td>Vernakalant</td>
<td>Acute atrial fibrillation</td>
<td>3 mg/kg i.v. over 10 min. If AF persists, a second 10-min-infusion of 2 mg/kg, 15 min later may be administered</td>
<td>-</td>
<td>Contraindicated if ACS within the last 30 days, severe aortic stenosis, SBP &lt; 100mmHg, HF class NYHA III/IV, severe bradycardia, sinus node dysfunction or 2nd or 3rd degree heart block</td>
</tr>
</tbody>
</table>

**DISCLAIMER:** The guidance suggested in this document does not override the individual responsibility of the healthcare professional to make appropriate decisions according to each patient's circumstances and profile, as well as local regulations and licenses.
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Full Form</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>ACT</td>
<td>Activated clotting time</td>
</tr>
<tr>
<td>AD</td>
<td>Aortic Dissection</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>aPRR</td>
<td>Activated partial thromboplastin time</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>CrCl</td>
<td>Creatinine clearance</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>
</tbody>
</table>
Abbreviations

DVT = Deep vein thrombosis
ECG = Electrocardiogram
ED = Emergency department
EG = Electrograms
EMB = Endomyocardial biopsy
EMS = Emergency medical services
EPS = Electrophysiological study
ERC = European Resuscitation Council
ESR = Erythrocyte sedimentation rate
ETT = Exercice treadmill testing
FMC = First medical contact
GER = Gastroesophageal reflux
GFR = Glomerular flow rate
GI = Gastrointestinal
GP = Glycoprotein
HF = Heart failure
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
LVD = Left ventricular dysfunction
LVEF = Left ventricular ejection fraction
LVH = Left ventricular hypertrophy
LVSD = Left ventricular systolic dysfunction
MCS = Mechanical circulatory support
MD = Maintenance dose
MDCT = Computed tomography with >4 elements
MI = Myocardial infarction
MRI = Magnetic resonance imaging
Mvo = Microvascular obstruction
NIV = Non-invasive ventilation
NOAC = New oral anticoagulants
NSAID = Non-steroidal anti-inflammatory drugs
NSTEACS = Non-ST-elevation ACS
NSTEMI = Non ST-segment elevation myocardial infarction
NTG = Nitroglycerin
NT-proBNP = N-terminal pro brain natriuretic peptide
NVAF = Non-valvular atrial fibrillation
NYHA = New York Heart Association
OH = Orthostatic hypotension
PAP = Pulmonary arterial pressure
PAU = Penetrating aortic ulcer
PCI = Percutaneous coronary intervention
PCM = Physical counter-measures
PCP = Pulmonary capillary pressure
PE = Pulmonary embolism
PEA = Pulmonary endarterectomy
PEEP = Positive end expiratory pressure
PR = Pulmonary regurgitation
ProCT = Procalcitonin
PRN = Pro re nata
PSVT = Paroxysmal supraventricular tachycardia
QD = Once a day
QPM = Every evening
rtPA = Recombinant tissue plasminogen activator
RV = Right ventricular
RVOT-VT = Right ventricular outflow tract ventricular tachycardia
SBP = Systemic blood pressure
s.c = Subcutaneous
SLE = Systemic lupus erythematosus
SMU = Syncope management units
STE-ACS = ST-segment elevation acute coronary syndrome
STEMI = ST-segment elevation myocardial infarction
SVT = Supraventricular tachycardia
SpO₂ = Oxygen saturation
TEE = Transesophageal echocardiography
TEVAR = Thoracic endovascular aortic aneurysm repair
TIA = Transient ischemic attack
TID = Three times a day
TLOC = Transient loss of consciousness
Tn = Troponin
TOE = Transoesophageal echocardiography
TSH = Thyroid-stimulating hormone
Abbreviations

TTE = Transthoracic echocardiography
UFH = Unfractionated heparin
ULN = Upper limit of normal
VF = Ventricular fibrillation
VR = Vascular resistance
VT = Ventricular tachycardia
VTE = Venous thromboembolism
VVS = Vasovagal syncope
WHO = World Health Organization
WPW = Wolff-Parkinson-White
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European Heart Journal Aug 2015, DOI: 10.1093/eurheartj/ehv320


European Heart Journal Nov 2014, 35 (43) 3033-3073; DOI: 10.1093/eurheartj/ehu283

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Acute Cardiovascular Care Association
Clinical Decision-Making
TOOLKIT

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