Management of Coronary Artery Disease including Stable and Acute Coronary Syndromes

Dr Sukhjinder Nijjer
Consultant Cardiologist

Chelsea & Westminster NHS Foundation Trust
Imperial College Healthcare NHS Trust

s.nijjer@imperial.ac.uk
Declarations

• Honoraria
  • AstraZeneca
  • Bayer
  • Volcano
• 3 hour exam

• 120 questions

• 20% of Questions linked to IHD

**Category 2 - Ischaemic Heart Disease (approximately 20% of the questions)**

• **2.4 Invasive cardiac imaging**
  2.7 Cardiovascular Disease Protection 1- risk factors 2- hypertension
  2.8 Acute Coronary Syndromes

• **2.9 Chronic IHD**
  2.19 Physical activity in sport and primary and secondary prevention 1- sports cardiology 2- rehabilitation

The exam will include about 18 questions on ischemic heart disease diagnosis, treatment and intervention and 6 more or less equally divided between the other topics
A 60 year old male with Type II Diabetes and hypertension attended the emergency department with 15 minutes of central chest pain. ECG taken at the time of pain is shown. ECGs normalise with antiplatelet therapy and remain normal. Angiography revealed subtotal LAD lesion with TIMI III flow and he underwent 3.25x28 DES. Post-PCI troponin was 400. How should you code the final diagnosis?
A 60 year old male with Type II Diabetes and hypertension attended the emergency department after 15 minutes of central chest pain. Angiography revealed subtotal LAD lesion with TIMI III flow and he underwent 3.25x28 DES. Post-PCI troponin was 400. How should you code the final diagnosis?

A – STEMI
B – NSTEMI
C – Unstable angina
D – Type 2 MI
E – Type 4b MI
# Universal Classification of Myocardial Infarction

<table>
<thead>
<tr>
<th>Type 1: Spontaneous myocardial infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous myocardial infarction related to atherosclerotic plaque rupture, ulceration, fissuring, erosion, or dissection with resulting intraluminal thrombus in one or more of the coronary arteries leading to decreased myocardial blood flow or distal platelet emboli with ensuing myocyte necrosis. The patient may have underlying severe CAD but on occasion non-obstructive or no CAD.</td>
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<table>
<thead>
<tr>
<th>Type 2: Myocardial infarction secondary to an ischaemic imbalance</th>
</tr>
</thead>
<tbody>
<tr>
<td>In instances of myocardial injury with necrosis where a condition other than CAD contributes to an imbalance between myocardial oxygen supply and/or demand, e.g. coronary endothelial dysfunction, coronary artery spasm, coronary embolism, tachy-/brady-arrhythmias, anaemia, respiratory failure, hypotension, and hypertension with or without LVH.</td>
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<tr>
<th>Type 3: Myocardial infarction resulting in death when biomarker values are unavailable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac death with symptoms suggestive of myocardial ischaemia and presumed new ischaemic ECG changes or new LBBB, but death occurring before blood samples could be obtained, before cardiac biomarker could rise, or in rare cases cardiac biomarkers were not collected.</td>
</tr>
</tbody>
</table>
### Universal Classification of Myocardial Infarction

#### Type 4a: Myocardial infarction related to percutaneous coronary intervention (PCI)

Myocardial infarction associated with PCI is arbitrarily defined by elevation of cTn values $> 5 \times 99^{th}$ percentile URL in patients with normal baseline values ($\leq 99^{th}$ percentile URL) or a rise of cTn values $> 20\%$ if the baseline values are elevated and are stable or falling. In addition, either (i) symptoms suggestive of myocardial ischaemia, or (ii) new ischaemic ECG changes or new LBBB, or (iii) angiographic loss of patency of a major coronary artery or a side branch or persistent slow- or no-flow or embolization, or (iv) imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality are required.

#### Type 4b: Myocardial infarction related to stent thrombosis

Myocardial infarction associated with stent thrombosis is detected by coronary angiography or autopsy in the setting of myocardial ischaemia and with a rise and/or fall of cardiac biomarkers values with at least one value above the $99^{th}$ percentile URL.

#### Type 5: Myocardial infarction related to coronary artery bypass grafting (CABG)

Myocardial infarction associated with CABG is arbitrarily defined by elevation of cardiac biomarker values $> 10 \times 99^{th}$ percentile URL in patients with normal baseline cTn values ($\leq 99^{th}$ percentile URL). In addition, either (i) new pathological Q waves or new LBBB, or (ii) angiographic documented new graft or new native coronary artery occlusion, or (iii) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
Definitions

• **STEMI** > 20min ST elevation (+pain)

• **NSTE-ACS**
  - NSTEMI - +ve hs-c Tn & one clinical feature
  - Unstable angina – no evidence of cardiomyocyte necrosis (-ve hs-c Tn)

• MI = +ve hs-c Tn + 1 supporting clinical feature

• Type 1 – plaque event
• Type 2 – non-plaque event
• Type 3 – MI death with no biomarker
• Type 4 - 2ndry to PCI
• Type 5 - 2ndry to PCI CABG)
Your expertise is requested for a 62-year-old man with no prior history of cardiovascular disease who underwent open surgical treatment of an abdominal aortic aneurysm 48 h earlier. An increase in high-sensitivity troponin from 100 to 200 ng/L (ULN<14 ng/L) was observed within 24 hr of surgery. The patient did not have chest pain but is receiving morphine and ECG is normal. His haemoglobin level is 9.5 g/dL. Echocardiography shows normal LV function in the absence of regional wall motion abnormalities. Should aspirin and/or therapeutic doses of parenteral anticoagulation be started? Is any further investigation

A – Aspirin 300mg, clopidogrel 600mg & LMWH
B – DAPT with unfractionated heparin infusion
C – Aspirin with unfractionated heparin infusion
D – Aspirin with bivalirudin infusion
E – Immediate angiography
A 60 year old female with hypertension and statin therapy presents with moderate left thoracic chest pain without radiation that started 3 hours prior to admission and resolved spontaneously in the ER. Obs and examination are normal. 12-lead ECG shows no abnormality. You are using the 0h/1h high sensitivity TnT protocol – the first TnT is 3 ng/L (Elecsys assay rule out < 5ng/L). How should you proceed?

A – Repeat a further TNT at 1 hours
B – Repeat a further TNT at 3 hours
C – NSTEMI ruled out – discharge with outpatient investigation
D – ‘Rule out’ inpatient CTCA
E – Exercise treadmill test and discharge if normal
0h/1h hs-cTn algorithm
A tool for rapid rule in (angio) or rule out (discharge +/- non-invasive imaging test)
Absolute value vs change (Δ)

Observe = repeat TNT 3 – 6 hours or investigate further based on clinical assessment
A 60 year old female with hypertension and statin therapy presents with moderate left thoracic chest pain without radiation that started 3 hours prior to admission and resolved spontaneously in the ER. Obs and examination are normal. 12-lead ECG shows no abnormality. You are using the 0h/1h high sensitivity TnT protocol – the first TnT is 3 ng/L (Elecsys assay rule out < 5ng/L). How should you proceed?

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E – Exercise treadmill test and discharge if normal
A 60 year old female with hypertension and statin therapy presents with moderate left thoracic chest pain without radiation that started 3 hours prior to admission and resolved spontaneously in the ER. Obs and examination are normal. 12-lead ECG shows no abnormality. The first TnT is 4 ng/L and repeat at 1 hour is 10ng/L. (Architect assay). How should you proceed?

A – NSTEMI ruled out – discharge with outpatient tests
B – Repeat a further TNT at 3 hours
C – Inpatient invasive angiography
D – ‘Rule out’ inpatient CTCA
E – Exercise treadmill test and discharge if normal
A 60 year old female with hypertension and statin therapy presents with moderate left thoracic chest pain without radiation that started 3 hours prior to admission and resolved spontaneously in the ER. Obs and examination are normal. 12-lead ECG shows no abnormality. The first TnT is 4 ng/L and repeat at 1 hour is 10ng/L. (Architect assay). How should you proceed?

A – NSTEMI ruled out – discharge with outpatient tests
B – Repeat a further TNT at 3 hours
C – Inpatient invasive angiography
D – ‘Rule out’ inpatient CTCA
E – Exercise treadmill test and discharge if normal
A 65 year old man with hypertension and statin treatment presents with moderate chest pain that started 3 hours prior to admission and resolved spontaneously in the ER. Obs and examination are normal. 12-lead ECG shows no abnormality. The first hs-c TnT is 120ng/L (Elecsys assay). How should you proceed?

A – Repeat a further TNT at 1 hours prior to ‘ruling in’ NSTEMI
B – I/P angio
C – I/P Non-invasive imaging stress test
D – Repeat a further TNT at 3 hours prior to ‘ruling in’ NSTEMI
E – I/P CTCA
0h/ 3h hs-cTn algorithm

Rule out = if negative after 6 hours from pain or 2 x sequential tests separated by 3 hours (but now includes GRACE < 140)

Rule in = highly abnormal (x5 ULN – 70ng/L) or significant change in 3hr

Grey area = < 70ng/L with no change after 3 hours – non-invasive
A 65-year-old hypertensive woman is admitted with suspected NSTE-ACS based on 2 hours of chest discomfort, non-specific ECG changes and mildly elevated high-sensitivity troponin [47 ng/L (ULN <14 ng/L) – 49 ng/L repeated 3 hours later]. She is on VKA for atrial fibrillation and the INR is 2.8. The CHA2DS2-VASc score is 3. Her ventricular rate is poorly controlled (140/min) in the absence of signs of heart failure. Kidney function is normal. Echocardiography shows normal LV systolic function, no regional wall motion abnormalities and moderate LV hypertrophy. What is the optimal next ischaemia test once AF controlled?

A – Exercise treadmill
B – CTCA
C – non-invasive imaging test
D – Invasive angiogram
E – Repeat TNT 6 hours post onset of pain
Straight from the ESC

• The patient should first undergo non-invasive testing. Accordingly, the elevation in enzymes could be solely related to tachycardia and LV hypertrophy (i.e. type 2 MI)

• CT angiography is only helpful to exclude relevant CAD in patients presenting with chest pain to the ER who are haemodynamically stable patients without known CAD, a normal ECG and a normal initial standard cardiac troponin level in the presence of a slow and regular heart rate.
40-year-old patient without known CAD and without CV risk factors presents to the ER with 10 minutes of acute and severe cardiac-sounding chest pain suggestive of ACS. Hs-c TnT at 0 and 3 hours are < 14ng/L and there are no ischaemic ECG changes. CXR and D-dimer are also normal. He has noticed similar milder chest over the last week, sometimes with exertion. What is your plan?

A – NSTEMI ruled out - discharge
B – Angio
C – Repeat TNT at 6 hours post pain
D – Exercise treadmill
E – CTCA
Straight from the ESC

- NSTE-MI ruled out. Unstable angina possible but low risk
- Coronary CT angiography should be considered to exclude CAD in patients with acute chest pain suggestive of NSTE-ACS, negative troponins and no ischaemic ECG changes
Rule in vs rule out
1. Rule in NSTEMI = angio

2. Rule out NSTEMI, history not suggestive of unstable angina, low GRACE – discharge

3. Rule out NSTEMI but hx suggestive of ACS: intermediate risk criteria = non-invasive imaging test Low-risk: CTCA if no hx IHD, good heart rate

In patients with no recurrence of chest pain, normal ECG findings and normal levels of cardiac troponin (preferably high-sensitivity), but suspected ACS, a non-invasive stress test (preferably with imaging) for inducible ischaemia is recommended before deciding on an invasive strategy.
A 60-year-old diabetic patient presents at night with central chest pain. Initial hs-c TnI is 200ng/L and ECG shows deep T wave inversion in the anterior chest leads. He is pain free. What medications should be started?

A – Aspirin 300 & ticagrelor 180
B – Aspirin 300 & prasugrel 90
C – Aspirin 300 & clopidogrel 600
D – Aspirin 300, clopidogrel 600mg and tirofiban
E – Aspirin 300, ticagrelor 180 or prasugrel 90 and tirofiban
A 50-year-old man with no prior relevant medical history, active smoker, seeks medical attention for recurrent tearing chest pain episodes, the last one occurring in the last 24 h and lasting a few seconds. He has no symptoms at exercise and takes no medication. ECG shows 0.5 mm ST depression in the lateral leads and is unchanged 1 h later. The first high-sensitivity cardiac troponin is within the normal range. What is your next action?

A – Aspirin 75mg and clopidogrel 300mg  
B – Aspirin 300mg and clopidogrel 600mg  
C – Aspirin 300mg and ticagrelor 180mg  
D – CTCA  
E – Repeat troponin after observation
A 67-year-old diabetic man with suspected non- ST-elevation ACS (NSTE-ACS) is referred to you. His chest pain characteristics are typical and T wave inversions in the anterior leads are detected. He has no co-morbidities. The first high-sensitivity troponin is 100 ng/L [upper limit of normal (ULN) <14 ng/L]. The patient has a prior history of aspirin allergy.

A – Medical management with ticagrelor alone
B – Medical management with prasugrel alone
C – Aspirin desensitization and then delayed angiography
D – Aspirin desensitization and then early angiography
E – Aspirin administration and urgent angiography
Aspirin Allergy

• Aspirin Allergy
  • Confirm it
  • True oedema / anaphylaxis

• Aspirin Desensitization
  • Inpatient
  • Aspirin 5mg, 10mg, 20mg, 40mg every 30 minutes
  • Then aspirin 75mg

• If STEMI
  • Ticagrelor with GPI, go to lab
  • Aspirin desensitisation after PCI
A 60-year-old diabetic patient with NSTEMI and LAD syndrome on ECG is transferred to CCU. They develop transient recurrent chest pain despite iv nitrate infusion and heart rate control with beta-blocker. They have been pre-treated with aspirin, ticagrelor and fondaparinux. ECG has remained the same with deep T wave inversion V1 – V4. Angiography is scheduled in the morning. How do you proceed?

A – Add tirofiban to the regime
B – Call the PPCI team for primary angioplasty
C – Switch ticagrelor to prasugrel
D – Arrange for angiography within 2 hours
E – Continue to observe closely on CCU unless dynamic ECG change or ongoing pain
### Table 13 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 insufficiency (eGFR <60 mL/min/1.73 m²)
- 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

**Immediate Invasive (<2 hr)**
- Unstable haemodynamics (shock), rhythm or ongoing ischaemia

**Early invasive (<24 hr)**
- NSTEMI

**Invasive (<72 hr)**
- Everyone else
Q11. A 56-year-old troponin-positive patient with no ST elevation on ECG is transferred from a peripheral hospital to your institution for coronary angiography. His prior medical history is unremarkable. He received aspirin and clopidogrel, given at a loading dose of 600 mg followed by 75 mg/day. The diagnostic coronary angiogram shows diffuse CAD but no critical lesion in proximal vessel segments and a conservative strategy is chosen. What should the patient be given on discharge?

A – No stent or acute lesion. Continue aspirin only long-term
B – Aspirin 75mg and clopidogrel 75mg 12 months
C – Aspirin 75mg and switch to ticagrelor (with an initial loading dose) for 12/12
D – Aspirin 75mg and prasugrel or ticagrelor based on the institutional preference for 12/12
E – Aspirin 75mg, clopidogrel 75mg 12/12 and rivaroxaban▼2.5mg bd 12/12
ESC 2015 Guidelines

• Ticagrelor preferred P2Y1₂ inhibitor (load patients pre-treated with Clopidogrel)

• Prasugrel only post-angio when no prior P2Y1₂
  • (no upstream treatment indication).
  • Contraindicated if
    • > 75 years
    • < 60kg
    • TIA or previous intracerebral haemorrhage

• GPIIbIIIa only for bailout during PCI (no upstream indication to delay PCI)
Longer vs Shorter

• **Shorter**
  - Significant reduction in bleeding (OR 0.58 0.36-0.92, p=0.02)
  - Equivalent number of ischaemia events

• **Longer**
  - *Reduced* MI & ST
  - More major bleeds
  - All cause-deaths increased, but not CVS deaths

• **Stent type?**
  - Patients with Everolimus-eluting and fast-release zotarolimus-eluting stents can have ~6 months DAPT with less major bleeding with similar survival and ST events, at the cost of increased MIs.
  - Biolimus eluting BioFreedom (LEADERS-FREE): non-inferior to BMS in high bleeding risk patients.
<table>
<thead>
<tr>
<th>Patient-related factors</th>
<th>≤12 months DAPT</th>
<th>≥12 months DAPT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with stable CAD</td>
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<tr>
<td>Patients with a history of bleeding</td>
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<tr>
<td>Patients with high risk of bleeding</td>
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<tr>
<td>Patients with ACS</td>
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<td>Patients with diabetes mellitus</td>
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<td>Patients with renal dysfunction</td>
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<td>Patients with CHF</td>
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<tr>
<td>Patients with previous ST</td>
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<td>Patients with PAD</td>
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</tbody>
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<thead>
<tr>
<th>Anatomy-related factors</th>
<th>≤12 months DAPT</th>
<th>≥12 months DAPT</th>
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</thead>
<tbody>
<tr>
<td>Short lesion</td>
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<tr>
<td>Single-vessel disease</td>
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<tr>
<td>Long lesion</td>
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<tr>
<td>Small vessel</td>
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<tr>
<td>Bifurcation lesion</td>
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<tr>
<td>Complex anatomy</td>
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<tr>
<td>Left-main coronary artery</td>
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</tbody>
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<thead>
<tr>
<th>Stent-related factors</th>
<th>≤12 months DAPT</th>
<th>≥12 months DAPT</th>
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<tbody>
<tr>
<td>Second-generation DES</td>
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<tr>
<td>First-generation DES</td>
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<tr>
<td>Long stent</td>
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<tr>
<td>Multiple stents</td>
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Early interruption

• Early interruption of DAPT – increased ST
  • Greatest risk within first 1 month of stopping
  • Minimum of 1 month for BMS & 3 months for new-gen DES

• If need to stop for high risk surgery (eg. neurosurgery)
  • 5 – 7 days for clopidogrel
  • 7 – 10 days for ticagrelor

  • restart as soon as possible
  • Consider bridging with GPIIb/IIIa or cangrelor (high risk cases)
  • Heparin DOES NOT provide sufficient cover

• If had no stent placed, then just stop P2Y1₂⁻inhibitor
A 75-year-old patient with high-risk NSTEMI is pretreated with ticagrelor in addition to aspirin and fondaparinux. ECG shows no ST deviation and no Q waves. Coronary angiography shows advanced three-vessel CAD including a critical left main stenosis, with a high SYNTAX score (34). Heart Team decision is to perform CABG. The patient is stable, has been free of chest pain for 48 hours and troponin is falling. The LV ejection fraction is 40% in the presence of diffuse hypokinesia. How do you manage perioperative antithrombotic therapy and the timing of CABG?

A – Continue DAPT, fondaparinux & operate now
B – Stop DAPT, continue fondaparinux and operate now
C – Stop ticagrelor, continue aspirin and fondaparinux and operate in 5 days
D – Stop ticagrelor, continue aspirin and fondaparinux and operate in 7 days
E – Risk of surgery high in the first 2/52 – schedule after “cooling period”. Stop ticagrelor and aspirin 5 days prior to surgery
P2Y1<sub>12</sub> pre CABG

Associated with 2 x bleeding risk

- Ticagrelor 5 days
- Clopidogrel 5 days
- Prasugrel 7 days
A 68-year-old patient is admitted 12 hours after an episode of severe chest pain. Angiography demonstrates subtotal occlusion of the proximal left anterior descending artery which underwent PCI with 3.5x28 and 3.0x15 DES. Pre-discharge echocardiography demonstrates moderate LV impairment with akinesia of the anterior wall from the mid to apical segments. Contrast echocardiography confirms the presence of apical thrombus. How would you proceed?

A – Aspirin 75mg od, ticagrelor 90mg bd for 1 year
B – Aspirin 75mg od, clopidogrel 75mg od, Warfarin as per INR for 1 year
C – Aspirin 75mg od, ticagrelor 60mg bd for 1 year with warfarin as per INR for 3 months
D – Aspirin 75mg od, clopidogrel 75mg od for 1 year with warfarin as per INR for 3 months followed by repeat echocardiography
E – Aspirin 75mg od, clopidogrel 75mg od for 1 year with warfarin as per INR for 9 months followed by repeat echocardiography
Ventricular Thrombus in STEMI

• ¼ of Anterior STEMI associated with mural thrombus
• Poor prognosis with high stroke rate

• Prior to DAPT & Stenting era: 6 months VKA

• If stent & DAPT: recommend 3 months & repeat Echo
  • If still present, then continue for 6 months
  • Little data: consensus opinion

A 50-year-old patient with unstable angina underwent stenting with a DES (everolimus eluting) of the proximal left circumflex coronary artery last week. He has chronic anaemia due to ulcerative colitis but no additional co-morbidities. The Gastroenterologists write to you regarding his DAPT.

A – Must continue DAPT for 1 year
B – Must continue DAPT for 6 months
C – Must continue DAPT for 3 months
D – Must continue DAPT for 1 month
E – Must continue DAPT for 1 year with PPI cover
A 58-year-old patient is admitted with on-going chest pain and ST elevation. Another patient is currently undergoing emergency PCI and there is no capacity to perform immediate PCI.

A – Transfer to the nearest hospital (2 hours away)
B – Thrombolysis if the delay will exceed 3 hours
C – Thrombolysis if the delay will exceed 60 minutes
D – Aspirin, ticagrelor, tirofiban and await PCI
E – Aspirin, cangrelor▼, heparin and await PCI
### Important delays and treatment goals in the management of acute STEMI

<table>
<thead>
<tr>
<th>Delays</th>
<th>Target</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preferred for FMC to ECG and diagnosis.</td>
<td>≤ 10 min</td>
</tr>
<tr>
<td>Preferred for FMC to fibrinolysis (‘FMC to needle’).</td>
<td>≤ 30 min</td>
</tr>
<tr>
<td>Preferred for FMC to primary PCI (‘door to balloon’) in primary PCI hospitals.</td>
<td>≤ 60 min</td>
</tr>
<tr>
<td>Preferred for FMC to primary PCI.</td>
<td>≤ 90 min&lt;br&gt;(≤ 60 min if early presenter with large area at risk) if this target cannot be met, consider fibrinolysis.</td>
</tr>
<tr>
<td>Acceptable for primary PCI rather than fibrinolysis.</td>
<td>≤ 120 min&lt;br&gt;(≤ 90 min if early presenter with large area at risk) if this target cannot be met, consider fibrinolysis.</td>
</tr>
<tr>
<td>Preferred for successful fibrinolysis to angiography.</td>
<td>3-24 h</td>
</tr>
</tbody>
</table>

FMC = first medical contacts; PCI = percutaneous coronary intervention.
Primary PCI Timings

• Must reperfuse within 12 hours symptoms

• Primary PCI if within 2 hours of First Medical contact
  • Ideally within 90 minutes

• Fibrinolysis if < 2hours but no PCI not available within 90 minutes
  • Transfer to a PCI centre
  • Rescue PCI if failed: <50% ST segments resolution at 1 hour
  • Emergency PCI if CCF or shock after thrombolysis

• Opening the vessel to restore flow can improve LV function, providing it remains viable
A 69-year-old farmer presents to the Emergency Department 24hrs after the onset of chest pain. He says that the pain was central and crushing and lasted in total for nearly 10hrs. He has a history of hypertension for which he takes atenolol and indapamide. On examination his BP is 80/40 mmHg, his pulse is 65/min, his heart sounds are normal and there are no murmurs. He has no significant pulmonary oedema on auscultation of his chest. ECG reveals ST elevation in V1 and deep Q-waves in III and AVF. Hand-held echocardiography demonstrates RV dilatation. Troponin is elevated 20,000 ng/L.

**Which therapy should be started immediately?**

A – Normal saline  
B – Thrombolysis with rTPA  
C – Intra-aortic balloon pump with heparin cover  
D – Aspirin 300mg and ticagrelor 180mg  
E – Aspirin 300mg, ticagrelor 180mg and heparin 5000 IU
Delayed Presentation

- Presenting after 12 hours
- Likely that the infarction has completed
- Q waves consistent with full thickness infarct

- General consensus: if patients have signs (ECG or clinical) of ongoing ischaemia then try PPCI
  - Occlusion can be dynamic so worth a try
  - If no such features, will need more elective angiography once stabilised

RV Infarction

- Inferior ECG ST elevation with triad:
  - Hypotension
  - Clear lung fields
  - Raised JVP: Swan Ganz shows RAP >10mmHg

- ECG changes
  - V4R ST elevation strongly suggestive
  - V1-V3 ST elevation with Q-waves

- Management is opposite to Shock from LV failure
  - MAINTAIN RV PRELOAD
    - Avoid opiates, nitrates, diuretics, ACE-I
    - Give rapid IV fluid bolus urgently - with careful monitoring
    - AF and CHB common – correct quickly else decompensate
    - PPCI asap
    - Fibrinolysis may be less effective (unclear)
A 65-year-old woman is admitted for NSTEMI with a markedly elevated high-sensitivity cardiac troponin level [350 ng/L (ULN <14 ng/L)] and an invasive strategy is planned. She is on vitamin K antagonist (VKA) for atrial fibrillation and the INR is 2.7. The CHA₂DS₂VASc score is 3 and radial access seems feasible. What is the appropriate next steps.

A – Stop warfarin and aim for angio when the INR < 2.0
B – Bridge – switch warfarin for LMWH and aim for angio when the INR < 2.0
C – Bridge - switch warfarin for fondaparinux and aim for angio when the INR < 2.0
D – Perform angiography via radial route on VKA and avoid bridging. No additional heparin is required during PCI
E – Bridge - switch warfarin for iv heparin continued through procedure and aim for angio when the INR < 2.0
Q16: A patient on aspirin and prasugrel following proximal LAD PCI with new generation DES for NSTE-ACS (1/12 ago) develops atrial fibrillation lasting >24 h. The CHA$_2$DS$_2$VASc score is 3 and HAS-BLED 2. What is your immediate antithrombotic treatment?

A – Switch prasugrel to clopidogrel, Start anticoag and continue aspirin (triple therapy)
B – Continue aspirin and prasugrel only
C – Continue on prasugrel only with anticoag (stop aspirin)
D – Continue aspirin only and anticoagulate (stop prasugrel)
E – Prasugrel, aspirin and anticoag (Triple therapy)
A 64 year old man has NSTEMI has 4.0x18 DES in the proximal LAD. He has pAF and is on rivaroxaban 20mg od but has never had embolic events. LV function is normal and there is no valvular disease. His current CHA₂DS₂VASc is 1. HAS-BLED is 2.0 He is currently on aspirin and clopidogrel

A – Give aspirin & clopidogrel alone for 1 year
B – Give aspirin & ticagrelor alone for 1 year then restart NOAC
C – Give aspirin & ticagrelor & NOAC for 6 months, then clopidogrel and NOAC for life
D – Give aspirin & ticagrelor & NOAC for 6 months, then aspirin and NOAC for life
E – Give aspirin & clopidogrel & NOAC for 6 months, then clopidogrel and NOAC to 1 year, then NOAC alone
You admit a high risk patient with NSTEMI and dynamic ST change who ideally requires immediate (< 2 hours) angiography. The have chronic AF (CHA₂DS₂VASc 3, HASBLED 3) and are treated with apixaban 5mg bd which was last taken 4 hours ago. In view of the anticoagulation what is your strategy?

A – Defer the angiogram for 12 hours post the last dose of apixaban. No additional immediate anticoagulation is required.
B – Stop apixaban with a view to switching to warfarin & DAPT post acute treatment. No additional immediate anticoagulation is required.
C – Defer the angiogram for 12 hours post the last dose of apixaban. Bridge the patient with iv heparin aiming for an ACT 250 – 300 until acute treatment is complete
D – Continue with immediate angiography and no further anticoagulation is required if PCI is required
E – Continue with immediate angiography and give additional Heparin if proceeding to PCI
2015 ESC Guidelines for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation