

## Criteria for acute myocardial infarction

The term myocardial infarction is defined as the presence of myocardial necrosis in a clinical setting consistent with myocardial ischaemia. Under these conditions any one of the following criteria meets the diagnosis for myocardial infarction:

- Detection of rise and/or fall of cardiac biomarkers (preferably troponin) with at least one value above the 99th percentile of the upper reference limit (URL) together with evidence of myocardial ischaemia with at least one of the following:
  - Symptoms of ischaemia;
  - ECG changes indicative of new ischaemia [new ST-T changes or new left bundle branch block (LBBB)];
  - Development of pathological Q waves in the ECG;
  - Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
- Sudden, unexpected cardiac death, involving cardiac arrest, often with symptoms suggestive of myocardial ischaemia, and accompanied by presumably new ST elevation, or new LBBB, and/or evidence of fresh thrombus by coronary angiography and/or at autopsy, but death occurring before blood samples could be obtained, or at a time before the appearance of cardiac biomarkers in the blood.
- For percutaneous coronary interventions (PCI) in patients with normal baseline troponin values, elevations of cardiac biomarkers above the 99th percentile URL are indicative of peri-procedural myocardial necrosis. By convention, increases of biomarkers greater than  $3 \times$  99th percentile URL have been designated as defining PCI-related myocardial infarction. A subtype related to a documented stent thrombosis is recognized.
- For coronary artery bypass grafting (CABG) in patients with normal baseline troponin values, elevations of cardiac biomarkers above the 99th percentile URL are indicative of peri-procedural myocardial necrosis. By convention, increases of biomarkers greater than  $5 \times$  99th percentile URL plus either new pathological Q waves or new LBBB, or angiographically documented new graft or native coronary artery occlusion, or imaging evidence of new loss of viable myocardium have been designated as defining CABG-related myocardial infarction.
- Pathological findings of an acute myocardial infarction.

## Criteria for prior myocardial infarction

Any one of the following criteria meets the diagnosis for prior myocardial infarction:

- Development of new pathological Q waves with or without symptoms.
- Imaging evidence of a region of loss of viable myocardium that is thinned and fails to contract, in the absence of a non-ischaemic cause.
- Pathological findings of a healed or healing myocardial infarction.

**Table 1** Clinical classification of different types of myocardial infarction

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**Type 1**

Spontaneous myocardial infarction related to ischaemia due to a primary coronary event such as plaque erosion and/or rupture, fissuring, or dissection

**Type 2**

Myocardial infarction secondary to ischaemia due to either increased oxygen demand or decreased supply, e.g. coronary artery spasm, coronary embolism, anaemia, arrhythmias, hypertension, or hypotension

**Type 3**

Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of myocardial ischaemia, accompanied by presumably new ST elevation, or new LBBB, or evidence of fresh thrombus in a coronary artery by angiography and/or at autopsy, but death occurring before blood samples could be obtained, or at a time before the appearance of cardiac biomarkers in the blood

**Type 4a**

Myocardial infarction associated with PCI

**Type 4b**

Myocardial infarction associated with stent thrombosis as documented by angiography or at autopsy

**Type 5**

Myocardial infarction associated with CABG

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**Table 3** ECG manifestations of acute myocardial ischaemia (in absence of LVH and LBBB)

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### ST elevation

New ST elevation at the J-point in two contiguous leads with the cut-off points:  $\geq 0.2$  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

### ST depression and T-wave changes

New horizontal or down-sloping ST depression  $\geq 0.05$  mV in two contiguous leads; and/or T inversion  $\geq 0.1$  mV in two contiguous leads with prominent R-wave or R/S ratio  $> 1$

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**Table 4** ECG changes associated with prior myocardial infarction

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Any Q-wave in leads  $V_2$ - $V_3$   $\geq 0.02$  s or QS complex in leads  $V_2$  and  $V_3$

Q-wave  $\geq 0.03$  s and  $\geq 0.1$  mV deep or QS complex in leads I, II, aVL, aVF, or  $V_4$ - $V_6$  in any two leads of a contiguous lead grouping (I, aVL,  $V_6$ ;  $V_4$ - $V_6$ ; II, III, and aVF)<sup>a</sup>

R-wave  $\geq 0.04$  s in  $V_1$ - $V_2$  and R/S  $\geq 1$  with a concordant positive T-wave in the absence of a conduction defect

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<sup>a</sup>The same criteria are used for supplemental leads  $V_7$ - $V_9$ , and for the Cabrera frontal plane lead grouping.

**Table 5** Common ECG pitfalls in diagnosing myocardial infarction

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**False positives**

Benign early repolarization

LBBB

Pre-excitation

Brugada syndrome

Peri-/myocarditis

Pulmonary embolism

Subarachnoid haemorrhage

Metabolic disturbances such as hyperkalaemia

Failure to recognize normal limits for J-point displacement

Lead transposition or use of modified Mason-Likar configuration<sup>24</sup>

Cholecystitis

**False negatives**

Prior myocardial infarction with Q-waves and/or persistent ST elevation

Paced rhythm

LBBB

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