INTRODUCTION — Myocardial infarction (MI) is defined as a clinical (or pathologic) event caused by myocardial ischemia in which there is evidence of myocardial injury or necrosis \[1,2\]. Criteria are met when there is a rise and/or fall of cardiac biomarkers, along with supportive evidence in the form of typical symptoms, suggestive electrocardiographic (ECG) changes, or imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

Initial care of the patient with suspected acute MI should include the early and simultaneous achievement of four goals:

- Confirmation of the diagnosis by ECG and biomarker measurement
- Relief of ischemic pain
- Assessment of the hemodynamic state and correction of abnormalities that may be present
- Initiation of antithrombotic and reperfusion therapy if indicated

The management of patients with an ST segment elevation (Q wave) MI or non-ST segment elevation acute coronary syndrome (ACS) is discussed elsewhere. (See "Overview of the acute management of ST-elevation myocardial infarction" and "Overview of the acute management of non-ST elevation acute coronary syndromes".)

A related issue is the evaluation of a patient who presents with chest pain suggestive of an ACS in whom the initial evaluation (ECG, cardiac enzymes) is not diagnostic. This issue is discussed separately. (See "Initial evaluation and management of suspected
Acute coronary syndrome — The term acute coronary syndrome (ACS) is applied to patients in whom there is a suspicion of myocardial ischemia. There are three types of ACS: ST elevation (formerly Q-wave) MI (STEMI), non-ST elevation (formerly non-Q wave) MI (NSTEMI), and unstable angina (UA). The first two are characterized by a typical rise and/or fall in biomarkers of myocyte injury.

Two multicenter, international surveys published in 2002, the Euro Heart Survey and the GRACE registry, determined the relative frequency of these disorders in approximately 22,000 patients admitted with an ACS [3,4]. STEMI occurred in 30 to 33 percent, NSTEMI in 25 percent, and UA in 38 to 42 percent.

Acute MI — For many years, the diagnosis of acute MI relied on the revised criteria established by the World Health Organization in 1979 [5]. These criteria were epidemiological and aimed at specificity. A joint European Society of Cardiology (ESC) and American College of Cardiology committee proposed a more clinically based definition of an acute, evolving, or recent MI in 2000 [1]. In 2007, the Joint Task Force of the European Society of Cardiology, American College of Cardiology Foundation, the American Heart Association, and the World Heart Federation (ESC/ACCF/AHA/WHF) refined the 2000 criteria and defined acute MI as a clinical event consequent to the death of cardiac myocytes (myocardial necrosis) that is caused by ischemia (as opposed to other etiologies such as myocarditis or trauma) [2]. This definition was not fundamentally changed in the third universal definition of MI released in 2012 by the ESC/ACCF/AHA/WHF [6].

According to the third universal definition, any one of the following criteria meets the diagnosis of MI:

- Detection of a rise and/or fall of cardiac biomarker values (preferably cardiac troponin [cTn] with at least one value above the 99th percentile upper reference limit [URL]) and with at least one of the following:
  - Symptoms of ischemia
  - Development of pathologic Q waves in the electrocardiogram (ECG)
  - New or presumed new significant ST-segment-T wave (ST-T) changes or new left bundle branch block (LBBB).
  - Identification of an intracoronary thrombus by angiography or autopsy
  - Imaging evidence of new loss of viable myocardium or a new regional wall motion abnormality.
Cardiac death with symptoms suggestive of myocardial ischemia and presumed new ischemia ECG changes or new LBBB, but death occurred before cardiac biomarkers were obtained, or before cardiac biomarker values would be increased.

Percutaneous coronary intervention (PCI)-related MI was defined by elevation of biomarker values (cTn is preferred) >5 \times 99^{\text{th}}\text{ percentile URL} in patients with normal baseline values (<99^{\text{th}}\text{ percentile URL}) or a rise of values >20 percent if the baseline values are elevated but stable or falling. In addition, either (i) symptoms suggestive of myocardial ischemia, (ii) new ischemic ECG changes or new LBBB, (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.

Stent thrombosis associated with MI when detected by coronary angiography or autopsy in the setting of myocardial ischemia and with a rise and/or fall of cardiac biomarkers with at least one value above the 99^{\text{th}}\text{ percentile}.

Coronary artery bypass graft surgery (CABG)-associated MI was defined by elevation of cardiac biomarker values >10 \times 99^{\text{th}}\text{ percentile URL} in patients with normal baseline cTn values. In addition, either (i) new pathologic Q waves or new LBBB, (ii) angiographic documented new graft or native coronary artery occlusion, or (iii) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

The joint task force further refined the definition of MI by developing a clinical classification according to the assumed proximate cause of the myocardial ischemia:

- **Type 1 (spontaneous MI):** MI consequent to a pathologic process in the wall of the coronary artery (eg, plaque erosion/rupture, fissuring, or dissection), resulting in intraluminal thrombus.

- **Type 2 (MI secondary to an ischemic imbalance):** MI consequent to increased oxygen demand or decreased supply (eg, coronary endothelial dysfunction, coronary artery spasm, coronary artery embolus, tachy-/brady-arrhythmias, anemia, respiratory failure, hypertension, or hypotension).

- **Type 3 (MI resulting in death when biomarker values are unavailable):** Sudden unexpected cardiac death before blood samples for biomarkers could be drawn or before their appearance in the blood.

- **Type 4a (MI related to PCI):** See criteria directly above

- **Type 4b (MI related to stent thrombosis):** See criteria directly above

- **Type 5 (MI related to CABG):** See criteria directly above

**Unstable angina** — UA is considered to be present in patients with ischemic symptoms suggestive of an ACS without elevation in biomarkers with or without ECG changes indicative of ischemia. Due to the insensitivity of creatine kinase MB fraction (CK-MB)
compared to troponin, the finding of a normal CK-MB does not entirely exclude the diagnosis of MI using the current definition. Elevations of troponin with contemporary assays probably take two to three hours, while elevations for CK-MB take longer. (See 'Cardiac biomarkers' below.)

UA and NSTEMI are frequently indistinguishable at initial evaluation. ST segment and/or T wave changes are often persistent in NSTEMI, while, if they occur in UA, they are usually transient. Regardless, of the category, ST segment change defines a higher-risk group [7].

After revascularization — The definitions of MI in the setting of PCI or CABG are presented above. (See 'Acute MI' above.)

Following revascularization with either CABG or PCI, cardiac biomarkers may rise. Transient elevations in troponins may represent necrosis, although the mechanism is unknown. Higher elevations are associated with worse prognosis after CABG. A discussion of MI following PCI is found elsewhere. (See "Periprocedural myocardial infarction following percutaneous coronary intervention".)

The Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction addressed the issue of biomarker rise after revascularization procedures (Type 4a) [6]. For patients undergoing PCI, a normal baseline troponin value (≥99th percentile URL) is mandatory in order to use the definition above. If the baseline value is elevated or rising, it is impossible to tell if elevations post-procedure are related to the initial insult or to additional injury. However, they also state that if the troponin values are stable or falling before revascularization, the use of the reinfarction criteria may be reasonable. We also believe it is reasonable to obtain a troponin prior to (within six hours of) PCI (a baseline value). (See 'Recommended approach' below.)

No criteria have been established to separate expected rises (such as needle trauma to the myocardium at CABG) from those that represent a complication of the procedure (such as unexpected coronary artery dissection due to wire trauma at PCI) [8]. In addition, the cut points given below are controversial.

SCAI definition — Not all cardiovascular societies or organizations have adopted the Universal Definition of MI presented above and this issue remains contentious. For example, in 2013, the Society for Cardiovascular Angiography and Interventions proposed the following definition for "clinically relevant MI" after coronary revascularization [9]. For patients undergoing PCI or CABG who have a normal baseline CK-MB, MI was defined as that resulting in CK-MB ≥10 X the upper limit of normal (ULN), or a lower threshold of ≥5 X the ULN in patients in whom new pathologic Q-waves in ≥2 contiguous ECG leads (or new persistent LBBB) develop after PCI. Separate criteria were given for individuals without a normal value for CK-MB at baseline.

Although we and others agree that there are likely to be changes to the Universal Definition based on a better understanding of the relationship between elevated postprocedural troponin values and the subsequent risk of clinical events, we recommend use of the Universal Definition [10].
Prior MI

According to the third universal definition of MI, any one of the following three criteria satisfies the diagnosis for a prior (established) MI [9]:

- Pathologic Q waves (≥0.04 sec) with or without symptoms in the absence of non-ischemic causes
- Pathologic findings of a healed or healing MI
- Evidence from an imaging study of a region of loss of viable myocardium that is thinned and fails to contract in the absence of a nonischemic cause. (See "Role of echocardiography in acute myocardial infarction", section on "Indications for echocardiography in MI").

INITIAL EVALUATION — For a patient presenting with a suspected acute myocardial infarction (MI), the characteristics of the chest pain and the electrocardiogram (ECG) findings permit initial risk stratification. An ECG and an abbreviated history and physical examination should be obtained within 10 minutes of patient arrival [11]. Other steps in the immediate management of patients suspected of an acute MI are discussed separately (see "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department", section on "Immediate ED interventions").

The history should be targeted toward pain duration, character, similarity to possible previous episodes, provoking factors, and past history of coronary disease risk factors. The physical examination (including auscultation of the heart and lungs, measurement of blood pressure in both arms, and assessment for heart failure or circulatory compromise, which are associated with a high early mortality).

Patients with a strong clinical history and ST segment elevation or new left bundle branch block should be assumed to have an acute MI and undergo immediate reperfusion therapy. (See "Acute ST elevation myocardial infarction: Selecting a reperfusion strategy").

"Ruling in" an acute MI with newer troponin assays occurs in 80 percent of patients by two to three hours after presentation; "ruling out" may take longer (up to six hours) (see 'Cardiac biomarkers' below).

CHEST PAIN — While chest pain is not required for the diagnosis of myocardial infarction (MI), its presence, particularly if characteristic for myocardial ischemia, may influence decision making about the likelihood of the presence of MI (table 1). A discussion of the characteristic of ischemic chest pain is found elsewhere. (See "Outpatient evaluation of the adult with chest pain").

Present — Among patients with chest pain characteristic of myocardial ischemia (angina pectoris), there are three primary presentations that suggest a change in the anginal pattern as acute coronary syndrome as opposed to stable or exertional angina:
- Rest angina, which is usually more than 20 minutes in duration
- New onset angina that markedly limits physical activity
- Angina that is more frequent, longer in duration, or occurs with less exertion than previous angina

**Absent** — Patients without features of typical angina are more likely to have another cause of chest pain. Common causes include other cardiovascular, pulmonary, and gastrointestinal disorders ([table 2](#)). (See "Outpatient evaluation of the adult with chest pain", section on 'Etiologies'.)

In a review of over 430,000 patients with confirmed acute MI from the National Registry of Myocardial Infarction 2, one-third had no chest pain on presentation to the hospital [12]. These patients may present with dyspnea alone, nausea and/or vomiting, palpitations, syncope, or cardiac arrest. They are more likely to be older, diabetic, and women. (See "Clinical features and diagnosis of coronary heart disease in women".)

The absence of chest pain has important implications for therapy and prognosis. In the Registry report, patients without chest pain were much less likely to be diagnosed with a confirmed MI on admission (22 versus 50 percent in those with chest pain) and were less likely to be treated with appropriate medical therapy and to receive fibrinolytic therapy or primary angioplasty (25 versus 74 percent) [12]. Not surprisingly, these differences were associated with an increase in in-hospital mortality (23.3 versus 9.3 percent, adjusted odds ratio 2.21, 95 percent confidence interval 2.17 to 2.26).

**ECG** — The electrocardiogram (ECG) is a mainstay in the initial diagnosis of patients with suspected acute coronary syndrome (ACS). It allows initial categorization of the patient with a suspected myocardial infarction (MI) into one of three groups based on the pattern:

- ST elevation MI (STEMI; ST elevation or new left bundle branch block [LBBB])

- non-ST elevation ACS, with either non-ST elevation MI (NSTEMI) or unstable angina (UA; ST-depression, T wave inversions, or transient ST-elevation)

- Undifferentiated chest pain syndrome (nondiagnostic ECG)

**ST elevation MI** — In patients with acute STEMI, the ECG evolves through a typical sequence. (See "Electrocardiogram in the diagnosis of myocardial ischemia and infarction" and "ECG tutorial: Myocardial ischemia and infarction".)

Although not frequently seen, the earliest change in an STEMI is the development of a hyperacute or peaked T wave that reflects localized hyperkalemia. Thereafter, the ST segment elevates in the leads recording electrical activity of the involved region of the myocardium; it has the following appearance:

- Initially, there is elevation of the J point and the ST segment retains its concave configuration
Over time, the ST segment elevation becomes more pronounced and the ST segment becomes more convex or rounded upward.

The ST segment may eventually become indistinguishable from the T wave; the QRS-T complex can actually resemble a monophasic action potential.

The joint European Society of Cardiology, American College of Cardiology Foundation, the American Heart Association, and the World Heart Federation (ESC/ACCF/AHA/WHF) committee for the definition of MI established specific ECG criteria for the diagnosis of ST elevation MI [6,13]:

- New ST segment elevation at the J point in two contiguous leads with the cut-points: >0.1 mV in all leads other than leads V2-V3.
- For leads V2-V3, the following cut points apply: ≥0.2 mV in men ≥40 years, ≥0.25 mV in men <40 years, or ≥0.15 mV in women.

Over time, there is further evolution of these ECG changes; the ST segment gradually returns to the isoelectric baseline, the R wave amplitude becomes markedly reduced, and the Q wave deepens. In addition, the T wave becomes inverted. These changes generally occur within the first two weeks after the event, but may progress more rapidly, within several hours of presentation.

In addition to patients with ST elevation on the ECG, two other groups of patients with an ACS are considered to have an STEMI: those with new or presumably new LBBB and those with a true posterior MI. (See 'Bundle branch block or paced rhythm' below and "Electrocardiogram in the diagnosis of myocardial ischemia and infarction", section on 'Posterior wall MI'.)

A separate issue, the assessment of patients with a suspected acute MI who have known LBBB or a paced rhythm, is discussed below. (See 'Bundle branch block or paced rhythm' below.)

**Absence of Q waves** — A subset of patients who present with initial ST segment elevation do not develop Q waves. These patients are treated for an STEMI. Such patients have a better prognosis than those who develop Q waves because of more frequent reperfusion, a less severe infarction, and, at follow-up, better left ventricular function and improved survival. (See "Electrocardiogram in the prognosis of myocardial infarction or unstable angina", section on 'Presence or absence of new Q waves'.)

**Localization** — The ECG can be used to localize the MI, and at times, predict the infarct-related artery. These issues are discussed separately. (See "Electrocardiogram in the diagnosis of myocardial ischemia and infarction".)

If there is ECG evidence of inferior wall ischemia (ST or T wave changes in leads II, III, and aVF), the right-sided leads V4R, V5R, and V6R should also be obtained to evaluate the possibility of right ventricular infarction. The recording of right-sided leads in this setting was given a class I recommendation by the 2004 ACC/AHA task force [11]. No changes to this approach were made in the 2007 focused update of the
2004 ACC/AHA guidelines for the management of patients with STEMI [14]. Posterior leads (V7-9) are also indicated for those who present with ST segment depression in the inferior leads to evaluate the possibility of posterior infarction [15].

**Other causes of ST elevation and Q waves** — Although ST segment elevation and Q waves are consistent with acute MI (particularly if new), each alone can be seen in other disorders (table 3 and table 4). As examples, ST segment elevation can occur in myocarditis, acute pericarditis, patients with old MI and persistent ST segment elevation often associated with wall motion abnormalities, and with the early repolarization variant; Q waves can be seen in hypertrophic cardiomyopathy. (See "Electrocardiogram in the diagnosis of myocardial ischemia and infarction" and "Pathogenesis and diagnosis of Q waves on the electrocardiogram" and "Clinical manifestations and diagnosis of myocarditis in adults").

**Non-ST elevation ACS** — A non-ST elevation ACS is manifested by ST depressions and/or T wave inversions without ST segment elevations or pathologic Q waves. These ST-T wave abnormalities may be present diffusely in many leads; more commonly, they are localized to the leads associated with the region of ischemic myocardium. (See "Electrocardiogram in the diagnosis of myocardial ischemia and infarction").

As noted above, the two forms of non-ST elevation ACS (UA and NSTEMI) are frequently indistinguishable at initial evaluation (prior to biomarker elevation). In a patient with an NSTEMI, ST segment depressions usually evolve over the subsequent few days to result in residual ST segment depression and T wave inversions, but not to the formation of pathologic Q waves. In a patient with UA, ST segment and T wave changes usually resolve completely.

The joint ESC/ACCF/AHA/WHF committee for the definition of MI established specific ECG criteria for the diagnosis of NSTEMI [6,13]: new horizontal or down-sloping ST depression ≥0.05 mV in two contiguous leads and/or T inversion ≥0.1 mV in two contiguous leads with prominent R wave or R/S ratio >1.

**Nondiagnostic initial ECG** — The initial ECG is often not diagnostic in patients with MI. In two series, for example, the initial ECG was not diagnostic in 45 percent and normal in 20 percent of patients subsequently shown to have an acute MI [16,17]. In patients clinically suspected of having an acute MI in whom the ECG is nondiagnostic, it is recommended that the ECG should be repeated at 20- to 30-minute intervals for any patient with ongoing pain in whom the suspicion of ACS remains high. In some patients, initially nondiagnostic ECG changes will evolve into ST elevation or ST depression [16,18].

The efficacy of repeated ECGs was addressed in a study of 1000 patients presenting to the emergency department with chest pain in whom serial ECGs were obtained every 20 minutes for an average of two hours. Serial ECGs were equally specific (95 percent) but more sensitive than an initial single ECG for detecting an acute MI (68 versus 55 percent) [16].
Bundle branch block or paced rhythm — Both LBBB, which is present in approximately 7 percent of patients with an acute MI [19], and pacing can interfere with the ECG diagnosis of MI or coronary ischemia. Of note, approximately one-half of patients with LBBB and an acute MI do not have chest pain [20]. New right bundle branch block, while generally not interfering with the ECG diagnosis of STEMI, connotes an adverse prognosis similar in degree to LBBB.

Patients with LBBB, compared to those without bundle branch block, are much less likely to receive aspirin, beta blockers, and reperfusion therapy [19,20], particularly if they present without chest pain [20]. Similar observations have been made in patients with a paced rhythm [21].

Careful evaluation of the ECG may show some evidence of coronary ischemia in patients with LBBB or a paced rhythm. However, the clinical history and cardiac enzymes are of primary importance in diagnosing MI in this setting. (See "Electrocardiographic diagnosis of myocardial infarction in the presence of bundle branch block or a paced rhythm".)

CARDIAC BIOMARKERS — A variety of biomarkers have been used to evaluate patients with a suspected acute myocardial infarction (MI). The cardiac troponins I and T as well as the MB isoenzyme of creatine kinase (CK-MB) are the most frequently used.

The use of these tests is discussed in detail elsewhere, but the general principles will be briefly reviewed here. Values ≥99 percentile of the upper reference limit should be considered abnormal [2]. This value for troponin and CK-MB will vary depending on the assay used. (See "Troponin testing: Clinical use".)

The following discussion will emphasize the diagnostic role of cardiac markers. These markers as well as many other factors are important for risk stratification. These issues for both ST elevation and non-ST elevation MI (STEMI and NSTEMI) are discussed separately. (See "Risk stratification after acute ST-elevation myocardial infarction" and "Risk stratification after non-ST elevation acute coronary syndrome" and "Risk factors for adverse outcomes after non-ST elevation acute coronary syndromes".)

An elevation in the concentration of troponin or CK-MB is required for the diagnosis of acute MI [1,2]. If both are measured and the troponin value is normal but the CK-MB is elevated, MB is likely due to release from noncardiac tissue. Follow-up on such individuals reveals that they do extremely well without subsequent events [22].

Troponin is the preferred marker for the diagnosis of myocardial injury for all diagnostic categories because of its increased specificity and better sensitivity compared to CK-MB [1,2,11]. However, an elevation in cardiac troponins must be interpreted in the context of the clinical history and electrocardiogram (ECG) findings since it can be seen in a variety of clinical settings and is therefore not specific for an acute coronary syndrome (ACS). The new guidelines endorse the concept that if there are elevations of cardiac troponin (cTn) in a situation where ischemia is not present, the term cardiac injury should be used. (See 'Other causes of biomarker elevation' below and "Elevated cardiac troponin concentration in the absence of an acute coronary syndrome".)
As there can be chronic elevations of troponin in patients who do not have acute events, the guidelines emphasize the need for a changing pattern of values [1,2,23]. The magnitude of change needed to operationalize this recommendation varies from assay to assay, so it is optimal when the clinical laboratory helps to make these distinctions [24].

Three points should be kept in mind when using troponin to diagnose acute MI:

- With contemporary troponin assays, most patients can be diagnosed within two to three hours of presentation [25].

- A negative test at the time of presentation, especially if the patient presents early after the onset of symptoms, does not exclude myocardial injury.

- Acute MI can be excluded in most patients by six hours, but the guidelines suggest that if there is a high degree of suspicion of an ACS, a 12-hour sample should be obtained. [1,2,11]. However, very few patients become positive after eight hours [26].

**Other causes of biomarker elevation** — Elevations of biochemical markers diagnose cardiac injury, not infarction due to coronary artery obstruction [27]. If an ischemic mechanism of injury is present, for example, as indicated by ischemic ECG changes, then an ACS is diagnosed.

Otherwise, other mechanisms for cardiac injury must be considered (eg, heart failure, rapid atrial fibrillation, myocarditis, anthracycline cardiotoxicity, subendocardial wall stress, myopericarditis, sepsis, etc). As an example, small amounts of cardiac injury can occur in critically ill patients, which may or may not represent an acute MI [28,29]. Troponin elevations also occur in chronic kidney disease. (See "Elevated cardiac troponin concentration in the absence of an acute coronary syndrome").

In the emergency department setting, life-threatening causes of chest pain with troponin elevation not due to coronary artery disease are acute pulmonary embolism, in which troponin release may result from acute right heart overload, myocarditis [27], and stress-induced cardiomyopathy. (See "Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism" and "Clinical manifestations and diagnosis of myocarditis in adults", section on 'Cardiac biomarkers'.)

**Absence of biomarker elevation** — Using older assays, some patients with STEMI who were rapidly reperfused did not develop a cardiac biomarker elevation. These patients were called "aborted MIs." With contemporary troponin assays, this does not occur or is extremely rare. For example, in an analysis of 767 patients with STEMI, the frequency of patients who had elevations of troponin above the 99th percent value was 100 percent [30]. For patients with ST elevation on the ECG and no biomarker elevation, one of the other causes of ST elevation should be considered (table 3). (See 'Other causes of ST elevation and Q waves' above and "Fibrinolysis for acute ST elevation myocardial infarction: Initiation of therapy", section on 'Introduction'.)
Discordant cardiac enzymes biomarkers — At least one-third of patients with an ACS have elevated troponins but normal CK-MB [22,31-33]. The frequency and prognostic significance of discordant troponin and CK-MB were illustrated in a review of almost 30,000 such patients from the multicenter CRUSADE initiative in the United States [33]. The following findings were noted:

- The results were discordant in 28 percent of patients despite the fact that many centers were still using either high cut-off values or inadequately sensitive troponin assays. Troponin was more sensitive, as 18 percent had elevated troponin but normal CK-MB values. In addition, 10 percent had false positive CK-MB elevations, as defined by normal troponin values.

- Compared to patients who were negative for both biomarkers, in-hospital mortality was not increased in patients who were Tn-negative and CK-MB-positive (ie, false positives; 3.0 versus 2.7 percent, adjusted odds ratio 1.02, 95% CI 0.75-1.38).

- Compared to patients who were negative for both biomarkers, there was a nonsignificant trend toward increased mortality in patients who were cTn-positive/CK-MB-negative (4.5 versus 2.7 percent, adjusted odds ratio 1.15, 95 percent CI 0.86-1.54) and a significant increase in mortality in patients who were positive for both biomarkers (5.9 versus 2.7 percent, adjusted odds ratio 1.53, 95 percent CI 1.18-1.98). The latter finding reflects the fact that patients with larger insults do worse [34]. The two discordant groups were treated similarly with antithrombotic agents and percutaneous coronary intervention, so differences in outcomes are less likely to be explained by differences in therapy. Thus, an isolated CK-MB elevation has limited prognostic value in patients with a non-ST elevation ACS. Several meta-analyses suggest that patients with isolated troponin elevations do much worse than those without elevations [35,36]. If high cut-off values are used, these effects are obfuscated by the admixture of patients with and without real troponin elevations.

Similar findings were noted among 1825 patients with a non-ST elevation ACS in the TACTICS-TIMI 18 trial in whom troponin T was measured; 668 (37 percent) had elevated CK-MB, almost all of whom had elevated troponin T, while 361 patients (20 percent) had an elevated troponin T with normal CK-MB [32]. The latter patients had the greatest benefit from an early invasive strategy. (See "Coronary angiography and revascularization for unstable angina or non-ST elevation acute myocardial infarction").

Recommended approach — The following general statements apply to the biochemical diagnosis of an acute MI [2].

Troponins are the markers of choice and should be used in preference to CK-MB. The one remaining area of controversy is in the evaluation of catheter-based periprocedural events [1,2]. (See "Troponin testing: Clinical use", section on 'Reinfarction'.)

We recommend the following approach [22]:

- Measure serum troponin-I or troponin-T at first presentation
● If the troponin is not elevated, repeat at six to nine hours. It is not uncommon to measure a second troponin earlier than six hours in patients who are highly suspected of having ongoing NSTEMI, since 80 percent of patients who rule in will do so in two to three hours [25]. In an occasional patient in whom the index of suspicion for acute MI is high, but the first two troponin measurements are not elevated, a repeat measurement at 12 to 24 hours may be necessary.

● CK-MB is measured when a troponin assay is not available. Previously, CK-MB was advocated to help diagnose reinfarction, but now troponin has subsumed that role [2]. Reinfarction is diagnosed if there is a ≥20 percent increase of the value in the second sample [2].

● Troponin elevations persist for one to two weeks after acute MI, but values are usually not rising or falling rapidly at this time, allowing one to distinguish acute from more chronic events [2,23].

SOCIETY GUIDELINE LINKS — Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Non-ST elevation acute coronary syndromes (non-ST elevation myocardial infarction)" and "Society guideline links: ST elevation myocardial infarction (STEMI)".)

INFORMATION FOR PATIENTS — UpToDate offers two types of patient education materials, “The Basics” and “Beyond the Basics.” The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on “patient info” and the keyword(s) of interest.)

● Beyond the Basics topic (see "Patient education: Heart attack (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS — Myocardial infarction (MI) is defined as a clinical (or pathologic) event caused by myocardial ischemia in which there is evidence of myocardial injury or necrosis. Criteria are met when there is a rise and/or fall of cardiac biomarkers, along with supportive evidence in the form of typical symptoms, suggestive electrocardiographic (ECG) changes, or imaging evidence of new loss of viable myocardium or new regional wall motion abnormality. (See 'Introduction' above.)
The criteria used to define MI differ somewhat depending upon the particular clinical circumstance of the patient: those suspected of acute MI based upon their presentation; those undergoing either coronary artery bypass graft surgery (CAB) or percutaneous coronary intervention (PCI); or those who have sustained sudden, unexpected cardiac arrest with or without death. (See 'Third universal definition of MI' above.)

We recommend the following approach to diagnose an acute MI (excluding patients who have just undergone revascularization):

- An ECG, an abbreviated history (which focuses on the chest pain), and physical examination should be obtained within 10 minutes of patient arrival. (See 'ECG' above and 'Chest pain' above.)

- Measure serum troponin-I or troponin-T at first presentation. (See 'Cardiac biomarkers' above.)

- If the serum troponin is not elevated, repeat at six to nine hours. It is not uncommon to measure a second troponin earlier than six hours in patients who are highly suspected of having ongoing NSTEMI. In an occasional patient in whom the index of suspicion for acute MI is high but the first two troponin measurements are not elevated, a repeat measurement at 12 to 24 hours may be necessary. (See 'Cardiac biomarkers' above.)

- Measure serum creatine kinase-MB when a troponin assay is not available. (See 'Cardiac biomarkers' above.)

- For patients who have undergone recent revascularization with either PCI or CABG, we suggest measurement of troponin after the procedure. In order to diagnose MI resulting from either PCI or CABG, the baseline troponin has to have been normal, and thus a troponin should be ordered prior to all revascularization procedures. (See 'After revascularization' above.)

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