Acute Coronary Syndromes

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Despite technologic advances in many diagnostic fields, the 12-lead ECG remains the basis for early identification and management of an acute coronary syndrome (ACS). Complete occlusion of coronary arteries (>90%) alters the epicardial surface electrical potentials and usually manifests as ST segment elevation (STE) in two or more adjacent leads. STE may range from <1 mm in a single lead to massive STE as great as 10 mm in multiple leads. This injury pattern represents a myocardial region at risk for (irreversible) myocardial infarction (MI). Such an injury pattern usually leads to at least some myocardial cell death (measured by troponin elevation) and is called ST elevation myocardial infarction (STEMI). STEMI indicates the potential for a substantial irreversible infarction (large risk area) and is the primary indication for emergent reperfusion therapy to salvage myocardium.

In ACS, the elevation of biomarkers (eg, troponin) without recorded STE indicates myocardial cell death, but not necessarily that which should be treated with urgent reperfusion therapy. This acute MI (AMI) without STE, though usually with ST segment depression (STD) or T-wave changes, is referred to as non-STEMI (NSTEMI). Unstable angina (UA) implies fully reversible ischemia without troponin release, and its initial clinical and ECG presentation is frequently indistinguishable from NSTEMI. Symptoms of UA are often brief, whereas symptoms of AMI are usually of at least 20 minutes duration; however, patients with 48 hours of symptoms may have UA and those with 5 minutes of symptoms, or none at all, may suffer from NSTEMI. UA and NSTEMI result from a nonocclusive thrombus, small risk area, brief occlusion (spontaneously reperfused), or an occlusion

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that maintains good collateral circulation. In many such cases, there would have been STE, or other ST segment or T-wave abnormalities, had an ECG been recorded at the appropriate time. Similarly, the presence of troponin elevation does not necessarily imply ongoing injury or ischemia; this is one reason that the recorded ECG may be normal in AMI. UA and NSTEMI do not require emergent percutaneous coronary intervention (PCI), but PCI within 48 hours reduces the morbidity and mortality of UA/NSTEMI [1].

Many patients who have STEMI who are eligible for emergent reperfusion therapy still do not receive it; this is largely because of difficulties in ECG interpretation, including subtle STE, STE in few leads, and left bundle branch block (LBBB) [2–6]. Patients who have AMI with subtle or nondiagnostic ECGs and atypical symptoms are most likely to be overlooked for reperfusion therapy. Up to 4% are discharged mistakenly from the emergency department, many because of misread ECGs, and they have a high mortality [7–10]. One third of patients diagnosed with AMI, including STEMI, present to emergency departments without chest pain [11]. It is important to record an ECG even in the presence of nonspecific or vague symptoms, and when the ECG is unequivocally diagnostic for STEMI, to act on the ECG despite even atypical symptoms.

Approximately half of AMI, as diagnosed by creatine kinase—MB (CK-MB), manifest clearly diagnostic STE [12–14]. This percentage is less in the era of troponin-defined diagnosis. Much AMI with subtle STE, however, goes unrecognized. Furthermore, most STE result from non-AMI etiologies (e.g., left ventricular hypertrophy, acute pericarditis, early repolarization, LBBB, and so on) [15–17]. There are thus false positives and false negatives. With such ECGs, the interpretation must be considered in the context of pretest probability of AMI (i.e., the clinical presentation) and by recognition of ECG patterns that mimic AMI [18].

Normal or nondiagnostic ECG as manifestation of non-ST elevation myocardial infarction

A normal initial ECG does not preclude the diagnosis of AMI. Combining two studies, approximately 3.5% of patients who had undifferentiated chest pain and a normal ECG were later diagnosed with AMI by CK-MB, and 9% of such patients who had a nonspecific ECG had an AMI [14,19]. A normal ECG recorded during an episode of chest pain, however, makes ACS a less likely etiology of chest pain, and when ACS is the etiology a normal ECG is associated with a better prognosis [20]. Many additional patients who have normal or nondiagnostic ECGs may have UA. Those who have suspected ACS with a nondiagnostic ECG have fewer in-hospital complications as long as subsequent ECGs remain negative [21,22]. Among patients who have chest pain subsequently diagnosed with AMI by CK-MB, 6% [23] to 8% [14,24,25] have normal ECGs and 22% [26] to 35% [14,19,26] have nonspecific ECGs. There is associated relative mortality risk for AMI.
even with a normal ECG (0.59) or a nonspecific ECG (0.70) when compared with a diagnostic ECG [26]. These figures are not surprising, especially considering that the coronary plaque is unstable and that many normal or nondiagnostic ECGs are recorded at a temporary moment of adequate perfusion. Accordingly, the sensitivity of the ECG for AMI, including STEMI, is greatly improved with the use of serial ECGs or ST segment monitoring in patients at high clinical suspicion [27]. Furthermore, prehospital tracings often reveal STE that is no longer present in the ED [28].

**Evolution of ST elevation myocardial infarction**

Within minutes of a coronary occlusion, if recorded on the ECG, hyperacute T waves may manifest, followed by STE (Fig. 1). If the occlusion persists, Q wave formation may begin within 1 hour and be completed by 8–12 hours (representing completed MI) [29]. STE that has peaked rapidly begins to fall slowly as irreversible infarction completes. Shallow T-wave inversion develops within 72 hours; stabilization of the ST segment usually within 12 hours [30], with or without full ST segment resolution over the ensuing 72 hours [31]. T waves may normalize over days, weeks, or months [32]. STE completely resolves within 2 weeks after 95% of inferior and 40% of anterior MI; persistence for more than 2 weeks is associated with greater morbidity [31]. Approximately 60% of patients who have MI with persistent ST segment displacement have an anatomic ventricular aneurysm [31].

**Hyperacute T waves**

Prominent T waves associated with the earliest phase of STEMI are termed hyperacute T waves (Figs. 2–4). Experimentally, these bulky and wide T waves, often with a depressed ST takeoff (see Fig. 3B and 4) [33,34], are localized to the area of injury and may form as early as 2 minutes after coronary ligation but typically present within the first 30 minutes

![Fig. 1. Evolution of inferior STEMI, lead III. Reprinted with permission from Wang K. Atlas of electrocardiography.](image-url)
following a clinical event [35–43]. This short-lived ECG feature rapidly progresses to STE and usually is bypassed in actual clinical situations. Even after STE develops, however, the T wave remains prominent (and often hyperacute), and the height of the T wave correlates with the acuteness of the injury. Even at this early phase, there is only subendocardial ischemia without cellular injury. Hence, there may be no associated elevation of troponin [44]. As hyperacute T waves are a marker of early occlusion,

![Hyperacute T waves](image-url)

**Fig. 2.** Hyperacute T waves. Proximal LAD occlusion manifesting hyperacute T waves in addition to ST elevation.

**Fig. 3.** Four examples of hyperacute T waves. (A) Lead V4, T wave is very large compared with QRS. (B) Lead V3, with depressed ST segment take-off and straightening of the ST segment. (C) wide and bulky, much larger than QRS. (D) This less common form is very peaked and tented, with an appearance of hyperkalemia. *Reprinted with permission from* Chan TC, Brady WJ, Harrigan RA, et al. ECG in emergency medicine and acute care. Elsevier; 2005.
reperfusion therapy begun while T waves are prominent correlates with better outcomes [45–48]. The sequence is reversible: if occlusion is brief, hyperacute T waves may be the last abnormality seen on a normalizing ECG after resolution of STE (Fig. 5).

**ST segment elevation**

STE should be measured from the upper edge of the PR segment (not the TP segment) to the upper edge of the ST segment at the J point; similarly, ST segment depression should be measured from the lower edge of the PR segment to the lower edge of the ST segment, also at the J point. If the ST segment is measured relative to the TP segment, atrial repolarization with a prominent negative Ta wave representing repolarization of the atrium results in an inaccurate measurement. Results are different between measurement at the J point versus 60 ms after the J point [49–52]. On the other hand, STE with a tall T wave, versus without, is much more suggestive of AMI, and measurement at 80 ms after the J point, where the ST segment is slurring up into a tall T wave, reflects the presence of a tall T wave better than measurement at the J point. Measurements are more important for research protocols, however, than for diagnosis of individual patients: a well-informed subjective interpretation of the appearance of the ST segment is more accurate than measured criteria [53,54].

To diagnose STEMI, STE must be new or presumed new. Various clinical trials of thrombolytic therapy have required different STE criteria: for voltage (1 or 2 mm [0.10 or 0.20 mV]) and for the number of leads required (1 or 2 leads) [55–62]. To obtain consistency, a consensus statement defined STEMI as ST segment elevation at the J point, relative to the PR segment, in two or more contiguous leads, with the cut-off points ≥0.2 mV (2 mm) in leads V1, V2, or V3 and ≥0.1 mV (1 mm) in other leads (contiguity in the frontal plane is defined by the lead sequence aVL, I, inverted aVR, II, aVF, aVF, aVF, aVF, aVF, aVF, aVF).
III) [63]. One should always assess the ST segment deviation, however, within the larger context of overall ECG morphology and clinical presentation. Minimal STE may well be the result of coronary occlusion; conversely, STE exceeding criteria may be the patient’s baseline.

With prolonged ischemia, the prominent T waves remain as STE develops. The ST segment evolves from an upwardly concave morphology to one that is straight and then convex (Fig. 6; and see Fig. 4). A concave ST morphology may persist but is more common in nonpathologic states. In anterior AMI, an upwardly concave waveform in V2–V5 is common (Fig. 7; and see Figs. 2 and 5) [64], but upwardly convex morphology is more specific for STEMI and is associated with greater infarct size and morbidity [65]. Coronary occlusion is often transient or dynamic with cyclic reperfusion and reocclusion (see Fig. 5) [66]. Indeed, transient STE caused by spontaneous reperfusion occurs in approximately 20% of STEMI, especially after aspirin therapy [67]. Occlusion may be associated with minimal STE, and if morphology is concave upward, the diagnosis may be missed—but should be suspected if the T wave towers over the R wave or over a Q wave (Fig. 8).

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**Fig. 5. Dynamic nature of ST segment elevation.** (A) Prehospital tracing of a patient with left hand weakness and numbness. There is high STE in V1–V4 with upwardly concave ST segments, but also STE in lead aVL with reciprocal depression in inferior leads. (B) First tracing in the ED, leads V1–V6 only. STE has resolved spontaneously. The only abnormality is depressed ST segment takeoff in V2 and V3 (limb leads were normal). Moments later, the ST segments re-elevated and the patient rapidly went into cardiogenic shock; he died before the LAD occlusion could be opened.
Because of cyclic reperfusion and reocclusion, symptoms may be pro-
longed in the absence of any significant irreversible infarction; the ECG it-
self is a better predictor of salvageable myocardium than is symptom
duration. In the presence of high STE and high upright T waves, prolonged
persistent occlusion with irreversible myocardial damage is unlikely, even
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Under the best of circumstances, the ECG has a sensitivity of 56% and specificity of 94% for all AMI as diagnosed by CK-MB [68], but studies vary [12–14]. Even STEMI often is not obvious, and ECG computer algorithms are especially insensitive for this diagnosis (Figs. 8–13) [53,54,69]. Nevertheless, if such algorithms incorporate clinical data, they may increase the percentage of patients who appropriately receive reperfusion therapy [70].

The ECGs with the greatest ST deviation typically result in the shortest time to treatment [71]. Factors such as myocardial mass, distance between the electrodes and the ischemic zone, opposing reciprocal voltage, and especially QRS complex amplitude, may affect the magnitude of STE, so that subtle STE (ie, elevation <2 mm in V1–V3, or ≤1 mm in other leads) may represent persistent coronary occlusion and may be missed easily.

Fig. 8. Unusually large T wave with subtle ST segment elevation. This tracing from a 91-year-old patient with LAD occlusion manifesting as tall T waves that tower over a tiny R wave (V3) and Q wave (V2). This was misinterpreted as early repolarization, which should have well developed R waves and is unusual in elderly patients. The computer did not detect this AMI.

Fig. 9. Inferoposterior STEMI completely missed by the computer. There is a QR wave in lead III very soon after occlusion. There is the obligatory reciprocal ST depression in aVL, and reciprocal STD in V2 and V3 diagnostic of posterior STEMI. STE in lead III is >STE in lead II; there is significant STD in lead I; thus it is an right coronary artery (RCA) occlusion (with posterior branches). Reprinted with permission from: Chan TC, et al. (Eds.), ECG in Emergency Medicine and Acute Care.
Such cases may be difficult to recognize or difficult to differentiate from other etiologies, or both. In the presence of a small QRS, STE may be minimal; amplitude ratios are more accurate than absolute amplitudes [72]. When deciding if any anterior ST elevation is due to left anterior descending coronary artery occlusion vs. due to normal variant, the height of the R wave appears to be most important, with a mean R wave $\geq 5$ mm in V2-V4 highly suggestive of STEMI [73]. STEMI is defined by STE of at least 1 mm; however, as expertise in ECG interpretation is improving in this era of angiographic correlation, many coronary occlusions manifesting lesser STE, or in only one lead, or simply hyperacute T waves, are being detected and treated with emergent PCI (see Figs. 7 and 11–13). Change from previous ECGs, changes over minutes to hours (see Fig. 7), the presence or absence of reciprocal STD, or presence of upward convexity, may help make the diagnosis. Circumflex or first diagonal occlusion may present with minimal or no STE [74–76] despite large myocardial risk area [77,78], because the lateral wall is more electrocardiographically silent.

Fig. 10. Inferoposterolateral STEMI completely missed by the computer. There is the obligatory reciprocal ST segment depression in lead aVL, and also reciprocal depression in leads V2 and V3 diagnostic posterior STEMI. Predictors of infarct-related artery are: STE in lead III $>$ STE in lead II (favoring RCA), STD in lead I is minimal (favoring the left circumflex (LCX)), and STE in leads V5 and V6 (favoring LCX); angiography showed LCX occlusion.

Fig. 11. RCA occlusion manifesting minimal ST deviation, also missed by the computer. There is STE in leads II, III, and aVF with reciprocal depression in leads I, aVL, and leads V2–V5, but all $<1$ mm.
Prognostic features of ST segment elevation

The following ECG features of STEMI, in decreasing order of importance, are associated with larger MI, higher mortality, and greater benefit from reperfusion therapy, and may help in determining the benefit/risk ratio of particularly risky (relatively contraindicated) therapies. Though the correlations are real, there remains wide individual variation such that some patients without these features may have a large AMI [79]: (1) anterior location, compared with inferior or lateral [80–84]; (2) total ST deviation or the absolute sum of STE and STD [50,85]; (3) ST score (the sum of all STE) greater than 1.2 mV (12 mm) (these last two features each take into account the prognostic effects of greater height of ST segments and greater number of leads involved) [83]; and (4) distortion of the terminal portion of

Fig. 12. Obtuse marginal occlusion, also missed by the computer. Reciprocal STD in leads II, III, and aVF is the most visible sign of STEMI. STE is 0.5 mm in leads I and aVL, but in the presence of a low voltage QRS complex. Also, there are nondiagnostic ST segment/T wave changes in leads V4–V6.

Fig. 13. First diagonal occlusion, also missed by the computer. Reciprocal STD in leads II, III, and aVF is the most visible sign of STEMI. There is left anterior fascicular block, but this does not obscure the diagnosis. STE is 0.5 mm in leads I and aVL, but large compared with a low voltage QRS complex. There is nondiagnostic T-wave inversion in leads V2–V6. Moments later this patient suffered a cardiac arrest. PCI was successful after resuscitation.
the QRS (loss of S-wave in leads with RS configuration, or J point ≥ 50% the height of the R wave) [86] (see Fig. 1).

ST segment depression

Primary STD (Fig. 14)—if not caused by posterior STEMI or reciprocal changes to STE—is an ECG sign of subendocardial ischemia, and in the context of ACS, indicates UA/NSTEMI. STD of even 0.5 mm from baseline is associated with increased mortality, but it is particularly significant when ≥1 mm (0.10 mV) in two or more contiguous leads [87]. This adverse prognostic association is independent of elevated troponin [88]. Although STD, especially upsloping STD, may be baseline and stable, the STD associated with UA/NSTEMI is transient and dynamic. Its morphology is usually flat or downsloping. Concurrent T-wave inversion may or may not be present. STD may be induced by exercise in the presence of stable coronary stenosis. Just as with STE, the proportionality or lead strength of STD is important: 1 mm of STD following a <10 mm R wave is more specific for ischemia but less sensitive than is 1 mm of STD following a >20 mm R wave [89–92].

STD >2 mm and present in three or more leads is associated with a high probability of elevated CK-MB and near universal elevation of troponin. In the absence of PCI, such STD is associated with a 30-day mortality of up to 35% and a 4-year mortality of 47% [93], whether or not there is complete coronary occlusion. Lesser degrees of STD (in the absence of PCI) are associated with 30-day mortality rates from 10%–26% [85,94] and also with a high incidence of left main or three-vessel disease [95]. Primary STD, with the exception of that which represents posterior STEMI or that which is reciprocal to other STE, is not an indication for thrombolytic therapy [96–98]. In the pre-interventional era, patients who had STD and AMI (by

![Fig. 14. ST segment depression. Leads V1–V6 only. Flat STD of LAD subendocardial ischemia is likely when STD reaches from leads V2–V6 and is transient. Thrombolytic therapy thus is not indicated. Furthermore, as in most UA/NSTEMI, the STD resolved quickly with medical therapy.](image-url)
CK-MB) had a higher mortality than those with STE who were eligible for thrombolytics (STEMI) and received them [94,99]. STD (even as little as 0.5 mm) [87,95], independently of and in addition to elevated troponin, is a strong predictor of adverse outcome and is one of the best indicators of benefit from early (within 48 hours) PCI, in addition to intensive medical therapy [1,95]. Persistent STD in the setting of persistent angina despite maximal medical therapy is an indication for urgent angiography with possible percutaneous coronary intervention, but not for thrombolytic therapy.

Reciprocal STD is the electrical mirroring phenomenon observed on the ventricular wall opposite transmural injury (see Figs. 4, 5, 9, and 10). This simultaneous STD improves the specificity for STEMI in the anatomic territory of the STE, but true reciprocal STD does not reflect ischemia in the territory of the STD. Hence, in the reciprocal territory, there will be no associated wall motion abnormality on echocardiogram or myocardial perfusion defect with nuclear imaging. Because a significant number of STEMIs do not develop reciprocal STD, absence of reciprocal ST depression does not rule out STEMI [16,100–104]. In the presence of abnormal conduction (eg, left ventricular hypertrophy [LVH], bundle branch block [BBB], or intraventricular conduction delay [IVCD]), STD may be secondary to this abnormal QRS complex, and, if so, it does not contribute substantially to the diagnosis [104].

Three situations frequently are called reciprocal; only the first represents true reciprocity or mirroring of STE present on the 12-lead: (1) true reciprocity of the leads with STE (eg, in inferior AMI, reciprocal STD in lead aVL, which is 150° opposite from lead III [see Fig. 9]); (2) posterior STEMI (ie, ST depression in leads V1–V4, with or without STE in leads V5 and V6 or leads II, III, and aVF), (Fig. 15; see Figs. 9 and 10). In this case, the STD is truly reciprocal, but only to what would be STE on posterior leads, not to inferior or lateral STE; (3) simultaneous UA/NSTEMI of another coronary distribution (not in any way reciprocal).

Anterior AMI manifests reciprocal STD in at least one of leads II, III, and aVF in 40%–70% of cases; this STD correlates strongly with a proximal left anterior descending (LAD) occlusion (see Figs. 4 and 5) [105–108]. In the presence of inferior AMI, reciprocal STD usually is present in leads I and aVL, and often in the precordial leads, especially V1–V3 (56% of cases) [109]. Reciprocal STD is associated with a higher mortality [85], but also with greater benefit from thrombolytics [50]. This is especially true of precordial STD in inferior AMI [109]. In some cases, reciprocal STD is the most visible sign of STEMI (see Figs. 4, 12, and 13) [34,110].

T-wave inversion

In the presence of normal conduction, the normal T-wave axis is toward the apex of the heart and is close to the QRS axis: the T wave is usually upright in the left-sided leads I, II, and V3–V6; inverted in lead aVR; and
variable in leads III, aVL, aVF, and V1, with rare normal inversion in V2. When abnormally inverted, if in the presence of symptoms suggesting ACS, such T waves should be assumed to be a manifestation of ischemia, although there are many nonischemic etiologies of T-wave inversion. Isolated or minimally inverted nondynamic T waves (<1 mm) may be caused by ACS but have not been shown to be associated with adverse outcomes compared with patients who have ACS and a normal ECG [95]; however, T-wave inversion caused by ACS that is >1 mm or in ≥2 leads is associated a higher risk of complications, especially if of Wellens pattern [111,112].

T-wave inversion caused by ACS may be transient (reversible) and may be without significant ST segment shift, indicating transient ischemia. In such a case there is usually no myocardial damage, as measured by troponin, and it is diagnosed as UA.

In general, sustained and evolving regional T-wave inversion suggests either (1) spontaneous reperfusion (of the infarct artery or through collaterals) (Fig. 16), or (2), in the presence of QS waves, prolonged occlusion (Fig. 17). After prolonged, non-reperfused coronary occlusion, as regional ST segments resolve toward the isoelectric level, T waves invert in the same region, but not deeply (up to 3 mm) [113]. Shallow T-wave inversions in the presence of deep QS waves recorded at patient presentation usually represent prolonged persistent occlusion, with (nearly) completed infarction [113]. Even with ongoing STE, it may be too late for thrombolytic therapy.

With reperfusion, whether spontaneous or as a result of therapy or caused by collateral flow, there is often regional terminal T-wave inversion [114,115]. This terminal inversion is identical to Wellens pattern A [34,111] and the cove-plane T [114,115]. The ST segments may retain some elevation, but the T waves invert, resulting in a biphasic appearance (Fig. 18A).

As time progresses after reperfusion, the ST segments recover to near the isoelectric level, are upwardly convex, and the inversion is more symmetric
and deep (> 3 mm) [113]. This is identical to Wellens pattern B [34,111] or the coronary T or Pardee T [116,117] (Fig. 18B and 19).

In both types of Wellens T-wave inversions, the R wave is preserved because reperfusion occurs before irreversible necrosis; both are believed to be a result of ischemia surrounding the infarct zone. If the T-wave inversion is persistent, there is nearly always some minimal troponin elevation, and this pattern frequently is termed non-Q wave MI. If no STE was recorded, this is appropriately termed NSTEMI, though frequently transient STE would have been present had an ECG been obtained at the appropriate moment.

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**Fig. 16.** Occlusion with reperfusion of a wraparound RCA, similar to a wraparound LAD (anterior and inferior AMI). Such widespread STE (inferior, anterior, lateral) with no reciprocal STD (it is absent in lead aVL because of lateral AMI) if the T waves are still upright, frequently is misdiagnosed as pericarditis. Inferior and lateral cove-plane (inverted) T waves clinch the diagnosis of AMI and signify reperfusion of these regions. Angiography confirmed inferior and lateral reperfusion by way of collaterals, but persistent ischemia to the anterior wall.

**Fig. 17.** Anterior STE with QS waves and terminal T-wave inversion. This is diagnostic of STE-MI, but QS waves suggest prolonged occlusion and deep T-wave inversion suggests (late) spontaneous reperfusion. Indeed, this 37-year-old patient’s symptoms had been constant for 32 hours. CK was 5615 IU/L. The LAD, however, was persistently occluded at angiography. Reprinted with permission from Smith SW, Zvosec DL, Sharkey SW, Henry TD. The ECG in acute MI: an evidence-based manual of reperfusion therapy. Fig. 33-8. 1st edition. Philadelphia: Lippincott, Williams, and Wilkins: 2002. p. 358.
Fig. 18. Wellens syndrome. (A) Wellens syndrome, pattern A (leads V1–V3 only). This patient’s chest pain had resolved recently and he now has subtle biphasic terminal T-wave inversion in lead V2; although the QT interval is short, suggesting benign T-wave inversion, but this would be unusual in lead V2 only. (B) Wellens syndrome, pattern A (leads V1–V6 only): the same patient 2 hours later. The ECG now has biphasic terminal T-wave inversion in V2–V5. The QTc interval is 0.45 sec, more typical of Wellens syndrome. This also helps to differentiate from benign T-wave inversion (QTc <0.40–0.425 sec). Troponin but not CK-MB was minimally elevated and there was a very tight LAD stenosis at angiography. (C) Wellens pattern B (leads V1–V6 only). The same patient 9 hours later. The T waves are now monophasic, inverted, and deep.

Wellens syndrome (see Fig. 18A,B) refers to angina with T-wave inversion in the LAD distribution, particularly V2–V4, in the presence of persistent R waves [34,111,118,119] and critical stenosis of the LAD [111,112]. At initial presentation, patients have normal or slightly elevated CK-MB and elevated troponin. The ECG pattern is present in a pain-free state. Wellens’ group noted, however, that without angioplasty, 75% of these patients developed an anterior wall AMI, usually within a matter of days, despite relief of symptoms with medical management. Identical T-wave morphology is recorded after approximately 60% of cases of successful reperfusion therapy for anterior STEMI [114,115], suggesting that Wellens syndrome is a clinical condition created by spontaneous reperfusion of a previously occluded critical stenosis. Similar patterns also occur in other coronary distributions, eg, inferior, lateral, or both (see Fig. 16), but the syndrome was described originally in the LAD. Wellens syndrome is to be distinguished from benign T-wave inversion by (1) longer QT interval (>425 ms as opposed to <400–425 ms) and (2) location V2–V4 (as opposed to V3–V5).

In the presence of prior T-wave inversion, reocclusion of the coronary artery manifests as ST segment re-elevation and normalization of terminal T-wave inversion, called T-wave pseudonormalization because the T wave flips upright (Fig. 20). With upright T waves, pseudonormalization should not be assumed if the previous ECG showing T-wave inversion was recorded more than 1 month earlier.

T-wave inversions with no STE are never an indication for thrombolytics. With symptoms of ACS, they represent UA/NSTEMI. Even in the presence

of persistent STE, they usually indicate spontaneous reperfusion or collateral flow, or, if new Q waves are present, a prolonged occlusion; thrombolitics should be given only if ongoing chest pain suggests persistent occlusion and serial ECGs fail to show resolution of STE (see Fig. 16).

**Q waves**

Before the thrombolytic era, MI was classified based on its clinical pathology: either as Q wave or non-Q wave MI, or as transmural versus subendocardial MI [120]. These terms later were discovered to be clinically and pathologically unrelated. Q waves correlate with the volume of infarcted myocardium, rather than the transmural extent of MI [121]. The Q wave/non-Q wave distinction remains useful, because Q waves are associated with a lower ejection fraction and a larger MI [121–125]. Most patients with non-reperfused STEMI ultimately develop Q waves, whereas a minority do not [126]; in any case, they often appear after the important initial diagnostic and therapeutic interventions have occurred. Hence, AMI is now classified as STEMI or NSTEMI.

A normal Q wave representing the rapid depolarization of the thin septal wall between the two ventricles may be found in most leads (Box 1). This initial negative deflection of the QRS complex is of short duration and of low amplitude. Pathologic Q waves, often a consequence of MI, are generally wider and deeper than normal Q waves. Following MI with significant

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**Box 1. Abnormal Q waves**

- Lead V2: any Q wave
- Lead V3: almost any Q wave
- Lead V4: >1 mm deep or at least 0.02 sec or larger than the Q wave in lead V5
- Any Q wave ≥0.03 sec (30 ms, 0.75 mm), except in leads III, aVR, or V1 (see below)
- Lead aVL: Q wave >0.04 sec or >50% of the amplitude of the QRS in the presence of an upright p-wave
- Lead III: Q wave ≥0.04 sec. A Q wave of depth >25% of R wave height is often quoted as diagnostic, but width is more important than depth
- Leads III, aVR, V1: normal subjects may have nonpathologic wide and deep Q waves

loss of myocardium, electrically inactive tissue fails to produce an R wave in the overlying leads; depolarization of the opposite wall in the opposite direction then gets recorded negatively (Q wave). A QR wave denotes a Q wave followed by a substantial R wave (see Fig. 16, inferior leads); a Qr wave denotes a Q wave followed by a very small R wave (see Fig. 9, lead III); a qR wave denotes a small Q wave preceding a large R wave (see Fig. 16, lateral leads); and a QS wave denotes a single negative deflection without any R wave (see Fig. 17).

There are several Q wave equivalents seen in the precordial leads. These include (1) R wave diminution—or poor R wave progression; (2) reverse R wave progression, in which R waves increase then decrease in amplitude across the precordial leads (although this must be distinguished from precordial electrode misconnection); and (3) tall R waves in leads V1 and V2, representing “Q waves” of posterior infarction.

Because Q waves commonly are considered markers of irreversible infarction, reperfusion therapy often is denied to patients whose ECGs already manifest Q waves. By 60 minutes after LAD occlusion, however, QR waves, but usually not QS waves, occur in the right precordial leads in 50% of patients, and they frequently disappear after reperfusion [127]. These QR waves are caused by ischemia of the conducting system and not irreversible infarction [29]. Although patients who have high ST segments and absence of Q waves have the greatest benefit from thrombolytic therapy, those who have high ST segment elevation and QR waves also receive significant benefit (see Figs. 9 and 16) [29,30]. Q waves signifying necrosis should be developed completely within 8–12 hours of onset of persistent occlusion [29,30], although at least 10% of patients do not develop them for up to 2 weeks [128]. In most patients, these Q waves persist indefinitely, but in up to 30% of AMI that receives no reperfusion therapy, the Q waves eventually disappear [129]. In contrast, when patients who have Q waves receive early thrombolytic therapy, the Q waves disappear within a few days to weeks [30,130].

**Significance of pathologic Q waves for reperfusion therapy in acute coronary syndromes**

When the significance of STE is uncertain, the presence of a pathologic QR wave in that lead increases the probability that the STE is caused by AMI (not pseudoinfarction) and is amenable to immediate reperfusion. Pathologic QR waves with or without acute ST segment/T-wave changes increase the likelihood of ACS, because it is strong evidence of the presence of coronary artery disease (CAD). Because pathologic QR waves may be present very early in AMI, and because they are associated with benefit from thrombolytic therapy [29], they should not in any way dissuade from reperfusion therapy. A QS pattern caused by MI represents lack of any R wave
depolarization of normal tissue, and thus usually indicates significant irreversible myocardial loss (see Fig. 17). QS waves in leads V1–V3, which may occur with STE, also may be caused by LBBB, left ventricular hypertrophy (LVH), cor pulmonale, or cardiomyopathy. If STE without deep T-wave inversion exists in the presence of a QS wave, LV aneurysm also should be considered.

Reperfusion and reocclusion

Together with angiographic evidence of microvascular perfusion, resolution of STE is the best predictor of outcome from STEMI [34,131]. On continuous ST segment monitoring after reperfusion therapy, a recovery of the ST segment to < 50% of its maximal height by 60 minutes is associated strongly with TIMI-3 reperfusion, and even more strongly associated with good microvascular perfusion [132].

A less sensitive but highly specific predictor of reperfusion is terminal T-wave inversion identical to that of Wellens T waves (see Figs. 16, 18, and 19) [114,115]. In patients who have STEMI, the presence of negative T waves very early after presentation or very soon after therapy is associated with a good prognosis [48]. Negative T waves at discharge of patients who have anterior STEMI is correlated strongly with ST recovery to baseline and TIMI-3 flow [133]. Whether reperfusion is spontaneous or therapy-induced, reocclusion can be detected by re-elevation of ST segments or by pseudonormalization of inverted T waves within hours, days, or weeks of the index AMI (Fig. 20). It may be confused with postinfarction regional pericarditis. T-wave normalization beyond 1 month is expected without reocclusion and is not necessarily pseudonormalization.

Regional issues in acute coronary syndrome

Anterior myocardial infarction

Anatomy

See Table 1 for correlations between coronary occlusion and location of STEMI, and Fig. 21 for coronary anatomy.

The left main coronary artery supplies the LAD artery and the left circumflex (LCX) artery. Persistent occlusion of the left main usually leads to cardiogenic shock and death. The LAD supplies the anterior wall, with branches supplying the anterolateral wall (diagonal arteries) and most of the septum (septal arteries); it may extend distally around the apex to the inferior wall (“wrap-around” LAD) (see Fig. 16). The first septal branch (S1) usually originates from the LAD proximal to the first diagonal branch (D1); but in some it originates distal to D1. The ramus intermedius may arise at the division of the left main, producing a trifurcation, and is present in one third of subjects [134]. It supplies the anterolateral wall.
Table 1
ST elevation, location of STEMI, and corresponding coronary artery

<table>
<thead>
<tr>
<th>ST elevation</th>
<th>Coronary artery (see Fig. 21)</th>
<th>AMI location</th>
</tr>
</thead>
<tbody>
<tr>
<td>II, III, aVF (reciprocal ST depression in aVL)</td>
<td>RCA 80% (III &gt;II, STD in I)</td>
<td>Inferior AMI</td>
</tr>
<tr>
<td></td>
<td>Dominant circumflex (II &gt;III, no STD in I)</td>
<td></td>
</tr>
<tr>
<td>II, III, aVF (reciprocal ST depression in aVL)</td>
<td>RCA proximal to RV marginal branch</td>
<td>Inferior and RV AMI</td>
</tr>
<tr>
<td>plus V1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right-sided ECG: V4R</td>
<td></td>
<td></td>
</tr>
<tr>
<td>II, III, aVF (reciprocal ST depression in aVL)</td>
<td>Dominant RCA (70%)</td>
<td>Inferoposterior AMI</td>
</tr>
<tr>
<td>plus ST depression in V1–V4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>II, III, aVF plus (I, aVL and/or V5, V6)</td>
<td>Dominant circumflex (30%)</td>
<td>Inferolateral AMI</td>
</tr>
<tr>
<td></td>
<td>or Dominant RCA with lateral branches</td>
<td></td>
</tr>
<tr>
<td>II, III, aVF plus (V5, V6) and/or (I, aVL) and</td>
<td>Dominant RCA with lateral branches or</td>
<td>Inferoposterolateral AMI</td>
</tr>
<tr>
<td>ST depression any of V1–V6</td>
<td>circumflex</td>
<td></td>
</tr>
<tr>
<td>V2–V4</td>
<td>Mid LAD, distal to diagonal and septal</td>
<td>Anterior AMI</td>
</tr>
<tr>
<td>I, aVL, V5 and/or V6, sometimes minimal; inferior</td>
<td>perforator</td>
<td></td>
</tr>
<tr>
<td>reciprocal STD very common, may be most obvious</td>
<td>First diagonal or circumflex or obtuse</td>
<td>Lateral AMI</td>
</tr>
<tr>
<td>feature</td>
<td>marginal artery</td>
<td></td>
</tr>
<tr>
<td>V1–V3, sometimes out to V5, with V1&gt;V2&gt;V3&gt;V4&gt;V5,</td>
<td>Right ventricular marginal (RVM), or by</td>
<td>Right ventricular AMI</td>
</tr>
<tr>
<td>with or without inferior STE or Qs</td>
<td>occlusion of RCA proximal to RVM</td>
<td>(pseudoanteroseptal AMI)</td>
</tr>
<tr>
<td>Also V1R–V6R, especially V4R</td>
<td></td>
<td></td>
</tr>
<tr>
<td>V1–V4, often without inferior reciprocal STD</td>
<td>LAD, possibly proximal to first septal</td>
<td>Anteroseptal AMI</td>
</tr>
<tr>
<td></td>
<td>perforator but distal to first diagonal</td>
<td></td>
</tr>
<tr>
<td>V1–V6, I, aVL, 80% with 1 mm STD in II, III, and</td>
<td>Proximal LAD</td>
<td>Anteroseptallateral AMI</td>
</tr>
<tr>
<td>aVF</td>
<td></td>
<td></td>
</tr>
<tr>
<td>STD in V1–V4, with or without lateral or inferior</td>
<td>LCX (absence of STE is possible, or</td>
<td>Posterior AMI</td>
</tr>
<tr>
<td>STE</td>
<td>lateral only)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>RCA (inferior STE, +/- lateral)</td>
<td></td>
</tr>
</tbody>
</table>


There is great variation among patients. Use this for guidelines only.
Mid left anterior descending occlusion (anterograde acute myocardial infarction)

STE in V2–V4, sometimes including V1, is the hallmark of occlusion distal to S1 and D1. There is frequently no inferior reciprocal STD (see Fig. 8). In the presence of a wrap-around LAD, there also may be STE in leads II, III and aVF (see Fig. 16).

Proximal left anterior descending occlusion (anterolateral, anteroseptal, anteroseptolateral acute myocardial infarction)

Lateral component. LAD occlusion proximal to D1 manifests as anterolateral AMI (with STE in leads V2–V4, maximal in V2–V3), and STE in lead aVL and occasionally leads I and V5–V6. Inferior reciprocal STD is present in approximately 80% of cases, depending largely on how STD is defined in number of leads and depth of depression [106,107]. These ST depressions are often reciprocal to STE in leads I and aVL, or to high anterobasal STE in V1. Together with STE in V2–V4, STE in aVL >0.5 mm is very sensitive, and >1.0 mm very specific, for occlusion of the LAD proximal to D1 [104]. Echocardiographic studies comparing proximal and distal occlusions show no difference in apical wall motion [135,136] (see Figs. 2 and 5).

Septal component. STE in V1 traditionally indicates occlusion proximal to S1 with involvement of the septum (anteroseptal MI). Using echocardiography, STE in V1, but not VZ, is associated with basal anterior, anteroseptal and septal regional dysfunction [136]. STE in V1 is not very specific,
however, unless >2.5 mm [107]. Other predictors of occlusion proximal to S1 are (1) STE in lead aVR, (2) STD in lead III > STE in lead aVL, (3) ST segment depression in V5, and (4) right BBB (RBBB) [107,108].

**Lateral and posterior acute myocardial infarction**

**Anatomy**

The left main bifurcates into the LAD and LCX. The lateral wall of the LV is supplied by the LCX and its obtuse marginal (OM) branches and occasionally by D1 from the LAD. When the LCX wraps around to both the posterobasal (posterior) and posteroapical (inferior) walls, giving off the posterior descending artery (PDA), it is the dominant vessel, meaning occlusion results in inferior AMI (STE in II, III, and aVF). Branches of a large dominant right coronary artery (RCA) also may supply the posterolateral wall [137]. Occlusion of a nondominant LCX or one of its obtuse marginal branches accounts for most isolated posterior AMIs (lateral AMI may be present, but rarely pronounced).

**Lateral acute myocardial infarction**

Lateral AMI is a result of occlusion of D1 off the LAD, or of the LCX or its obtuse marginal branches. It may be simultaneous with anterior AMI if the occlusion is in the LAD proximal to D1; it may be simultaneous with posterior AMI in occlusion of the LCX, and with posterior and inferior AMI in occlusion of a dominant LCX (see Fig. 10) or dominant RCA. STE is often <1 mm (0.1 mV), especially in aVL, and especially with low QRS voltage (see Figs. 12 and 13). The sensitivity of STE for detection of lateral AMI is low: with LCX occlusion there is (1) STE in inferior or lateral leads in only 36%, (2) STE >2 mm in only 5%, (3) Isolated STD in 30%, (4) some STE or STD in two thirds of cases, and (5) neither STE nor STD in 33% [74–76,138–140]. This contrasts markedly with LAD or RCA occlusion, which manifest STE in 70%–92% of cases [74,75]. Reciprocal STD in inferior leads is often the most pronounced effect of occlusion of D1 or an OM. STD in V5, V6, and aVL also corresponds with disrupted perfusion of D1 or OM1. Lateral AMI caused by LCX or OM occlusion frequently is accompanied by posterior AMI (see later discussion) (see Figs. 12 and 13).

**Posterior acute myocardial infarction**

From 3.3%–8.5% of all AMIs as diagnosed by CK-MB are posterior AMIs that present without STE on the standard 12-lead ECG, and thus the diagnosis often is missed (see Fig. 15) [34,78,141–144]. These isolated posterior wall AMIs usually present with precordial reciprocal STD, however, often upsloping, in leads V1–V4. STD in V1–V4 may be caused by anterior subendocardial ischemia of the LAD, but this is usually downsloping and transient and extends out to V6 (see Fig. 14). Most individuals have some STE at baseline in V2 and V3 [17], and any amount of STD may
represent a large change in ST amplitude (delta ST). As always, comparison with a baseline tracing is helpful.

Posterior leads V7–V9 reveal posterior STE (Fig. 22). Leads V7–V9 are more specific than precordial leads for posterior AMI (84% versus 57%), with similar sensitivity for both (approximately 80%) [145]. If 0.5 mm is used as a cutoff in leads V7–V9, sensitivity is greatly enhanced but with an unknown change in specificity [144]. Placement of V7–V9 should be in the fifth intercostal space (at the same level as V6), with V7 at the posterior axillary line, V8 at the scapular tip, and V9 at the left paraspinal border. Routine use of posterior leads for all patients who have chest pain is unwarranted, because the vast majority are normal [146,147].

Inferior and right ventricular acute myocardial infarction

Anatomy

The RCA is the dominant vessel in 80% of individuals; that is, it gives off the PDA to supply the inferior wall. The LCX is dominant in 20%, in which case the RCA supplies little more than the right ventricle (RV). RCA occlusion is the most common cause of inferior AMI. Occlusion proximal to the RV marginal branch of the RCA results in concurrent RV AMI (Fig. 23). Occlusion of the RCA with a large posterolateral branch leads to inferolateral, inferoposterior (see Fig. 9), or inferoposterolateral AMI. Occlusion of the LCX in a left-dominant heart manifests as an inferoposterolateral AMI (see Fig. 10). Inferior AMI produces STE in II, III, and aVF. There is almost always reciprocal STD in lead aVL. On occasion, lateral AMI hides

this, and there is usually STE in V5 or V6; however, if STD in aVL is not present, the diagnosis of inferior AMI must be questioned (see Fig. 16).

Determining the culprit vessel may be important: RCA occlusion, if proximal, leads to RV AMI and may result in hypotension, which responds well to fluids and can be exacerbated greatly by nitrates.

LCX (versus RCA) occlusion is strongly predicted by (1) greater STE in lead II than in lead III, and by (2) the absence of reciprocal STD in lead I or aVL [148,149], but also by (3) isoelectric or elevated ST segment in lead I or aVL [150], (4) an abnormally tall R wave in lead V1 [74], or (4) STE in lateral precordial leads V5 and V6 [150]. Additionally, ECG findings of RV AMI suggest RCA occlusion (see later discussion).

RCA occlusion is strongly predicted by (1) STE in lead III > II [149], (2) STD in lead I or aVL with deviation in lead aVL > lead I [150,151], (3) STE in V1, V4R, or both, and (4) STE in V1–V4, a sign of concomitant RV involvement (Fig. 24) [152].

In inferior AMI, simultaneous STD in leads V1–V4 reflects concurrent posterior wall injury (it infrequently reflects anterior wall subendocardial ischemia) and a larger amount of myocardium at risk, higher mortality, and greater benefit from reperfusion; however, this finding does not discriminate between LCX or RCA occlusions [105,109,150,153,154] (see Figs. 9 and 10).

Inferior AMI is sometimes associated with conduction defects at the AV node, including first degree AV block, Mobitz type I (but not type II) or Wenkebach-type AV block, and complete heart block. With complete block, there is usually a stable junctional rhythm with narrow QRS and pulse rate greater than 40 beats per minute (BPM). It is typically transient, and does not require permanent pacing. This type of AV block contrasts sharply to that associated with an extensive anterior AMI. In this infrequent condition the conduction block occurs distal to the AV node, and is associated with Mobitz type II AV block, bifascicular block, or complete heart block. Here, the complete block manifests as a wide junctional or ventricular escape rhythm less

Fig. 23. Acute inferior MI with right-sided leads reflecting RV involvement. The limb leads demonstrate STE in the inferior leads (lead III > lead II), together with reciprocal STD in lead aVL > lead I—all suggesting RCA occlusion. The precordial leads are actually leads V1R–V6R, or right-sided leads. The STE in leads V3R–V6R indicate RV infarction.
than 40 BPM. These patients often present or soon develop cardiogenic shock and have significant mortality regardless of temporary pacing.

**Right ventricular acute myocardial infarction**

Right ventricular acute myocardial infarction (RV AMI) should be suspected when there is acute (or old) inferior MI caused by RCA occlusion (see previous discussion), especially if there is STE in V1 or clinical hypotension. RV AMI is seen in practice exclusively with proximal RCA occlusion or branch occlusion of an RV marginal artery. RV AMI has high short-term morbidity and mortality, especially without reperfusion [155–157], but patients who survive beyond 10 days have a good prognosis [158]. RV AMI
is often associated with conduction defects at the AV node, including complete heart block.

Isolated RV AMI presents with STE in V1–V3, and it may mimic LAD occlusion (pseudo-anteroseptal AMI), except that the STE usually peaks in V1 or V2 and declines progressively as far as V5, whereas in LAD occlusion, STE peaks in V2 and V3 [152]. Large RV AMI may seem to be an anterior MI (of the LV) even in the presence of inferior STE (Fig. 24). Such extensive and profound STE is especially true in the presence of RV hypertrophy [34,159].

When RCA occlusion is suspected, record a right-sided ECG in which lead V1 becomes lead V2R, lead V2 becomes lead V1R, and leads V3R–

![ECG Diagram](image)

Fig. 26. STEMI in the presence of LBBB. The previous ECG (not shown) had LBBB with typical discordant ST segments and T waves. There is now concordant STE and upright T waves in leads I, aVL, V5, and V6. There is concordant reciprocal STD in the inferior leads II, III, and aVF. LAD occlusion was opened with percutaneous coronary intervention Reprinted with permission from Chan TC, Brady WJ, Harrigan RA, et al. ECG in emergency medicine and acute care. Fig. 26-B. Elsevier; 2005.
V6R are placed across the right chest in mirror image to their left precordial counterparts (ie, V3–V6). Q waves across the right-sided ECG are normal. Absence of STE in leads V2R–V7R nearly rules out RV AMI. One millimeter of STE in lead V4R alone has a sensitivity and predictive accuracy for RV infarction of 93% [155,160]. STE up to 0.6 mm in V4R may be normal; however, in the context of inferior AMI, STE > 0.5 mm should be interpreted as RV AMI (see Fig. 23) [161,162].

Right and left bundle branch block
AMI with associated RBBB or LBBB is greatly under treated with reperfusion therapy [163]. BBB is associated with a high mortality (8.7%) compared with normal conduction (3.5%), especially if persistent (20%) versus transient (5.6%). Mortality with persistent LBBB was 36% versus 12% for RBBB; both were associated with LAD occlusion in approximately 50% of cases [164].

In RBBB, the ST segment is, by general consensus and by electrophysiologic theory, as reliable as it is in normal conduction. Assessment of ST segment amplitude (ie, of STE), however, may be hindered by difficulty in determining where the QRS complex ends and the ST segment begins. To do so, examine other leads to find the true QRS complex duration, and then compare that millisecond measurement within the lead in question. The J point is then at the end of this measured QRS. The T wave in RBBB usually is inverted in leads with an rSR’ (right precordial leads), often with up to 1 mm of STD, especially in lead V2. This should not be mistaken for primary STD. Because of this STD secondary to RBBB, minimal STE may represent a large delta ST (Fig. 25); comparison with a previous ECG may be invaluable. Finally, RBBB in the presence of LV aneurysm may present with a QR wave and STE that mimics acute MI.

The ST segment/T-wave complex in uncomplicated LBBB is opposite in direction (discordant) to most of the QRS complex. To the uninitiated, this normal STE may mimic AMI. Furthermore, LBBB also has a reputation for hiding AMI. There is some electrophysiologic rationale for this. The clinical data, however, may have suffered at the hands of the following faulty logic [165]: only 40%–50% of AMI (by CK-MB) in the presence of LBBB have diagnostic criteria as defined by Sgarbossa and colleagues (Box 2) [165–170]. What may be forgotten is that this is also true of normal conduction: approximately 45% of AMI (by CK-MB) in normal conduction manifests diagnostic STE [12–14]. It seems that the Sgarbossa criteria have similar sensitivity and specificity for AMI (as does STE in normal conduction) and are as sensitive and specific for detection of ongoing epicardial coronary occlusion that requires emergent reperfusion therapy (ie, STEMI). Nevertheless, until there are more data, it is prudent to also treat patients who have high suspicion of AMI and new LBBB with reperfusion therapy, even in the absence of Sgarbossa criteria. Additionally, comparison with a previous ECG
and serial ECGs are useful for identifying coronary occlusion in the presence of LBBB [34,168,171,172] (Fig. 26).

References


Bayley RH, LaDue JS, York DJ. Electrocardiographic changes (local ventricular ischemia and injury) produced in the dog by temporary occlusion of a coronary artery, showing a new stage in the evolution of myocardial infarction. Am Heart J 1944;27:164–9.


Bayley RH, LaDue JS, York DJ. Electrocardiographic changes (local ventricular ischemia and injury) produced in the dog by temporary occlusion of a coronary artery, showing a new stage in the evolution of myocardial infarction. Am Heart J 1944;27:164–9.


Massel D, Dawdy JA, Melendez LJ. Strict reliance on a computer algorithm or measurable ST segment criteria may lead to errors in thrombolytic therapy eligibility. Am Heart J 2000;140:221–6.


[61] TIMI III A. Early effects of tissue-type plasminogen activator added to conventional therapy on the culprit coronary lesion in patients presenting with ischemic cardiac pain at rest. Results of the Thrombolysis in Myocardial Ischemia TIMI (Thrombolysis in Myocardial Infarction) IIIA trial. Circulation 1993;87:38–52.


