Diagnostics

The use of a 4-step algorithm in the electrocardiographic diagnosis of ST-segment elevation myocardial infarction by novice interpreters

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Abstract The electrocardiographic (ECG) diagnosis of ST-segment elevation myocardial infarction (STEMI) represents a challenge to all health care providers, particularly so for the novice ECG interpreter. We have developed—and present in this article—a 4-step algorithm that will detect STEMI in most instances in the prehospital and other nonemergency department (ED) settings. The algorithm should be used in adult patients with chest pain or equivalent presentation who are suspected of STEMI. It inquires as to the presence of ST-segment elevation as well as the presence of STEMI confounding/mimicking patterns; the algorithm also makes use of reciprocal ST-segment depression as an adjunct in the ECG diagnosis of STEMI. If STEMI is detected by this algorithm, then management decisions can be made based upon this ECG diagnosis. If STEMI is not detected using this algorithm, then we can only note that STEMI is not “ruled in”; importantly, STEMI is not “ruled out.” In fact, more expert interpretation of the ECG will be possible once the patient (and/or the ECG) arrive in the ED where ECG review can be made with the more complex interpretation used by expert physician interpreters.

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1. Introduction

Ischemic heart disease describes an entire spectrum of illness, ranging from acute to chronic entities related to coronary artery disease, including angina pectoris, acute myocardial infarction (AMI), cardiomyopathy, and malignant dysrhythmia. Acute coronary syndromes (ACS) have been defined as unstable angina and AMI. Acute myocardial infarction is separated into infarction with ST-segment elevation (STEMI) and infarction without elevation of the ST segments (non-STEMI or nSTEMI). ST-segment elevation myocardial infarction is the more severe form of AMI and typically occurs when there is an atherosclerotic plaque rupture and subsequent thrombus formation and accompanying vasospasm [1]. Approximately 935 000 people in the United States experience an AMI every year with approximately one third of these infarctions being STEMI [2]. The American Heart Association Guidelines 2010 recommend reducing time to reperfusion treatment through rapid identification of STEMI via a number of different systems of care, including the use of the prehospital 12-lead electrocardiogram (ECG). Common medical opinion along with many investigations have shown that prearrival identification of ST-segment elevation (STE) AMI via the emergency medical services (EMS)-performed 12-lead ECG greatly reduces the time to reperfusion therapy with markedly lower mortality rates, including both lower 30-day and 5-year mortality rates (7.3% vs 15.3% and 11.6% vs...
In addition, between 1990 and 2006, the median door to therapy time for patients with STEMI among 2157 hospitals fell from 59 to 29 minutes and correlated with a decline in mortality from 7.0% in 1994 to 6.0% in 2006. Furthermore, for patients undergoing primary percutaneous coronary intervention, the door to effective intervention time decreased from 111 minutes in 1994 to 79 minutes in 2006. This time reduction corresponded to a decline in mortality from 8.6% to 3.1%. This reduction in time to therapy, regardless of the mode of reperfusion treatment used, is largely due to prehospital identification of STEMI and prearrival notification of the event.

A very important point regarding the clinical management of the STEMI is the time-sensitive nature of the therapy and relation to outcome. In a very basic sense, shorter times to reperfusion therapy are associated with markedly better outcomes, including reduced morbidity and mortality. Early detection and treatment of a STEMI has the potential to limit the myocardial damage and acute cardiovascular complications, reduce mortality, and improve the quality of life.

The ECG diagnosis of STEMI represents a challenge to all health care providers, particularly so for the novice ECG interpreter. We have developed—a 4-step algorithm that will detect STEMI in most instances in the prehospital and other nonemergency department (ED) settings. The algorithm should be used in adult patients with chest pain or equivalent presentation who are suspected of STEMI. If STEMI is detected by this algorithm, then management decisions can be made based upon this ECG diagnosis. If STEMI is not detected using this algorithm, then we can only note that STEMI is not “ruled in”; importantly, STEMI is not “ruled out.” In fact, more expert interpretation of the ECG should follow in a time-appropriate fashion.

Fig. 1 A, ST-segment elevation in a single ECG lead consistent with STEMI. B, Inferior wall STEMI with STE in leads II, III, and aVF.
Fig. 2  A, The LVH by voltage pattern with strain. Significantly large QRS complexes are seen on this ECG with very prominent negative (i.e., Q waves) waveforms in leads V1 through V3 and large positive (i.e., R waves) waveforms in leads V4 to V6. B, When evaluating for the LVH by voltage pattern, one can use the Sokolow-Lyon criteria. This approach considers the amplitude of the various components of the QRS complex in leads V1/V2 and V5/V6. In our algorithm, we have used this approach but restyled the question to ask if the LVH pattern is absent. To use this approach, the clinician notes the negative QRS complex (Q wave) and positive QRS complex (R wave) in leads V1/V2 and V5/V6 sections, respectively. If the amplitude summation of these 2 structures is less than 35 mm, then the ECG diagnosis of the LVH using voltage pattern is unlikely.
2. The ECG diagnosis of STEMI

The 12-lead ECG plays a pivotal role in the early diagnosis of STEMI [8]. Not only rapid but also accurate interpretation of the ECG is, of course, mandatory for this diagnosis, yet such interpretation is a skill requiring significant experience. The many challenges involved in ECG interpretation in the setting of possible STEMI include subtle pattern detection as well as the numerous causes of STEMI mimics (i.e., ECG entities causing STE that are not related to STEMI). The differentiation of STE resulting from acute STE infarction from STE due to other, noninfarction etiologies is a vital component of early AMI diagnosis and management.

One study in an academic ED demonstrated the challenge of STE differentiation and STEMI diagnosis by emergency physicians. In this study, practicing emergency physicians misinterpreted STE at a rate of 12 (2.9%) of 202 patients [9]. In this study, the approximate 3% error rate includes both the missed diagnosis of STEMI and the incorrect diagnosis of STEMI when it was not present. Of course, a missed diagnosis of STEMI is associated with a missed opportunity for emergent reperfusion with related worsened outcome. And, conversely, the incorrect diagnosis of STEMI can potentially subject the patient to unnecessary risk of invasive management.

Simple identification of STE on the 12-lead ECG is insufficient to diagnose STEMI as several commonly encountered non-AMI ECG entities demonstrate STE. In the prehospital population, STEMI is not the most common cause of STE. In fact, Otto and Aufderheide noted in one study that 51% (63/123) of patients whose prehospital ECG had 1 mm or more STE were not experiencing AMI. In this study, the left ventricular hypertrophy (LVH) by voltage and the left bundle branch block (LBBB) patterns were the most common causes of the STE in the prehospital chest pain patient population [10]. Also, in one retrospective study of an ED chest pain population, of 202 patients who had STE of at least 1 to 2 mm in magnitude oriented in 2 anatomically contiguous ECG leads, only 15% were diagnosed with STEMI. The most common causes of non-STEMI STE included LVH (25%), LBBB (15%), benign early repolarization (BER, 12%), and right bundle branch block (5%) [11].

Thus, the ECG diagnosis of STEMI is quite a challenge. A standardized approach to the ECG diagnosis of STEMI has

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**Fig. 3** Two ECG patterns with significantly widened QRS complexes. In both cases, note the markedly widened QRS complexes. A, Left bundle branch block; B, ventricular paced pattern.
the potential to not only increase the efficiency of evaluation of such patients (i.e., hasten the time to reperfusion therapy) but also reduce the rate of misdiagnosis (i.e., incorrect and/or missed diagnosis of STEMI). Unfortunately, a single standardized approach to STEMI diagnosis is not found in the literature. In the past, we have recommended the use of various flow charts and decision tools in this task [12]; unfortunately, the current decision tools are cumbersome and difficult to apply in the acute setting. In an attempt to facilitate the prehospital recognition of STEMI, we have developed a 4-step algorithm aimed at not only diagnosing STEMI but also ruling out the more common causes of non-STEMI ST-segment elevation. By its design, the algorithm is specific for ruling in STEMI, yet its ability to rule out STE AMI is likely limited. Such an algorithm, with a higher specificity and somewhat lower sensitivity, would be ideal for the prehospital environment. In this attempt, the EMS provider, perhaps with a real-time medical control physician review of the ECG, will diagnose most STEMI presentations, yet a certain minority of STEMI will be missed using such an algorithm in the prehospital setting. However, these missed STEMI will hopefully be diagnosed once the patient arrives in the ED with “more expert” ECG interpretation possible. Thus, an algorithmic approach to the interpretation of the ECG aimed at early STEMI detection is ideal to reduce error and to insure consistent, accurate, and rapid identification of STEMI. This approach would provide the opportunity for earlier reperfusion therapy.

3. Four-step ECG algorithm for the detection of STEMI

This algorithm will detect STEMI in most instances in the prehospital and other non-ED settings. If STEMI is detected using this algorithm, then management decisions can be made based upon this ECG diagnosis. If STEMI is not detected using this algorithm, then we can only note that STEMI is not “ruled
More expert interpretation of the ECG will be possible once the patient (and/or the ECG) arrive in the ED. At this time, initial ECG review can be made using the more complex interpretation used by physician interpreters. In addition, repeat ECGs can be used if the diagnosis is in question.

The algorithm that we have developed for STEMI recognition involves 4 simple steps:

1. Is there STE of at least 1 to 2 mm in 2 anatomically oriented leads (Fig. 1)?
2. Is sum of the Q wave in lead V1/V2 + R wave in lead V5/V6 less than 35 mm (Fig. 2A and B)?
3. Is the QRS complex less than 0.12 second in width (Fig. 3A and B)?
4. Is there ST-segment depression present in at least 1 lead (Fig. 4)?

Each step is presented in the form of a question. An affirmative, or YES, answer to each question leads the clinician to the next question. If all 4 questions are answered in the affirmative, then STEMI is theorized to be present to a very high degree of certainty. A negative, or NO, answer to any question will halt the use of the algorithm and place the diagnosis of STEMI in some degree of doubt.

A brief description of the algorithm, with the intent of each question, is as follows:

1. The diagnosis of STEMI is, of course, dependent upon the presence of STE of at least 1 to 2 mm in magnitude and occurring in at least 2 anatomically oriented ECG leads (Fig. 1). This question simply notes the presence of STE yet because of the multitude of non-STEMI causes of STE, we must proceed further to evaluate this STE—and determine if it results from STEMI.
2. Now that we have established that a potentially concerning pattern of STE is present, we must determine if the diagnosis is STEMI. This question is aimed at the detection of the LVH by voltage pattern, inquiring about the size of the QRS complexes in leads V1 and V6 (Fig. 2A). If the QRS complexes are greater than the noted size, then the LVH by voltage pattern is potentially present; it is recalled that this finding is frequently associated with significant ST-segment changes, including STE. On the contrary, if the QRS complexes are of a normal size, then question is answered to the affirmative, indicating most likely that the LVH pattern (as diagnosed by voltage) is not present. In this instance, the observed STE does not result from LVH-related repolarization abnormality—and we have now ruled out the most common cause of non-STEMI ST-segment elevation from consideration on the ECG.
3. This question inquires as to the width of the QRS complex; it asks if the QRS complexes are less than a specific width. If the QRS complexes are greater in duration than the noted width, then the answer to this question is NO and abnormal patterns, such as bundle branch block (BBB) and ventricular paced patterns are likely, among other entities (Fig. 3A and B). If such patterns are present, then related repolarization abnormalities, including significant STE, are encountered. Thus, the STE probably results from the repolarization abnormalities encountered with these patterns. Conversely, if the QRS complexes are normal in width, then the answer to this question is YES, indicating that the observed STE does not result from either BBB or ventricular-paced patterns; we can now rule out significant, non-STEMI causes of STE.
4. The final question focuses on ST-segment depression also occurring on the ECG. At this point, we have excluded several of the most frequent causes of non-STEMI ST-segment elevation. Of the common causes of STE remaining, we have STEMI, BER, acute pericarditis, and left ventricular aneurysm left to consider. Here, we will use the presence of reciprocal ST-segment depression (Fig. 4) to rule in STEMI. If present, reciprocal change strongly supports the ECG diagnosis of STEMI. Unfortunately, not all STEMI presentations will demonstrate reciprocal ST-segment depression. These STEMI presentations lacking reciprocal change will be missed if we rely solely on the algorithm. Although this is not favorable, we must realize that this interpretation is occurring early in the patient’s course; the patient will receive immediate
emergency physician interpretation of the ECG upon arrival at the ED—and STEMI will likely be diagnosed in the ED.

4. A consideration of the 4 steps

A more in-depth explanation of the algorithm, and its various steps, is included in the following:

No. 1: Is there STE of at least 1 to 2 mm in 2 anatomically oriented leads? The first step in the algorithm is simply to determine if there is STE (Fig. 1A) of at least 1 to 2 mm in at least two anatomically oriented leads. The 12-lead ECG in Fig. 1B demonstrates an inferior STEMI with ST-segment elevation in leads II, III, and aVF—in this case, 3 leads in the same inferior anatomical region.

In years past, patients complaining of chest pain who demonstrated STE were considered to have an AMI and were potentially offered reperfusion therapy in the form of fibrinolysis. However, it is now apparent that there are many causes of ST elevation besides STEMI. Left bundle branch block, LVH, and left ventricular aneurysm both occur in patients with coronary artery disease (CAD) and can mimic STEMI on the ECG. Benign early repolarization and acute pericarditis can also resemble STEMI but are not associated with CAD—yet these patterns are relatively common in the prehospital and ED chest pain populations [10,11]. Refer to Fig. 5 for a depiction of the frequency of STE types among patients with prehospital and ED chest pain.

Therefore, if there is ST elevation of at least 1 to 2 mm in at least 2 anatomically oriented leads, the clinician can answer “YES” to this step and move on to the next 2 questions to eliminate several of the common causes of ST elevation other than STEMI.

No. 2: Is the Q wave in lead V1/V2 + R wave in lead V5/V6 less than 35 mm? The second step in the algorithm evaluates the amplitude, or size, of the QRS complex. If the size of the largest Q wave in either lead V1 or V2 is added to the amplitude of the largest R wave in either V5 or V6 and the summation is less than 35 mm, then the diagnosis of LVH is unlikely. If the sum is less than 35 mV, then the clinician may answer “YES” to this step (Fig. 2B).

Many models, with varying specificity and sensitivity, exist for the diagnosis of LVH using the ECG. However, these models exist to diagnose underlying LVH—not to identify secondary repolarization abnormalities due to LVH [13]. Given the complexity of some of these models, we chose to use the Sokolow-Lyon criteria for our analysis [14]. If the size of the largest Q wave in either lead V1 or V2 is added to the size of the largest R wave in either V5 or V6 and the total is smaller than 35 mm, then the diagnosis of LVH is unlikely. For simplicity, we chose to ignore the modified...
Sokolow-Lyon criterion that uses lead aVR as well as other LVH ECG decision tools. Advanced providers may choose to consider other LVH criterion.

Why should the clinician be concerned about the size of the QRS complex when one is reviewing the ST segment? First, the ECG LVH pattern and the related repolarization changes are frequently encountered in the ED; in fact, the ECG LVH pattern is a frequently seen cause of STE (Fig. 5) in patients with ED chest pain [11]. Second, the ECG abnormalities associated with LVH are not infrequently mistaken for ACS, including STEMI. The repolarization changes resulting from the LVH pattern alter the morphology of the ST segment and/or the T wave (Fig. 2A); not only does the LVH with strain pattern mimic STEMI but it also hinders, or reduces, the ECG’s ability to detect acute coronary ischemic changes.

The first step in the consideration of the LVH-related ECG changes is the detection of the LVH by voltage pattern. The LVH pattern produces large Q (or S) waves in the anterior precordial leads (V₁ through V₃) and large R waves in lateral precordial leads (V₅ and V₆). Once this pattern is noted, then an awareness of its impact on the ECG evaluation of the chest pain patient can occur.

Approximately 75% of patients with the LVH by voltage pattern demonstrate the “strain pattern”; the strain pattern includes significant ST-segment changes (elevation and depression) and T-wave abnormalities (prominent T waves and T-wave inversion). The LVH with strain pattern is associated with poor R-wave progression, most commonly producing a QS pattern; these complexes are located in leads V₁, V₂, and V₃. ST-segment elevation is encountered in this distribution along with prominent T waves. The STE seen in this distribution may be greater than 5 mm in height, mimicking acute anterior wall STEMI. The initial, upsloping portion of the ST segment–T wave complex is frequently concave in LVH compared with the either flattened or convex pattern observed in the patient with AMI. The lateral leads, leads I, aVL, V₄, and V₆, demonstrate large, prominent, positively oriented QRS complexes with marked ST-segment depression and T-wave inversion—again, consistent with ACS-related change. In a general sense, the ST segment–T wave abnormalities can be predicted based upon the direction of the QRS complex. The ST segment–T wave complex is directed opposite from the polarity of the QRS complex. The anterior leads demonstrate largely negatively oriented QRS complexes; in these leads, the ST segment is elevated and the T wave is upright and at times rather prominent. The lateral leads display positively oriented QRS complexes; in these leads, ST-segment depression and inverted T waves are seen. Refer to Fig. 2A

Applying the algorithm to this ECG, the answer to question no. 1 is YES; thus, clinician is directed to questions no. 2, no. 3, and no. 4, which all have YES answers. In this case using the algorithm, the diagnosis of STEMI is made. This ECG demonstrates an inferolateral STEMI with reciprocal change in the lateral leads.
for an example of the ST-segment changes encountered in the patient with the LVH by voltage ECG pattern.

With regard to question no. 2, if the LVH pattern is present (i.e., the summation of the QRS complex forces is greater than 35 mV), then the observed ST-segment changes (in this case, STE) can result from either LVH with strain or STEMI. Conversely, if the sum is less than 35 mV, then the LVH pattern is probably not present; the observed ST-segment changes then could result from STEMI or other non-AMI STE etiologies. Further investigation regarding the remaining differential is performed by proceeding to questions no. 3 and no. 4.

No. 3: Is the QRS complex less than 0.12 second in width? The third step in the algorithm is to evaluate the width of the QRS complex. The absence of a widened QRS complex excludes both bundle branch block and ventricular paced patterns as the cause of the STE. However, if the QRS complex is wide, then the observed STE could result from either the bundle branch block or ventricular paced patterns—or STEMI.

As with the LVH with strain pattern discussed earlier, both bundle branch block and ventricular paced patterns confound the ECG’s ability to detect changes related to ACS. Of these patterns, LBBB is the most widely studied. In this context, LBBB markedly reduces the diagnostic power of the ECG. Not only does LBBB confound the ECG’s ability to detect ACS but also it mimics the ECG manifestations of ACS.

The ECG in the LBBB pattern is noted with a widened QRS complex, usually greater than 0.12 seconds in duration; large, monophasic R waves are seen in the lateral leads (leads I, aVL, V5, and V6), whereas prominent QS complexes are seen in the right precordial leads (leads V1 and V2). As noted earlier, the ECG demonstrates altered ST-segment and T-wave configurations; importantly, these expected ST-segment configurations are discordant, directed opposite from the terminal portion of the QRS complex. Thus, leads with prominent, positively oriented QRS complexes will demonstrate ST-segment depression with T-wave inversion; conversely, those leads with prominent, negative QRS complexes will be associated with STE and large, upright T waves. The right precordial leads with either QS or rS complexes may have markedly elevated ST segments and tall, vaulting T waves. The lateral leads, with the large monophasic R wave, demonstrate ST-segment depression with T waves that are frequently inverted. Refer to Fig. 3A for an example of the LBBB pattern and related ST-segment abnormalities.

As with the LBBB pattern, the right ventricular paced rhythm (VPR) pattern may both mimic and mask ACS. The ECG demonstrates a broad, mainly negative QS or rS complex in leads V1 to V6 with either poor R-wave progression or QS complexes. A large monophasic R wave is encountered in the lateral leads I and aVL. Leads V5 and V6 may demonstrate either a QS or monophasic R-wave
complex. QS complexes may also be encountered in the inferior leads. As with the LBBB pattern, the anticipated or expected ST segment–T wave configurations are discordant, directed opposite from the terminal portion of the QRS complex. Refer to Fig. 3B for an example of an implanted paced ECG pattern.

With regard to question no. 3, if the LBBB or VPR are present (i.e., the QRS complexes are of abnormal width), the observed ST-segment changes (in this case, STE) can result from either bundle branch block, ventricular paced pattern, or STEMI. Conversely, if the QRS complex is of normal width, then these patterns are likely not present; the observed ST-segment changes then could result from STEMI or other non-AMI STE etiologies. At this point in the algorithm, we have excluded the most frequent causes of non-STEMI ST-segment elevation in the patient with chest pain—namely LVH, LBBB, and VPR. Thus, STEMI, benign early repolarization, acute pericarditis, and other less common entities remain in the differential consideration. We will now use the presence of reciprocal ST-segment depression in question no. 4 to rule in STEMI.

No. 4: Is there ST-segment depression present in at least 1 lead? ST-segment depression with coexisting STE resulting from STEMI is termed reciprocal ST-segment depression, or reciprocal change (Fig. 4). The presence of ST-segment depression of at least 1 mm that is distant from the ST elevation increases the diagnostic accuracy of STEMI. The presence of reciprocal change on an ECG with STE, especially after eliminating confounding patterns such as LVH and LBBB, provides very strong evidence that STEMI is present. Benign early repolarization is an example of a pattern that is not associated with reciprocal ST-segment depression. Thus, if this is present, it is strongly suggestive of ischemia as the cause for ST elevation [15]. Once we have eliminated all ECGs that do not meet the criteria for steps no. 1 to no. 3 in the algorithm, finding reciprocal change on an

![LVH Pattern](image)

**Fig. 9** Applying the algorithm to this ECG, the answer to question no. 1 is YES; thus, the clinician is directed to the next question. The answer to question no. 2 is NO; thus, the algorithm is no longer applicable. The diagnosis of STEMI is not made using the algorithm. This ECG demonstrates the LVH using voltage pattern and no evidence of STEMI.
ECG leads to a specificity of 93% and a positive predictive value of 93% that the patient is experiencing STEMI [16]. However, if all patients with chest pain and ST elevation are considered (and steps 2 and 3 are eliminated), the presence of reciprocal change does not help to differentiate between AMI and non-AMI causes of STE (specificity is 34% and positive predictive value is 30%). Thus, once LVH and BBB are eliminated, reciprocal change helps to differentiate between AMI and non-AMI causes of STE [16]. Conditions such as LVH, RVPR, and BBB can all produce conduction abnormalities that have both ST depression and ST elevation. However, most conditions would elicit a negative answer to questions no. 2 and no. 3 and thus be eliminated from the algorithm.

In patients who do not meet all of the criteria of the algorithm, STEMI can still exist, but further analysis is needed. This statement is true in a number of different situations. First, patients can experience NSTEMI. This diagnosis is established using the presence of a symptom (usually chest pain or dyspnea), ECG abnormality (excluding STE), and positive serum markers (with the typical rise and fall pattern of AMI). In that STE is not seen in these patients, the algorithm would not apply and thus would not be used in such patient evaluations.

Second, as noted earlier, 3 ECG patterns (LVH, LBBB, and VPR) confound the ECG diagnosis of STEMI. In fact, not only do these patterns conceal ischemic ECG abnormalities but also they produce ST-segment and T-wave abnormalities that can mimic ACS-related changes. From the perspective of confounding the ECG evaluation of STEMI, the Sgarbossa criteria [17] can assist to a limited extent the diagnosis of AMI with either LBBB or VPR present. Yet, such patterns markedly limit the value of the ECG, and thus the clinician’s ability to detect ACS in general and STEMI in particular. These are complicated ECG presentations requiring more expert ECG interpretation via the emergency physician, intensivist, and cardiologist.

Last, the algorithm will probably miss the diagnosis of STEMI when reciprocal ST-segment depression is not encountered. Reciprocal ST-segment depression is seen in

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**Fig. 10** Applying the algorithm to this ECG, the answers to questions no. 1 and no. 2 are YES; thus, the clinician is directed to question no. 3. The answer to question no. 3 is NO; thus, the algorithm is no longer applicable. The diagnosis of STEMI is not made using the algorithm. This ECG demonstrates a LBBB pattern and no evidence of STEMI.
Applying the algorithm to this ECG, the answers to questions no. 1, no. 2, and no. 3 are YES; thus, the clinician is directed to question no. 4. No ST-segment depression is noted; thus, reciprocal change is not present—the answer to question no. 4 is NO. The diagnosis of STEMI is not possible using the algorithm despite the obvious appearance of a STEMI. The STEMI without reciprocal change is one shortcoming of this algorithm.

Applying the algorithm to this ECG, the answers to questions no. 1, no. 2, and no. 3 are YES; thus, the clinician is directed to question no. 4. No ST-segment depression is noted; thus, reciprocal change is not present—the answer to question no. 4 is NO. The diagnosis of STEMI is not possible using the algorithm that, in this case, is appropriate. Benign early repolarization is the ECG diagnosis responsible for the STE. ST-segment elevation myocardial infarction is not present.
most STEMI presentations, yet not all. In fact, inferior STEMI most often demonstrates reciprocal ST-segment depression, whereas anterior STEMI is associated less often with such ST-segment depression. The STEMI presentations lacking reciprocal ST depression will be missed if the algorithm is the sole interpretative tool used in the prehospital environment. Such a “temporary miss” is acceptable, assuming that interpretation by an ED physician follows soon after hospital arrival. Of course, many EMS providers are skilled at ECG interpretation and will recognize STEMI despite the absence of reciprocal STE.

One asks the question “why consider reciprocal ST segment depression in this setting if we will miss certain STEMI patterns?” This query is quite valid as is the question in step 4. If we include all STE presentations after the use of steps 1, 2, and 3, we will have excluded common causes of non-STEMI STE, yet we will still encounter the possibility of BER and pericarditis, among other non-AMI entities. If we desire a specific algorithm for this “first-pass” ECG review, step 4 is necessary. We certainly do not desire to diagnose STEMI when BER or pericarditis is present.

5. Illustrative ECG cases

Please refer to Figs. 6 to 13 for illustrative cases of ECG interpretation using this 4-step algorithm. In Fig. 6, the ECG is normal. ST-segment elevation is not noted thus the algorithm does not apply. ST-segment elevation myocardial infarction is not present. In Figs. 7 and 8, STEMI with reciprocal change is present thus answers to all questions are YES—the diagnosis of STEMI is possible and correct. Figs. 9 and 10 demonstrate non-STEMI causes of STE—LVH by voltage and LBBB patterns, respectively. The algorithm does not suggest STEMI and that is appropriate in that ECG STEMI is not present in either case. Fig. 11 illustrates the primary shortcoming of this algorithm—STEMI without reciprocal change. Although the algorithm does not rule out

**Acute Pericarditis**

Fig. 13 Applying the algorithm to this ECG, the answers to questions no. 1, no. 2, and no. 3 are YES; thus, the clinician is directed to question no. 4. No ST-segment depression is noted; thus, reciprocal change is not present—the answer to question no. 4 is NO. The diagnosis of STEMI is not possible using the algorithm that, in this case like in Fig. 12, is also appropriate. Acute pericarditis (or myopericarditis) is the ECG diagnosis responsible for the STE. ST-segment elevation myocardial infarction is not present.
STEMI, the algorithm is also not able to rule-in acute infarction due to the absence of reciprocal change. Figs. 12 and 13, involving benign early repolarization and acute pericarditis, respectively, do not demonstrate STEMI as noted by the algorithm nor any traditional methods of interpretation. Thus, the algorithm in these 2 instances functions appropriately and leads the interpreter away from the diagnosis of STEMI in the setting of a STEMI mimic.

6. Conclusions

This algorithm will probably detect most STEMI presentations—which probably represent the majority. The interpreter, however, must also note that the algorithm will probably not detect certain STEMI presentations, such as those STEMI patterns without reciprocal change or the more complex patterns such as STEMI with the confounding patterns. Also, this algorithm provides the novice or less experienced interpreter with the ability to detect most STEMI presentations at an early stage in the process. Furthermore, the algorithm would also reduce the “false-positive” STEMI activation. The algorithm would be of value in the prehospital setting as well as in the hands of other less experienced ECG interpreters, such as nonemergency medicine, noncardiologist, noncritical care clinicians. The STEMI presentations that will be missed by the algorithm will hopefully be detected by the “more expert” interpreter once either the ECG or the patient arrives in the ED.

References