Diagnosis of ST-Elevation Myocardial Infarction in the Presence of Left Bundle Branch Block With the ST-Elevation to S-Wave Ratio in a Modified Sgarbossa Rule

Stephen W. Smith, MD, Kenneth W. Dodd, MD, Timothy D. Henry, MD, David M. Dvorak, MD, Lesly A. Pearce, MS

From the Hennepin County Medical Center, Minneapolis, MN (Smith, Dodd); the University of Minnesota School of Medicine, Minneapolis, MN (Smith); the Minneapolis Heart Institute, Abbott Northwestern Hospital, Minneapolis, MN (Henry); the Fairview Southdale Hospital, Edina, MN (Dvorak); and Biostatistical Consulting, Minot, ND (Pearce).

Study objective: Sgarbossa's rule, proposed for the diagnosis of acute myocardial infarction in the presence of left bundle branch block, has had suboptimal diagnostic utility. We hypothesize that a revised rule, in which the third Sgarbossa component (excessively discordant ST-segment elevation as defined by ≥5 mm of ST-segment elevation in the setting of a negative QRS) is replaced by one defined proportionally by ST-segment elevation to S-wave depth (ST/S ratio), will have better diagnostic utility for ST-segment elevation myocardial infarction (STEMI) equivalent, using documented coronary occlusion on angiography as reference standard.

Methods: We collected admission ECGs for all patients with an acutely occluded coronary artery and left bundle branch block at 3 institutions. The ECGs of emergency department patients with chest pain or dyspnea and left bundle branch block, but without coronary occlusion, were used as controls. The R or S wave, whichever was most prominent, and ST segments, relative to the PR segment, were measured to the nearest 0.5 mm. The ST/S ratio was calculated for each lead that has both discordant ST deviation of greater than or equal to 1 mm and an R or S wave of opposite polarity; others were set to 0. The cut point for the most negative ST/S ratio with at least 90% specificity was determined. The revised rule is unweighted, requiring just 1 of 3 criteria. Diagnostic utilities of the original and revised Sgarbossa rules were computed and compared. McNemar's test was used to compare sensitivities and specificities.

Results: The study and control groups included 33 and 129 ECGs, respectively. The cut point selected for relative discordant ST-segment elevation was −0.25. Excessive absolute discordant ST-segment elevation of 5 mm was present in at least one lead in 30% of ECGs in patients with confirmed coronary occlusion versus 9% of the control group, whereas excessive relative discordant ST-segment elevation less than −0.25 was present in 58% versus 8%. Sensitivity of the revised rule in which ST-segment elevation with an ST/S ratio less than or equal to −0.25 replaces ST-segment elevation greater than or equal to 5 mm was significantly greater than either the weighted (*P*<.001) or unweighted (*P*=.008) Sgarbossa rule: 91% (95% confidence interval [CI] 76% to 98%) versus 52% (95% CI 34% to 69%) versus 67% (95% CI 48% to 82%). Specificity of the revised rule was lower than that of the weighted rule (*P*=.002) and similar to that of the unweighted rule (*P*=1.0): 90% (95% CI 83% to 95%) versus 98% (95% CI 93% to 100%) versus 90% (95% CI 83% to 95%). Positive and negative likelihood ratios for the revised rule were 9.0 (95% CI 8.0 to 10) and 0.1 (95% CI 0.03 to 0.3). The revised rule was significantly more accurate than both the weighted (16% difference; 95% CI 5% to 27%) and unweighted (12% difference; 95% CI 2% to 22%) Sgarbossa rules.

Conclusion: Replacement of the absolute ST-elevation measurement of greater than or equal to 5 mm in the third component of the Sgarbossa rule with an ST/S ratio less than −0.25 greatly improves diagnostic utility of the rule for STEMI. An unweighted rule using this criterion resulted in excellent prediction for acute coronary occlusion. [Ann Emerg Med. 2012;60:766-776.]

Please see page 767 for the Editor's Capsule Summary of this article.

A **feedback** survey is available with each research article published on the Web at www.annemergmed.com.
A **podcast** for this article is available at www.annemergmed.com.

INTRODUCTION

Timely and accurate identification of acute coronary occlusion in the setting of ischemic symptoms is critical to initiating urgent angiography and appropriate reperfusion therapy. Although the increase or decrease of cardiac biomarker levels is essential to the diagnosis of acute myocardial infarction, positive biomarker results alone do not differentiate ST-elevation myocardial infarction (STEMI) from non-STEMI. ST

Editor's Capsule Summary

What is already known on this topic

Diagnosis of ST-segment elevation myocardial infarction in the setting of a left bundle branch block is difficult.

What question this study addressed

Whether changing one component of the Sgarbossa rule from an absolute (>5-mm discordant ST elevation) to a proportional criterion (any ST-segment to S-wave ratio less than −0.25, with at least 1 mm ST elevation) improves prediction of acute coronary occlusion.

What this study adds to our knowledge

The revised rule, developed with 162 patients with left bundle branch block, 33 of whom had an acute occlusion, was more accurate than the original rule. It has a positive likelihood ratio of 9 and negative likelihood ratio of 0.1.
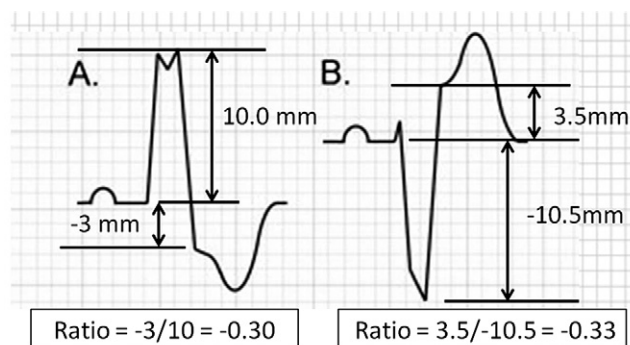
How this is relevant to clinical practice

This rule may not be "user friendly" enough for clinicians unless incorporated into ECG machine interpretations and should be validated in a distinct set of ECGs.

Figure 1. Abnormal, excessive discordance, with the ST segment and T wave in the opposite direction from QRS. Method of measurement: ST segment is measured at the J point, relative to the PR segment. R wave and S wave are also measured relative to the PR segment.

elevation on the ECG is the primary indication for emergency reperfusion therapy; however, identification of STEMI in the setting of left bundle branch block remains challenging.[1]

In the setting of left bundle branch block, ST-segment elevation or ST-segment depression commonly occurs in the absence of acute myocardial infarction and is predictable in that the ST-segment and T-wave abnormalities are normally "discordant" to (in the opposite direction of) the majority of the QRS (Figure 1). "Concordant" is the term used when the ST segment or T wave is in the same direction as the QRS and is not normally observed in baseline (normal) left bundle branch block.

Sgarbossa et al proposed requiring at least 3 points from the following criteria for the diagnosis of acute myocardial infarction in the presence of left bundle branch block: (1) concordant ST-segment elevation of 1 mm (0.1 mV) in at least 1 lead (5 points), (2) concordant ST-segment depression of at least 1 mm in leads V1 to V3 (3 points), or (3) excessively discordant ST-segment elevation, defined as greater than or equal to 5 mm of ST-segment elevation when the QRS result is negative (2 points)[2] (Figure 2). There have been many evaluations of Sgarbossa's criteria, with variable methodologies and patient populations.[3-16] In a systematic review, although specificity for greater than or equal to 3 Sgarbossa points was high (98%), sensitivity was only 20%.[17] For a score greater than

or equal to 2 (ie, the unweighted rule), the sensitivities in the studies ranged from 20% to 79%, and specificities ranged from 61% to 100%.

Two main issues may contribute to the low sensitivity of Sgarbossa's rule. First, all validating studies cited above used a reference standard of creatine kinase (CK) (with or without MB fraction) for acute myocardial infarction, not coronary occlusion by angiography, meaning non-STEMI (emergency reperfusion therapy unnecessary) and STEMI (emergency reperfusion required) were included in the acute myocardial infarction group. Second, anterior STEMI is most often diagnosed by ST-segment elevation in leads V1 to V4; however, in left bundle branch block, these leads normally already have discordant ST-segment elevation. Therefore, some means of assessment of excessive anterior ST-segment elevation is necessary to diagnose most anterior STEMI. Specifically, Sgarbossa's rule uses an absolute 5-mm cutoff for discordant ST-segment elevation when an ST-segment elevation proportional to the preceding QRS or S wave may be more useful. We sought to evaluate the performance of the Sgarbossa rule in patients with left bundle branch block and angiographic evidence of coronary occlusion. We hypothesized that changing the third component of the Sgarbossa rule to a proportional rule would improve its sensitivity and specificity.

MATERIALS AND METHODS
Study Design and Setting

Data for this study were collected at 3 Minnesota hospitals: Hennepin County Medical Center, a trauma center in Minneapolis; at the Minneapolis Heart Institute at Abbott Northwestern Hospital, which has a large regional STEMI system; and at Fairview Southdale Hospital, a community hospital in suburban Minneapolis that also takes transfers for primary percutaneous coronary intervention (PCI). Institutional review board approval was obtained at all institutions.
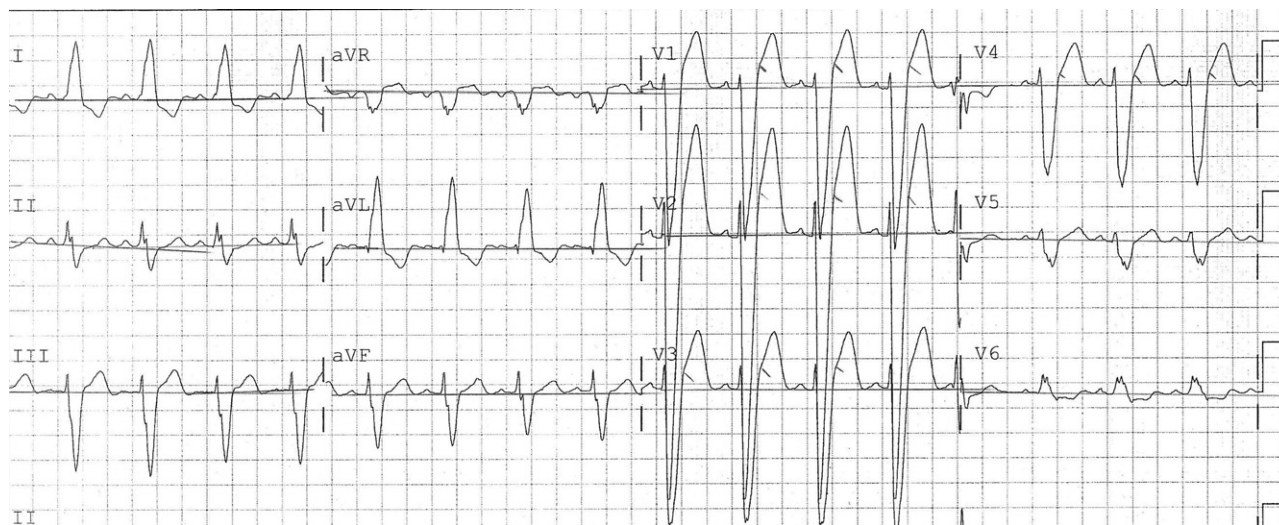
Figure 2. Baseline ECG of a patient with left bundle branch block without any acute myocardial infarction or ischemia; lead V2 shows 7 mm of ST-segment elevation but also a (–)53-mm S wave, for an ST/S ratio of −0.13.

Selection of Participants

ECGs from 2 groups of patients were collected. From patients with left bundle branch block and symptoms of acute myocardial infarction (chest pain, shortness of breath, or both), we searched for a STEMI group with angiographic evidence of occlusion and for a control group with no occlusion. To identify the STEMI group, we did the following: (1) at Hennepin County Medical Center, we crossed the databases (1994 to 2007) of the catheterization and electrocardiography laboratories to find all patients with left bundle branch block who had a coronary angiogram; and (2) at Minneapolis Heart Institute (July 2004 to March 2008) and at Fairview Southdale Hospital (April 2004 to October 2006), we searched the STEMI databases for patients referred for primary angioplasty for possible STEMI with left bundle branch block on the ECG and then reviewed the catheterization reports. Angiographic evidence of occlusion included either occlusion (thrombolysis in myocardial infarction 0 to 1 flow) or stenosis with either thrombosis or ulcerated culprit lesion and peak 24-hour cardiac troponin I level greater than or equal to 10 ng/mL. A cardiac troponin I cutoff of 10 ng/mL is higher than the level used for diagnosis of acute myocardial infarction, which ranged from 0.1 to 0.6 ng/mL during the period. Ten nanograms per milliliter was chosen to include in the STEMI group patients with a culprit lesion but an open artery who might have had coronary occlusion at the time of the ECG but had spontaneous reperfusion by the time of the angiogram because most non-STEMIs (acute myocardial infarctions that are not STEMI) have a peak troponin I level less than 10 ng/mL.[18-20] In the setting of an open infarct-related artery (thrombolysis in myocardial infarction 3 flow), if serial cardiac troponin I testing had been conducted and the peak level was less than 10 ng/mL, we did not classify it as a STEMI.

For the control group, we searched the ECG databases at Hennepin County Medical Center (September 2000 to June 2003) for patients presenting to the emergency department (ED) with left bundle branch block. We included all patients presenting with ischemic symptoms (chest pain or dyspnea) but without acute coronary occlusion. Absence of coronary occlusion was defined as (1) all cardiac troponin I levels being negative within 24 hours; (2) any positive cardiac troponin I level with an angiogram showing either no culprit lesion or a culprit lesion but both no occlusion and peak level of serial cardiac troponin I less than 10 ng/mL; or (3) if no angiogram, an echocardiogram with no wall motion abnormality and peak cardiac troponin I less than 10 ng/mL. There were multiple troponin assays in use during the period, with variable cut points for diagnosis of acute myocardial infarction ranging from 0.1 ng/mL to 0.6 ng/mL.

For both cases and controls, patients with hyperkalemia (potassium >5.5 mEq/L), extreme tachycardia (rate >130 beats/min), severe hypertension (diastolic blood pressure >120 mm Hg), or pulmonary edema with respiratory failure, as defined by need for ventilatory support, were excluded because their ECGs commonly mimic occlusion and these patients would require intensive care, often including catheter laboratory activation, regardless of ST-segment changes.

Left bundle branch block was determined by the overreading cardiologist and included an rS complex in V1, QRS duration greater than or equal to 120 ms, monophasic R in V6, and intrinsicoid deflection of at least 50 ms in V6.

Methods of Measurement

We used the first recorded ECG from each patient's initial presentation. ECG measurements for all patients in the STEMI and control groups were conducted independently by 2 medical students with no ECG reading experience who were trained to

make the measurements by an emergency physician (S.W.S.) and who were blinded to the outcome. Each reader independently chose a representative complex for each of the 12 leads and decided whether the QRS was mostly positive or mostly negative. If mostly positive, then the R-wave amplitude was measured. If mostly negative, the S-wave amplitude was measured as a negative number. ST deviation was measured at the J point because this was the method used by Sgarbossa[21] and is recommended by the American College of Cardiology and American Heart Association.[22] All measurements were to the nearest 0.5 mm (0.05 mV) and relative to the PR segment (Figure 1). Measurements between the 2 readers were then compared: Pearson correlation coefficients were 0.74 to 0.96 for measurements of ST deviation and 0.98 to 0.99 for measurements of QRS. Any measurement that differed by greater than 1.0 mm was reviewed and remeasured by the lead author (S.W.S.), also blinded to the outcome. All discrepancies of this magnitude were due to beat-to-beat variability, wandering baseline, measurement of ectopic beats, and incorrect labeling of equivocal QRS complexes as primarily positive or negative. Such discrepancies were present in 40 of 1,944 (2.1%) measurements of ST deviation and in 157 of 1,944 (8.1%) measurements of QRS complexes, all with large voltage and some beat-to-beat variability. Among the discrepancies of greater than 1.0 mm that were remeasured, fewer were measurements by one of the medical students; therefore, we chose to assess these measurements, with the corrected discrepancies incorporated.

Five distinct rules (I to V) using combinations of 4 different components (a through d) were evaluated (Table 1). All components required at least 1 mm of ST-segment elevation or ST-segment depression. Computation of the cut point for component c-ii required first determining the most negative ST/S ratio (most discordant) in leads that had at least 1 mm ST-segment elevation and a negative QRS measurement. Any lead with less than 1 mm ST-segment elevation or concordance was assigned an ST/S value of 0. A receiver operator characteristic curve was then fit and area under the curve was computed. The cut point from the curve with at least 90% specificity was determined and defined as the optimal cut point. Computation of the cut point for component d was similar, except that discordance was defined as ST-segment elevation combined with a negative QRS result or ST-segment depression combined with a positive QRS result (thus, ST/S values <0 indicated discordance). Any lead with less than 1 mm ST-segment elevation or ST-segment depression or a QRS of 0 was assigned a value of 0. Any lead with concordance had a positive ST/S ratio.

Rules I and II are Sgarbossa's rules, weighted and unweighted, respectively. For the weighted rule (the original Sgarbossa rule), a score of 3 or greater was necessary to diagnose acute myocardial infarction. For the unweighted rule, a score of 2 or greater was used (any one of the 3 criteria positive). Rule III is the same as Sgarbossa's unweighted rule (II), with the

Table 1. Components and diagnostic rules.

Component	Description
a	ST-segment elevation ≥1 mm and concordant with the QRS in at least 1 lead
b	ST-segment depression ≥1 mm in any of leads V1–V3
c	Excessively discordant ST-segment elevation in any one lead
c-i	Absolute as defined by ST-segment elevation ≥5 mm in at least 1 lead
c-ii	Proportional as defined by most negative ratio of ST/S and at least 1 mm of STE
	Result: Cut point for ST/S ratio with >90% specificity determined to be <–0.25
d	Excessively discordant ST-segment deviation (elevation *or* depression) defined by most negative ST/S ratio in any lead with >1 mm ST-segment elevation or depression
	Result: Cut point for ST/S ratio with >90% specificity determined to be <–0.30
Rule	
I	
a, b, c-i	Sgarbossa rule (original; with weighting): >3 points from components a (5 points), b (3 points), c-i (2 points)
II	
a, b, c-i	Sgarbossa Rule without weighting, equivalent to a score >2 points: at least 1 of components a, b, c-i
III	
a, b, c-ii	Modified Sgarbossa rule (no weighting, proportional discordant STE): at least 1 of components a, b, c-ii
IV	
a, b, or d	Modified Sgarbossa rule (no weighting, proportional discordant STE or STD): at least 1 of components a, b, d
V	
d	Overall proportional discordance rule

STE, ST elevation; *STD*, ST-segment depression.

modification of using a relative measure of ST-segment elevation discordance instead of an absolute measure. Rule IV further modifies rule II by using a relative measure of discordant ST deviation, whether ST-segment elevation or ST-segment depression, in place of absolute ST-segment elevation discordance (component d). Last, rule V is based solely on component d (overall proportional discordance of the ECG, whether by ST-segment elevation or ST-segment depression).

Primary Data Analysis

Demographics and ECG characteristics were compared between the acute coronary occlusion and control groups, using a *t* test for continuous measures and χ^2 test for categorical measures. Sensitivity, specificity, positive likelihood ratio, and negative likelihood ratio were computed for each rule. McNemar's rule was used to compare sensitivity and specificity between pairs of rules and 95% confidence intervals (CIs) for differences computed. All tests were 2 sided, and statistical significance was accepted at the .05 level. Statistics were computed with SPSS (version 18.0; SPSS, Inc., Chicago, IL) and MedCalc (version 12.2.1.0; Mariakerke, Belgium).

RESULTS

Characteristics of Study Subjects

At the 3 institutions, we identified 45 patients with acute coronary occlusion, 33 of whom had an ECG available for analysis. Overall, of 33 included cases, 27 had complete occlusion and 6 had incomplete occlusion and maximum cardiac troponin I level of at least 10 ng/mL. The culprit artery was the left anterior descending artery in 20 patients, the right coronary artery in 9, and the circumflex in 4.

A total of 129 patients met criteria for the control group. Of the 323 Hennepin County Medical Center ED patients screened, 117 met entry criteria of ischemic symptoms and left bundle branch block but no coronary occlusion; 12 of these had acute myocardial infarction by biomarkers but no evidence of coronary occlusion. Another 12 controls with acute myocardial infarction but no occlusion were identified through review of Fairview Southdale Hospital catheterization laboratory reports. Thus, of the 129 control patients, 105 had no acute myocardial infarction and 24 had acute myocardial infarction without an acute occlusion. Chest pain was the only presenting symptom in 42 patients, dyspnea only in 49, and both symptoms in 38.

Patients with an acute occlusion and left bundle branch block were older (mean age 73 versus 67 years) and more often men (59% versus 46%) than the controls. At least 1 cardiac troponin I measurement was available for 24 of 27 patients in the STEMI group (median 60.5 ng/mL; interquartile range 16 to 121): the peak value measured ranged from 3.4 to 553 ng/mL, with 3 values below 10 ng/mL in patients with angiographic occlusion. Results were not available for 3 of the patients with angiographic occlusion. Serial cardiac troponin I levels were available and positive for all 24 non-STEMI patients included in the control group (median peak value=3.1 ng/mL; interquartile range 0.8 to 4.93 ng/mL); peak values ranged from 0.3 ng/mL to 11.4 ng/mL.

Main Results

Area under the receiver operator characteristic curve for component c-ii (proportionally excessive discordant ST-segment elevation) was 0.89 (95% CI 0.84 to 0.94), with the criterion for the ST/S ratio determined to be less than −0.25. Area under the receiver operator characteristic curve for component d (proportionally excessive discordant ST-segment elevation or ST-segment depression) was 0.96 (95% CI 0.91 to 0.98), with the criterion for the ST/S ratio determined to be less than −0.30.

Each of components a to d was more frequently observed on the ECGs of patients with acute occlusion versus the control group (Tables 2 and 3). Less than half as many STEMI patients had absolute excessive discordant ST-segment elevation (component c-i) versus proportionally excessive ST-segment elevation (component c-ii) (30% versus 79%): 10 versus 17 of 20 left anterior descending artery acute coronary occlusions showed absolute versus proportional, and 0 versus 8 of 9 right coronary arteries showed absolute versus proportional (Table 4) excessive ST-segment elevation. Absolute excessive discordance,

Table 2. Prevalence of components by leads in acute coronary occlusion and control groups.

No. of leads and location in which each rule component was found	Control (n=129)	ACO (n=33)
	% (n)	
Component a: concordant ST elevation		
No. of leads		
0	98 (127)	58 (19)
1	2 (2)	27 (9)
≥2	0	15 (5)
Location		
Any of V1–V4, aVR	0	3 (1)
Any of II, III, aVF	2 (2)	21 (7)
Any of I, aVL, V5, V6	0	21 (7)
Component b: ST depression ≥1 mm in any of V1–V3	1 (1)	21 (7)
Component c-i: excessively discordant ST elevation, absolute ≥5 mm		
No. of leads		
0	91 (118)	70 (23)
1	5 (6)	21 (7)
≥2	4 (5)	9 (3)
Location		
Any of V1–V4, aVR	9 (11)	30 (10)
Any of II, III, aVF	0	0
Any of I, aVL, V5, V6	0	0
Component c-ii: excessively discordant ST elevation, proportional (ST/S ≤0.25)		
No. of leads		
0	91 (117)	21 (7)
1	9 (12)	27 (9)
≥2	0	52 (17)
Location		
Any of V1–V4, aVR	8 (10)	58 (19)
Any of II, III, aVF	2 (2)	27 (9)
Any of I, aVL, V5, V6	0	9 (3)
Component d: excessive discordance overall, proportional (ST/S ≤0.30)		
No. of leads		
0	88 (113)	0
1	10 (13)	27 (9)
≥2	2 (3)	73 (24)
In any lead	12 (16)	100 (33)

ACO, Acute coronary occlusion.

when observed, was infrequently observed in more than 1 lead; in contrast, proportionally excessive discordance, when observed, was often observed in multiple leads (Table 2). All of the ECGs in patients with STEMI showed either proportionally excessive discordant ST-segment elevation or proportionally excessive ST-segment depression (component d) compared with 12% of the control group, and most had excessive discordance in multiple leads.

Sensitivity and specificity of the 5 diagnostic rules are shown in Table 4. Sensitivities of rules III, IV, and V, all incorporating a measure of proportionally excessive discordance, were not statistically different from one another: the 95% CI for the difference in sensitivity between rules III versus IV and III versus V was −4% to 9%. Sensitivity of rule III (91%), in

Table 3. Frequency of components by location of occlusion.

Component, n	Control, N=129	Acute Coronary Occlusion, N=33	Left Anterior Descending Artery, N=20	Circumflex, N=4	Right Coronary Artery, N=9
a					
Concordant ST-segment elevation	2	14	7	3	4
Any of leads aVR, V1–V4	0	1	0	1	0
Any of leads II, III, aVF	2	7	1	2	4
Any of leads I, aVL, V5, V6	0	7	7	0	0
b					
Concordant ST-segment depression, any of leads V1–V3	1	7	1	4	2
c-i					
Excessively discordant ST-segment elevation, absolute	11	10	10	0	0
Any of leads V1–V4, aVR	11	10	10		
Any of leads II, III, aVF	0	0	0		
Any of leads I, aVL, V5, V6	0		0	0	
c-ii					
Excessively discordant ST-segment elevation, proportional	12	26	17	1	8
Any of leads V1-V4, aVR	10	19	16	0	3
Any of leads II, III, aVF	2	9	2	1	6
Any of leads I, aVL, V5, V6	0	3	3	0	0
d					
Excessive discordance overall, proportional	16	33	20	4	9

Table 4. Performance characteristics of the Sgarbossa weighted, unweighted, and revised rules for acute coronary occlusion in presence of left bundle branch block.

Rule	95% CI			
	Sensitivity	Specificity	Positive Likelihood Ratio	Negative Likelihood Ratio
I, Sgarbossa weighted (a, b, c-i)	52 (34–69)	98 (93–100)	22 (16–31)	0.5 (0.2–1.6)
II, Sgarbossa unweighted (a, b, or c-i)	67 (48–82)	90 (83–95)	6.6 (5.2–8.5)	0.4 (0.2–0.8)
III, Modified Sgarbossa (a, b, or c-ii)	91 (76–98)	90 (83–95)	9.0 (8.0–10)	0.1 (0.03–0.3)
IV, Modified Sgarbossa (a, b, or d)	100 (89–100)	86 (79–92)	7.2 (6.7–7.7)	0
V, Overall discordance (d)	100 (89–100)	88 (81–93)	8.1 (7.6–8.6)	0

which an ST/S ratio less than or equal to −0.25 replaces ST-segment elevation greater than or equal to 5mm, was significantly greater than that of either rule I (52%), the weighted Sgarbossa rule (95% CI for difference 19% to 39%), or rule II (67%), the unweighted Sgarbossa rule (95% CI for difference 6% to 24%). Specificity of rules III versus IV and III versus V was also not significantly different: the 95% CI for the difference in specificity between rules III versus IV was −3% to 9%; and for rules III versus V, −5% to 8%. Specificity of rule III was lower than that of rule I (90% versus 98%; 95% CI for difference 3% to 8%) and similar to that of rule II (both 90%; 95% CI for difference −6% to 6%). Positive and negative likelihood ratios for rule III were 9.0 (95% CI 8.0 to 10) and 0.1 (95% CI 0.03 to 0.3), respectively.

By replacing the absolute criterion of 5 mm (criterion c-i) with the proportional one (c-ii), the revised rule (rule III) was significantly more accurate than rule I (16% difference; 95% CI 5% to 27%) or rule II (12% difference; 95% CI 2% to 22%), with sensitivity of 91% and specificity of 90%. The most accurate (94%; 95% CI 89% to 97%) and sensitive rule was one that included proportionally excessively discordant ST depression (rule V, with 100% sensitivity and 88% specificity).

LIMITATIONS

It is likely that other patients with both left bundle branch block and an acute coronary occlusion were treated at the 3 institutions during this period and were not identified by our methods. All controls did not have angiograms; thus, we cannot rule out acute coronary occlusion, but this seems unlikely with our strict criteria. Some patients were excluded for lack of complete data, and how this might bias the study is unknown. Furthermore, we used angiographic reports. Our definition relied on a culprit lesion, but a culprit alone was not enough because these may also be found in non-STEMI. Thus, we required a minimum peak cardiac troponin I level for cases that did not have documented occlusion. Our ECG measurements were from an inexperienced ECG reader, with selective overreading by an experienced reader. This may have introduced some unknown measurement bias. Last, the cut points for the ratios were derived in this study and thus need future validation. The acute coronary occlusion group included 33 individuals, which limited our power to detect small differences in sensitivity. Specifically, we had approximately

80% power to detect a 25% increase in sensitivity between rules.

DISCUSSION

To our knowledge, this is the first and only study to use angiographic endpoints to evaluate the accuracy of the ECG in the diagnosis of acute myocardial infarction in the presence of left bundle branch block. The American College of Cardiology and American Heart Association guidelines for the treatment of STEMI recommend reperfusion therapy for patients with chest pain and new, or presumably new, left bundle branch block.[1,23] The 2004 updated version suggests also using the Sgarbossa ECG criteria.[1] The 2007 and 2009 focused updates do not further comment on this issue.[24,25] This recommendation for treating all new left bundle branch block is based on early fibrinolytic trials in which bundle branch block or left bundle branch block (new and old) were eligibility criteria for the trial, and those patients with ischemic symptoms and left bundle branch block who received the drug had a lower overall mortality compared with those who received placebo.[26] However, there were no subgroup analyses of the ECGs to elucidate characteristics of left bundle branch block that make response to fibrinolytics more or less likely. Moreover, there was no differentiation between new and old left bundle branch block. Thus, all patients with left bundle branch block have been treated the same by clinical guidelines, regardless of ECG features that might distinguish them. It is possible, or even likely, that many or most of the patients enrolled in these trials did not have coronary occlusion, in contrast to patients with normal conduction who were enrolled. In fact, patients with left bundle branch block and known acute myocardial infarction have higher mortality than patients with normal conduction and acute myocardial infarction.[27,28] In contrast, patients with left bundle branch block who receive reperfusion therapy for presumed acute myocardial infarction have been reported to have lower mortality than their counterparts with normal conduction,[15] likely because the data include patients with left bundle branch block without acute coronary occlusion who received reperfusion therapy.

The Sgarbossa criteria come from an analysis of the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries or GUSTO-1 trial, in which, of 41,021 patients who received fibrinolytic therapy, only 133 had left bundle branch block and a positive CK-MB result.[2] Among patients with normal conduction (no bundle branch block), only approximately 45% of patients with myocardial infarction by CK-MB have a complete coronary occlusion.[29-32] If this is also true of patients with left bundle branch block and acute myocardial infarction, there would have been approximately 65 patients with occlusion in GUSTO-1; however, angiography was not available.

In reality, despite guideline recommendations, patients with left bundle branch block and ischemic symptoms infrequently undergo reperfusion therapy, or it is delayed, and this is true even for those who ultimately receive a biomarker diagnosis of acute myocardial infarction.[33-37] In a study of National Registry of Myocardial Infarction-1 (NRMI-1) data, only 6.7% of all acute myocardial infarction and only 1% of reperfusion cases had left bundle branch block.[27] A study of NRMI-2 data found that only 3.8% of acute myocardial infarction had left bundle branch block, and only 8.4% of these patients received reperfusion therapy (0.32% of all acute myocardial infarction patients had left bundle branch block and reperfusion therapy).[28] In NRMI-3 and -4, only 2% of patients undergoing reperfusion therapy had left bundle branch block,[36,38] as was also true in the reperfusion trial Hirulog Early Reperfusion Occlusion or HERO-2.[15] This suggests that clinicians do not in fact indiscriminately use reperfusion therapy in patients with left bundle branch block and ischemic symptoms.

This absence of use of reperfusion therapy for all patients with LBBB is likely due to clinical experience, confirmed by literature, which suggests that chest pain in the presence of left bundle branch block is infrequently due to acute myocardial infarction and even less frequently due to coronary occlusion or near occlusion (STEMI).[39] The incidence of CK-MB–diagnosed myocardial infarction (STEMI+non-STEMI) among consecutive patients with possible ischemic symptoms and left bundle branch block was low (13%) in 2 ED studies.[8,10] (The more recent study by Kontos et al,[9] with a higher incidence of myocardial infarction, included a more select population) (personal communication, Michael C. Kontos, MD, Virginia Commonwealth University, August 2012). A more recent ED study found the incidence of troponin-diagnosed acute myocardial infarction to be much lower still, whether new left bundle branch block (7.3%) or old left bundle branch block (5.2%).[40] Several other studies confirmed the low incidence of acute myocardial infarction, and especially of occlusion, with simple new left bundle branch block.[17,41,42] Given that the incidence of STEMI (occlusion) in troponin-diagnosed acute myocardial infarction is approximately 30%,[43] then, by extrapolation, only 1.5% to 4% of patients with ischemic symptoms and left bundle branch block have acute occlusion. This was confirmed in a more recent study in which only 6 of 177 patients with left bundle branch block referred for primary PCI received it and only 1 had 100% occlusion.[44] Because undifferentiated new left bundle branch block is also nonspecific for acute coronary occlusion, one analysis predicted better outcomes by using Sgarbossa criteria or a positive troponin result for the fibrinolytic decision than by treating all patients with new left bundle branch block.[45] A newer algorithm uses hemodynamic instability, Sgarbossa criteria, bedside echocardiography, and serial biomarker testing to guide emergency reperfusion.[39] Thus, it would be useful to have an ECG guideline that is more accurate than the Sgarbossa criteria for diagnosing acute coronary occlusion (STEMI) in the presence of left bundle branch block to guide decisions on reperfusion therapy.

Electrophysiologically, repolarization voltages must be proportional to depolarization voltages. During stress testing,
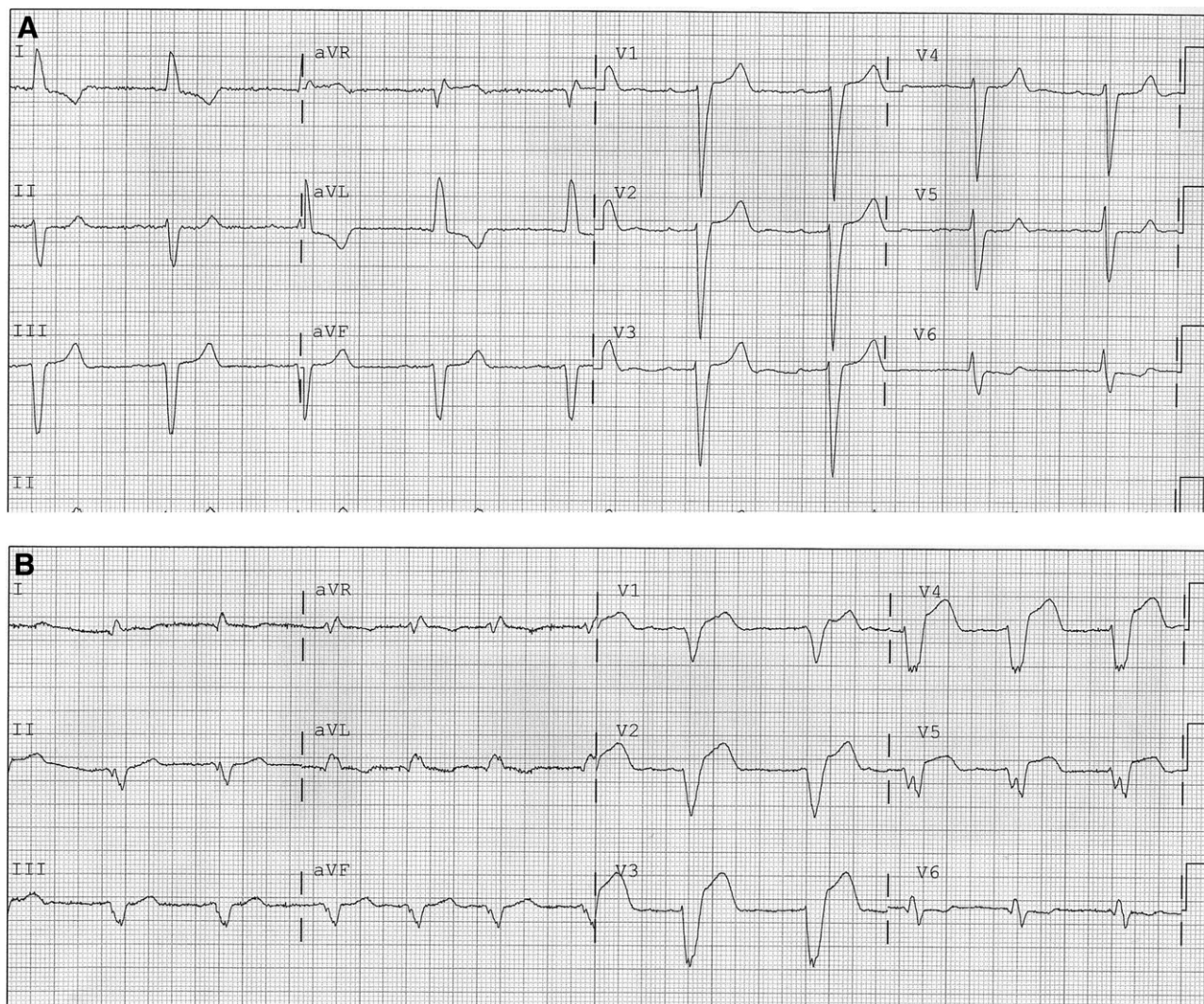
Figure 3. The ECGs of a patient who presented with chest pain and left bundle branch block and had a left anterior descending artery occlusion, compared with his baseline ECG. *A,* The patient's baseline ECG with left bundle branch block. Maximum ST elevation at the J point is 2 mm in lead V2, with an ST/S ratio of 2/23=0.087. *B,* The patient's ECG when he presented with chest pain. There is no concordant ST deviation (no concordant ST-segment elevation or ST depression). Maximum ST-segment elevation is higher but still less than 5 mm (4.5 mm) and thus does not meet even the unweighted Sgarbossa criteria (it does not earn 2 points). However, ST/S ratios in V1 to V3 were, respectively, 2.5/–9.5=−0.26, 4.5/−12=−0.38, and 3/−9.5=−0.32; all 3 are less than −0.25 but only 1 needs to be so to fulfill the new criteria. This patient was taken for emergency angiography and PCI of a 100% acute left anterior descending artery occlusion.

the significance of ST depression depends on the preceding R-wave amplitude.[46-48] T-wave to QRS amplitude ratio distinguishes left ventricular "aneurysm" morphology (persistent ST elevation after previous myocardial infarction) from acute STEMI,[49,50] and R-wave to T-wave amplitude ratio distinguishes early repolarization from acute STEMI.[51] Madias et al[52] showed that 8 of 128 (6%) patients with left bundle branch block without acute myocardial infarction had at least 1 lead in V1 to V3 with at least 5-mm ST-segment elevation. They did not calculate a ratio but did show one example that had a very deep S-wave and an ST to S-wave ratio of less than

−0.25. In another study of patients with baseline left bundle branch block (without acute myocardial infarction) and greater than or equal to 5-mm discordant ST-segment elevation, the mean preceding S wave was 46 mm (range 28.0 to 71.0 mm), for a ratio consistently less than −0.25.[11] Of 223 consecutive ED patients with left bundle branch block without acute coronary occlusion, ST/S ratio was more specific than an absolute value of greater than or equal to 5 mm.[53] Figure 2 shows the baseline ECG of a patient with left bundle branch block without ischemia; it shows 7 mm of ST-segment elevation but also a (–)53-mm S-wave, for an ST/S ratio of −0.13. Our

data confirm the hypothesis that, by substituting a proportional criterion for an absolute one, the diagnostic characteristics are improved.

Anterior STEMI caused by acute left anterior descending artery occlusion results in ST-segment elevation in leads V1 to V4, as well as in any or all of leads V5, V6, I, and aVL when occlusion is proximal to the first diagonal artery. In left bundle branch block, the normal discordance results in ST-segment elevation in leads V1 to V4 at baseline. Therefore, in the setting of a mid left anterior descending artery occlusion, the diagnosis of STEMI will rely exclusively on excessive discordance in leads V1 to V4. Sgarbossa's weighted criteria give only 2 points for excessive discordance and thus will "miss" a large number of anterior STEMIs, as they did in our study. Predictably, the unweighted criteria were more sensitive (52% versus 67%) for acute myocardial infarction; however, they were less specific (98% versus 90%). By replacing the absolute criterion of 5 mm (criterion c-i) with the proportional one (c-ii), the rule was significantly more accurate than either of the others, with sensitivity of 91% and specificity of 90%. The most accurate and sensitive rule was one that included proportionally excessively discordant ST-segment depression (rule V, with 100% sensitivity and 88% specificity). If validated, it may be another instance in which ST-segment depression, without any ST-segment elevation, is a STEMI equivalent that is an indication for reperfusion therapy. The only such indication at present is marked ST-segment depression in leads V1 to V4, indicative of posterior STEMI, analogous to Sgarbossa's second criterion.[1]

Our study suggests that by using appropriate criteria, the ECG may be more sensitive at diagnosing acute coronary occlusion in the presence of left bundle branch block than it is given credit for. This belief of poor sensitivity stems from previous literature that does not distinguish between non-STEMI and STEMI and lacks angiographic data. Supporting the notion that the ECG may be nearly as sensitive for STEMI in the presence of left bundle branch block as in normal conduction, Stark et al[54] found that in patients with baseline left bundle branch block, a change in the ST segments of 1 mm was 80% sensitive for angiographic balloon occlusion (mean Δ ST 2.7 mm); it was 75% sensitive in patients with normal conduction.

Figure 3 demonstrates the Sgarbossa and revised rules by showing the ECGs of a patient who had previous left bundle branch block and presented with chest pain and proven left anterior descending artery occlusion.

The new criteria should be helpful in managing patients with ischemic symptoms in the presence of left bundle branch block. If confirmed with a validation study, this ratio would potentially provide a tool to guide the need for reperfusion therapy. Sgarbossa's criteria are associated with suboptimal sensitivity in the identification of acute myocardial infarction, as diagnosed by biomarkers, partly because the rule does not consider the relative amplitudes of the ST segment and the S-wave (proportionality) and because the ECG is never very sensitive for acute myocardial infarction as diagnosed by biomarkers. When proportionality is taken into account, despite the presence of left bundle branch block, the ECG may be much better than previously thought at discriminating between patients with and without acute coronary occlusion, especially a left anterior descending artery occlusion.

Diagnosis of acute coronary occlusion in the setting of left bundle branch block, particularly left anterior descending artery occlusion, remains a challenging clinical problem. In this derivation study, the ratio of amplitude of ST elevation to the preceding S-wave depth (ST/S ratio) was significantly different, and with a significantly greater diagnostic sensitivity and accuracy, than maximum ST elevation. Furthermore, replacing criterion 3 (excessively discordant ST elevation) as defined by greater than or equal to 5 mm with a proportional criterion (ST/S ratio <-0.25) as measured in any one lead greatly improved the diagnostic characteristics of the Sgarbossa criteria. Proportionally excessive discordant ST-segment elevation or depression may prove to be even more valuable.

REFERENCES
1. Antman EM, Anbe DT, Armstrong PW, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction—executive summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1999 Guidelines for

the Management of Patients With Acute Myocardial Infarction).
J Am Coll Cardiol. 2004;44:671-719, e29–e30.

2. Sgarbossa EB, Pinski SL, Barbagelata A, et al. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle branch block. *N Engl J Med*. 1996;334:481-487.

3. Eriksson P, Gunnarsson G, Dellborg M. Diagnosis of acute myocardial infarction in patients with chronic left bundle branch block. Standard 12-lead ECG compared to dynamic vectorcardiography. *Scand Cardiovasc J*. 1999;33:17-22.

4. Al-Faleh H, Fu Y, Wagner G, et al. Unraveling the spectrum of left bundle branch block in acute myocardial infarction: insights from the Assessment of the Safety and Efficacy of a New Thrombolytic (ASSENT 2 and 3) trials. *Am Heart J*. 2006;151:10-15.

5. Edhouse JA, Sakr M, Angus J, et al. Suspected myocardial infarction and left bundle branch block: electrocardiographic indicators of acute ischaemia. *J Accid Emerg Med*. 1999;16:331-335.

6. Gula LJ, Dick A, Massel D. Diagnosing acute myocardial infarction in the setting of left bundle branch block: prevalence and observer variability from a large community study. *Coron Artery Dis*. 2003;14:387-393.

7. Gunnarsson G, Eriksson P, Dellborg M. ECG criteria in diagnosis of acute myocardial infarction in the presence of left bundle branch block. *Int J Cardiol*. 2001;78:167-174.

8. Kontos MC, McQueen RH, Jesse RL, et al. Can myocardial infarction be rapidly identified in emergency department patients who have left bundle branch block? *Ann Emerg Med*. 2001;37:431-438.

9. Kontos MC, Aziz HA, Chau VQ, et al. Outcomes in patients with chronicity of left bundle branch block with possible acute myocardial infarction. *Am Heart J*. 2011;161:698-704.

10. Li SF, Walden PL, Marcilla O, et al. Electrocardiographic diagnosis of myocardial infarction in patients with left bundle branch block. *Ann Emerg Med*. 1999;36:561-566.

11. Madias JE, Sinha A, Ashtiani R, et al. A critique of the new ST-segment criteria for the diagnosis of acute myocardial infarction in patients with left bundle branch block. *Clin Cardiol*. 2001;24:652-655.

12. Ozment A, Garvey L, Littmann, et al. An analysis of ECG criteria for acute myocardial infarction in the presence of left bundle branch block. *Acad Emerg Med*. 1999;6:423-424.

13. Shlipak MG, Lyons WL, Go AS, et al. Should the electrocardiogram be used to guide therapy for patients with left bundle branch block and suspected myocardial infarction? *JAMA*. 1999;281:714-719.

14. Sokolove PE, Sgarbossa EB, Amsterdam EA, et al. Interobserver agreement in the electrocardiographic diagnosis of acute myocardial infarction in patients with left bundle branch block. *Ann Emerg Med*. 2000;36:566-572.

15. Wong CK, French JK, Aylward PE, et al. Patients with prolonged ischemic chest pain and presumed-new left bundle branch block have heterogeneous outcomes depending on the presence of ST-segment changes. *J Am Coll Cardiol*. 2005;46:29-38.

16. Maynard SJ, Menown IB, Manoharan G, et al. Body surface mapping improves early diagnosis of acute myocardial infarction in patients with chest pain and left bundle branch block. *Heart*. 2003;89:998-1002.

17. Tabas JA, Rodriguez RM, Seligman HK, et al. Electrocardiographic criteria for detecting acute myocardial infarction in patients with left bundle branch block: a meta-analysis. *Ann Emerg Med*. 2008;52:329-336.

18. Antman EM, Tanasijevic MJ, Thompson B, et al. Cardiac-specific troponin I levels to predict the risk of mortality in patients with acute coronary syndromes. *N Engl J Med*. 1996;335:1342-1349.

19. Hallen J, Buser P, Schwitter J, et al. Relation of cardiac troponin I measurements at 24 and 48 hours to magnetic resonance–determined infarct size in patients with ST-elevation myocardial infarction. *Am J Cardiol*. 2009;104:1472-1477.

20. Chia S, Senatore F, Raffel OC, et al. Utility of cardiac biomarkers in predicting infarct size, left ventricular function, and clinical outcome after primary percutaneous coronary intervention for ST-segment elevation myocardial infarction. *J Am Coll Cardiol*. 2008; 1:415-423.

21. Sgarbossa EB. Recent advances in the electrocardiographic diagnosis of myocardial infarction: left bundle branch block and pacing. *Pacing Clin Electrophysiol*. 1996;19:1370-1379.

22. Kligfield P, Gettes LS, Bailey JJ, et al. Recommendations for the standardization and interpretation of the electrocardiogram: part I: the electrocardiogram and its technology: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society Endorsed by the International Society for Computerized Electrocardiology. *Circulation*. 2007;115:1306-1324.

23. Ryan TJ, Antman EM, Brooks NH. 1999 Update: ACC/AHA guidelines for the management of patients with acute myocardial infarction. *J Am Coll Cardiol*. 1999;34:890-911, e46.

24. Kushner FG, Hand M, Smith SC Jr, et al. 2009 focused updates: ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction (updating the 2004 guideline and 2007 focused update) and ACC/AHA/SCAI guidelines on percutaneous coronary intervention (updating the 2005 guideline and 2007 focused update): a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*. 2009;54:2205-2241.

25. Antman EM, Hand M, Armstrong PW, et al. 2007 Focused update of the ACC/AHA 2004 guidelines for the management of patients with ST-elevation myocardial infarction. *J Am Coll Cardiol*. 2008;51:210-247.

26. Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomised trials of more than 1000 patients. *Lancet*. 1994;343:311-322.

27. Go AS, Barron HV, Rundle AC, et al. bundle branch block and in-hospital mortality in acute myocardial infarction. National Registry of Myocardial Infarction. *Ann Intern Med*. 1998;129:690-697.

28. Shlipak MG, Go AS, Frederick PD. Treatment and outcomes of left bundle branch block patients with myocardial infarction who present without chest pain. *J Am Coll Cardiol*. 2000;36:706-712.

29. Fesmire FM, Percy RF, Bardoner JB, et al. Usefulness of automated serial 12-lead ECG monitoring during the initial emergency department evaluation of patients with chest pain. *Ann Emerg Med*. 1998;31:3-11.

30. Fesmire FM, Percy RF, Wears RL, et al. Initial ECG in Q wave and non-Q wave myocardial infarction. *Ann Emerg Med*. 1989;18:741-746.

31. Rouan GW, Lee TH, Cook EF, et al. Clinical characteristics and outcome of acute myocardial infarction in patients with initially normal or nonspecific electrocardiograms (a report from the Multicenter Chest Pain Study). *Am J Cardiol*. 1989;64:1087-1092.

32. Rude RE, Poole WK, Muller J, et al. Electrocardiographic and clinical criteria for recognition of acute myocardial infarction based on analysis of 3,697 patients. *Am J Cardiol*. 1983;52:936-942.

33. Tricomi AJ, Magid DJ, Rumsfeld JS, et al. Missed opportunities for reperfusion therapy for ST-segment elevation myocardial infarction: results of the Emergency Department Quality in

Myocardial Infarction (EDQMI) study. *Am Heart J*. 2008;155:471-477.

34. Melgarejo MA, Galcera TJ, Garcia A, et al. The incidence, clinical characteristics, and prognostic significance of a left bundle branch block associated with an acute myocardial infarct. *Rev Esp Cardiol*. 1999;52:245-252.

35. Krumholz HM, Murillo JE, Chen J, et al. Thrombolytic therapy for eligible elderly patients with acute myocardial infarction. *JAMA*. 1997;277:1683-1688.

36. McNamara RL, Wang Y, Herrin J, et al. Effect of door-to-balloon time on mortality in patients with ST-segment elevation myocardial infarction. *J Am Coll Cardiol*. 2006;47:2180-2186.

37. Archbold RA, Ranjadayalan K, Suliman A, et al. Underuse of thrombolytic therapy in acute myocardial infarction and left bundle branch block. *Clin Cardiol*. 2010;33:E25-29.

38. McNamara RL, Herrin J, Bradley EH, et al. Hospital improvement in time to reperfusion in patients with acute myocardial infarction, 1999 to 2002. *J Am Coll Cardiol*. 2006;47:45-51.

39. Neeland IJ, Kontos MC, de Lemos JA. Evolving considerations in the management of patients with left bundle branch block and suspected myocardial infarction. *J Am Coll Cardiol*. 2012;60:96-105.

40. Chang AM, Shofer FS, Tabas JA, et al. Lack of association between left bundle branch block and acute myocardial infarction in symptomatic ED patients. *Am J Emerg Med*. 2009;27:916-921.

41. Jain S, Ting HT, Bell M, et al. Utility of left bundle branch block as a diagnostic criterion for acute myocardial infarction. *Am J Cardiol*. 2011;107:1111-1116.

42. Poon K, Frederick PD, French WJ. Abstract 4317: does a new or presumed new left bundle branch block have equivalent mortality to an acute ST-elevation myocardial infarction? *Circulation*. 2009; 120(18 suppl 2).

43. Roe MT, Parsons LS, Pollack CV Jr, et al. Quality of care by classification of myocardial infarction: treatment patterns for ST-segment elevation vs non-ST-segment elevation myocardial infarction. *Arch Intern Med*. 2005;165:1630-1636.

44. Joshil NV, Bawamia BR, Jamieson S, et al. Evaluating a nurse led triage process in treating patients with left bundle branch block

(LBBB) referred for primary percutaneous coronary intervention (pPCI) [abstract]. *Heart*. 2011;97(Suppl 1):A10.

45. Tabas JA, Choen C, Conrads-Frank A, et al. What is the optimal approach to select patients with left bundle branch block and suspected AMI for fibrinolytic therapy [abstract]? *Acad Emerg Med*. 2009;16:s73-s74.

46. Ellestad MH, Crump R, Surbur M. The significance of lead strength on ST changes during treadmill stress tests. *J Electrocardiol*. 1992;25(suppl):31-34.

47. Hollenberg M, Go JJ, Massie BM, et al. Influence of R-wave amplitude on exercise-induced ST depression: need for a "gain factor" correlation when interpreting stress electrocardiograms. *Am J Cardiol*. 1985;56:13.

48. Santinga JT, Brymer JF, Smith F, et al. The influence of lead strength on the ST changes with exercise electrocardiography (correlative study with coronary arteriography). *J Electrocardiol*. 1977;10:387.

49. Smith SW. T/QRS amplitude ratio best distinguishes the ST elevation of anterior left ventricular aneurysm from anterior acute myocardial infarction. *Am J Emerg Med*. 2005;23:279-287.

50. Beeman W, Smith SW. T/QRS amplitude ratio is significantly higher in acute anterior ST elevation myocardial infarction than in previous myocardial infarction with persistent ST elevation (left ventricular aneurysm morphology): a validation (abstract 371). *Ann Emerg Med*. 2011;58(4 suppl):S302.

51. Smith SW, Khalil A, Henry TD, et al. Electrocardiographic differentiation of early repolarization from subtle anterior ST-segment elevation myocardial infarction. *Ann Emerg Med*. http://dx.doi.org/10.1016/j.annemergmed.2012.02.015.

52. Madias JE, Sinha A, Agarwal H, et al. ST-segment elevation in leads V1-V3 in patients with left bundle branch block (LBBB). *J Electrocardiol*. 2001;34:87-88.

53. Smith SW, Bertog SC, Lathrop LM, et al. ST/S ratio distinguishes left bundle branch block from left bundle branch block with simultaneous anterior myocardial infarction [abstract]. *Acad Emerg Med*. 2006;13(5 suppl):S160-S161.

54. Stark KS, Krucoff MW, Schryver B, et al. Quantification of ST-segment changes during coronary angioplasty in patients with left bundle branch block. *Am J Cardiol*. 1991;67:1219-1222.