EDUCATIONAL ADVANCES

Electrocardiographic ST-segment Elevation: Correct Identification of Acute Myocardial Infarction (AMI) and Non-AMI Syndromes by Emergency Physicians

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Abstract. **Objective:** To determine the emergency physician's (EP's) ability to identify the cause of ST-segment elevation (STE) in a hypothetical chest pain patient. **Methods:** Eleven electrocardiograms (ECGs) with STE were given to EPs; the patient in each instance was a 45-year-old male with a medical history of hypertension and diabetes mellitus with the chief complaint of chest pain. The EP was asked to determine the cause of the STE and, if due to acute myocardial infarction (AMI), to decide whether thrombolytic therapy (TT) would be administered (the patient had no contraindication to such treatment). Rates of TT administration were determined; appropriate TT administration was defined as that occurring in an AMI patient, while inappropriate TT administration was defined as that in the non-AMI patient. **Results:** Four hundred fifty-eight EPs completed the questionnaire; levels of medical experience included the following: postgraduate year 2–3, 193 (42%); and attending, 265 (58%). The overall rate of correct interpretation of the study ECGs was 94.9% (4,782 correct interpretations out of 5,038 instances). Acute myocardial infarction with typical STE, ventricular paced rhythm, and right bundle branch block were never misinterpreted. The remaining conditions were misinterpreted with rates ranging between 9% (left bundle branch block, LBBB) and 72% (left ventricular aneurysm, LVA). The overall rate of appropriate thrombolytic agent administration was 83% (1,525 correct administrations out of 1,832 indicated administrations). The leading diagnosis for which thrombolytic agent was given inappropriately was LVA (28%), followed by benign early repolarization (23%), pericarditis (21%), and LBBB without electrocardiographic AMI (5%). Thrombolytic agent was appropriately given in all cases of AMI except when associated with atypical STE, where it was inappropriately withheld 67% of the time. **Conclusions:** In this survey, EPs were asked whether they would give TT based on limited information (ECG). Certain syndromes with STE were frequently misdiagnosed. Emergency physician electrocardiographic education must focus on the proper identification of these syndromes so that TT may be appropriately utilized. **Key words:** electrocardiogram; ST-segment elevation; emergency physicians; diagnosis. ACADEMIC EMERGENCY MEDICINE 2001; 8:349–360

PATIENTS presenting to the emergency department (ED) with acute cardiovascular syndromes are evaluated in rapid fashion by the emergency physician (EP); one of the tools that the EP uses frequently in the ED is the electrocardiogram (ECG), either single-lead rhythm analysis or the 12-lead ECG. Many such cardiovascular syndromes manifest electrocardiographic abnormality —whether it be ventricular tachycardia in the syncope patient or ST-segment elevation (STE) in the chest pain patient. Correct management decisions rely heavily on the accurate interpretation of the ED evaluation, including the ECG. Regardless of the type of presentation, the EP must be an expert in the interpretation of the ECG. In fact, the EP is frequently the initial clinician who examines the chest pain patient, interprets the ECG, and makes the early therapeutic decisions. Accurate interpretation of the ECG and the correct diagnosis of acute myocardial infarction (AMI) among the numerous causes of STE are mandatory skills for the EP. The ability of the EP to correctly interpret the

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A related commentary appears on page 382.

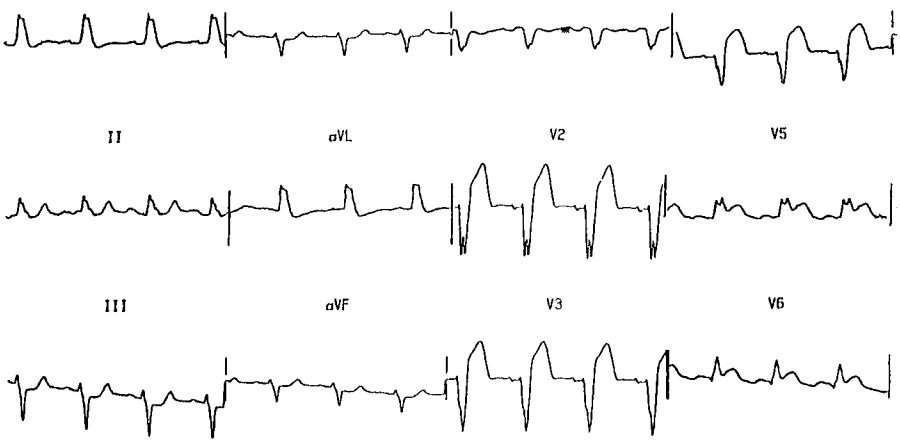

Figure 1. Left bundle branch block with acute myocardial infarction.

ECG findings in such patients directly and immediately impacts on management decisions as well as influences patient outcome.[1–4]

The rapid and accurate identification of AMI is a formidable challenge for the EP. The ECG remains a powerful clinical tool in the evaluation of AMI patients and assists the EP in the selection of the proper therapy, in particular the acute reperfusion therapy, as well as securing an adequate inpatient disposition. The ECG, however, has numerous shortcomings when used in the evaluation of the chest pain patient. In most instances, STE resulting from AMI is easily noted. Confounding patterns, however, such as left bundle branch block (LBBB), ventricular paced rhythms, and left ventricular hypertrophy (LVH), may obscure the typical electrocardiographic findings of AMI as well as produce noninfarctional STE, which may lead the uninformed EP astray. Other STE patterns, including benign early repolarization (BER) and acute pericarditis, occur in the individual with chest discomfort and may suggest the incorrect diagnosis of AMI, exposing the patient to unnecessary and potentially dangerous therapies.

We believe that the EP is able to interpret the ECG accurately, arriving at the correct diagnosis in most electrocardiographic situations. Certain electrocardiographic syndromes, however, may represent challenges in interpretation. We undertook the following study to explore this issue further. The following investigation focuses on this ability to identify the cause of electrocardiographic STE in a hypothetical ED chest pain patient.

METHODS

Study Design. A questionnaire-based study of EPs was performed, investigating the clinicians' ability to interpret the ECG—specifically to determine the cause of electrocardiographic STE in a hypothetical patient. The institutional review board (IRB)/human investigation committee (HIC) of the authors' two institutions reviewed the study protocol; both IRB/HICs considered this study to be exempt from informed consent.

Study Setting and Population. Study participants were a convenience sample of all EPs (senior-level emergency medicine residents [postgraduate year 2 or 3] or post-residency) who attended a continuing medical education lecture on electrocardiography. These lectures were given by the authors at both community and university-affiliated EDs. The study was completed prior to the initiation of the lecture on any given day.

Study Protocol. Eleven ECGs with STE were given to the study EPs. The patient in each instance was a hypothetical 45-year-old male with a medical history of hypertension and diabetes mellitus with the chief complaint of chest pain. The physical examination was remarkable only for an anxious appearance. The EP was asked to determine the cause of the STE and, if due to AMI, to decide whether thrombolytic therapy would be administered assuming the patient had no contraindication to such treatment and that a cardiac catheterization laboratory (for primary angioplasty) was not available.

The 11 ECGs with electrocardiographic STE involved AMI and non-AMI diagnoses. For each ECG, the cardiologist's interpretation, clinical diagnosis, and other objective clinical data (cardiac serum markers, echocardiography, etc.) were used to confirm the individual study 12-lead ECG's diagnosis with respect to the STE; in all instances, all three parameters were in agreement. The following lists the 11 ECGs (Figs. 1–11) with their respective diagnoses and objective clinical data (when applicable):

• Figure 1—Left bundle-branch block with *electrocardiographic* AMI: Positive serum creatinine

phosphokinase–MB fraction; and obviously abnormal ST-segment deviations and morphologies for LBBB pattern. The ST-segment abnormalities supporting the electrocardiographic diagnosis of AMI are based on a clinical decision rule reported by Sgarbossa and colleagues.[5] Concordant STE (leads V5 and V6) is strongly suggestive of AMI, while excessive discordant STE (leads V2, V3, and V4) is a less substantial marker of AMI.[5]

• Figure 2—Lateral AMI with atypical (concave ST-segment morphology): Positive serum creatinine phosphokinase–MB fraction; and echocardiogram revealing lateral akinesis.

• Figure 3—Acute pericarditis without AMI: Negative rule-out myocardial infarction protocol (negative serial creatinine phosphokinase and troponin and serial 12-lead ECG); and echocardiogram with absence of wall motion abnormality and presence of small pericardial effusion.

• Figure 4—Inferoposterior AMI with minimal STE: Positive creatinine phosphokinase and troponin; and cardiac catheterization with inferoposterior wall motion abnormality and distal right coronary artery occlusion due to acute thrombus.

• Figure 5—Left ventricular aneurysm (LVA) without AMI: Negative rule-out myocardial infarction protocol (negative serial creatinine phosphokinase and troponin and serial 12-lead ECG); and echocardiogram (past and present) with anterior wall dyskinesis.

• Figure 6—Left ventricular hypertrophy without electrocardiographic AMI: Negative rule out myocardial infarction protocol (negative serial creatinine phosphokinase and troponin and serial 12-

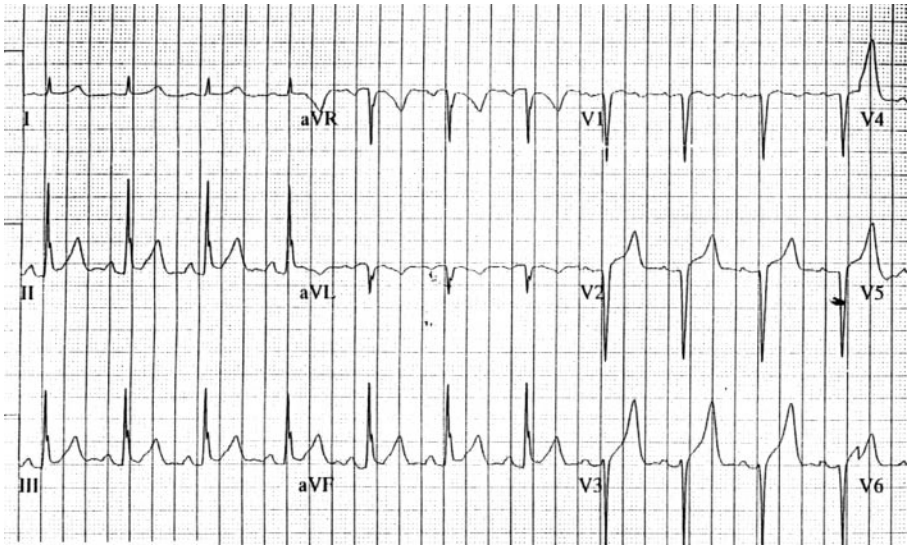

Figure 2. Lateral acute myocardial infarction with atypical (concave ST-segment) morphology.

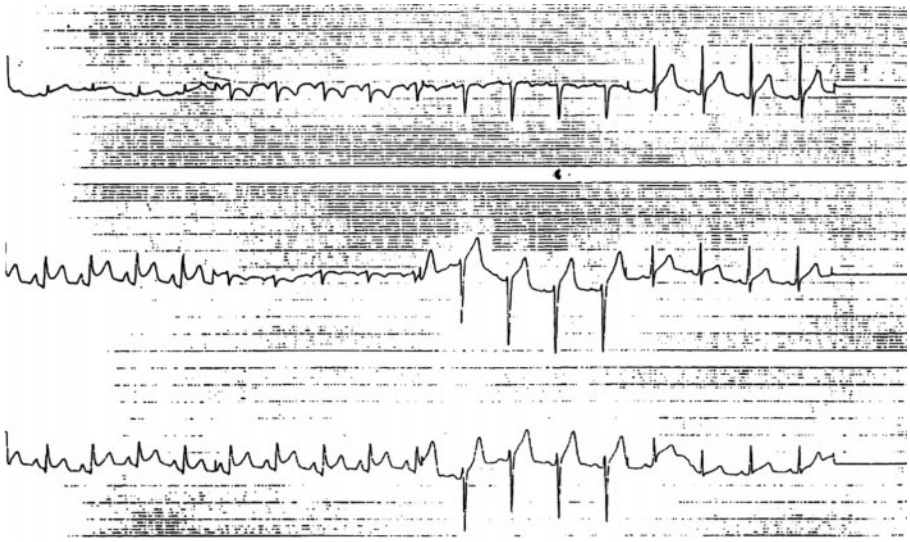

Figure 3. Acute pericarditis without acute myocardial infarction.

lead ECG); and echocardiogram with LVH and normal anterolateral wall motion.

• Figure 7—Left bundle branch block without electrocardiographic AMI: Negative rule-out myocardial infarction protocol (negative serial creatinine phosphokinase and troponin and serial 12-lead ECG); and echocardiogram with past anterior-wall AMI and no evidence of new wall motion abnormality. No electrocardiographic criteria are present suggestive of AMI.[5]

• Figure 8—Right bundle branch block (RBBB) without AMI: Negative rule-out myocardial infarction protocol (negative serial creatinine phosphokinase and troponin and serial 12-lead ECG).

• Figure 9—Right ventricular paced rhythm without electrocardiographic AMI: Negative rule-out myocardial infarction protocol (negative serial creatinine phosphokinase and troponin and serial 12-lead ECG).

• Figure 10—Right bundle branch block with AMI: Positive creatinine phosphokinase and troponin; and cardiac catheterization with left ante-

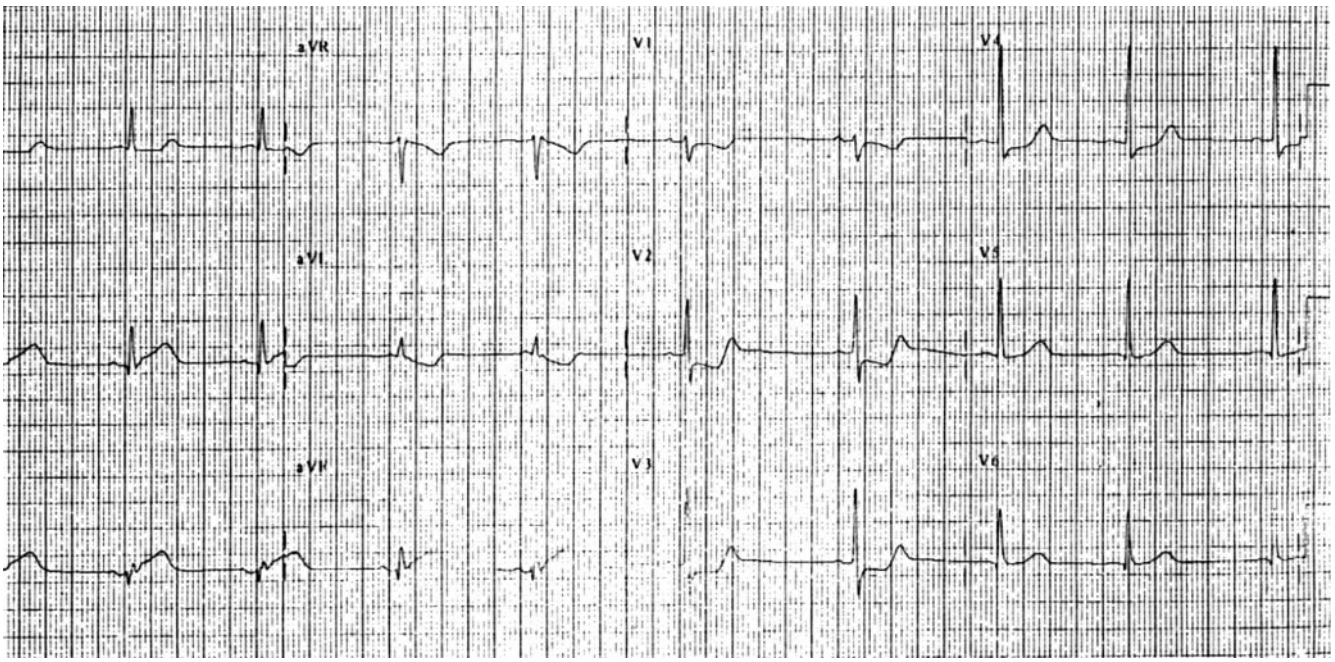

Figure 4. Inferoposterior acute myocardial infarction with minimal ST-segment elevation.

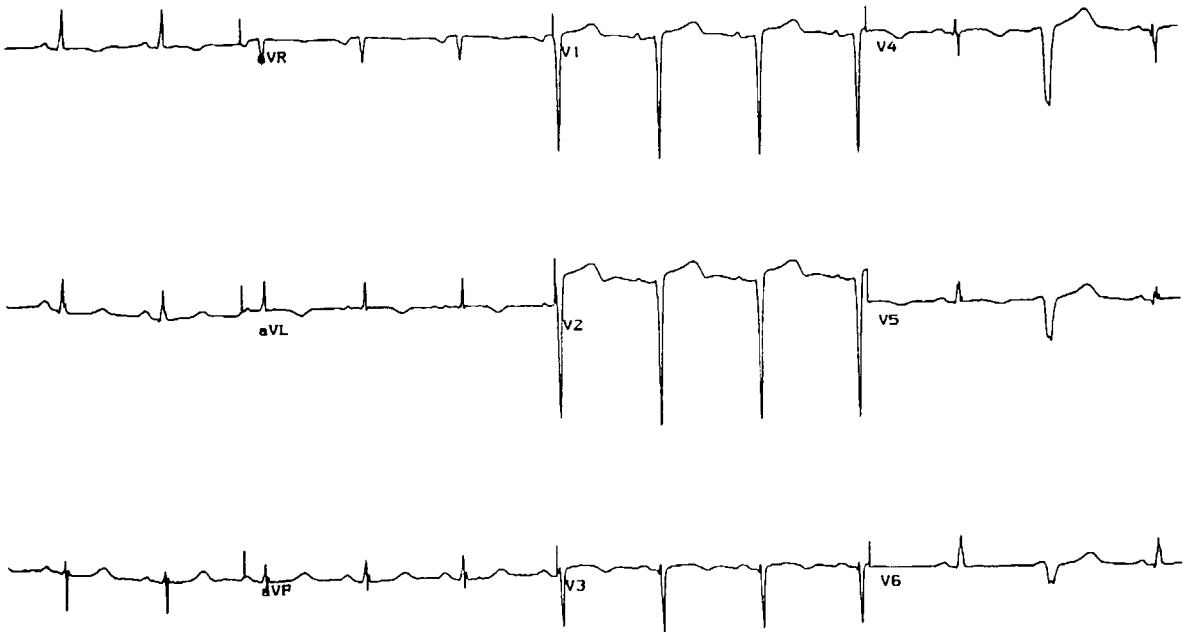

Figure 5. Left ventricular aneurysm without acute myocardial infarction.

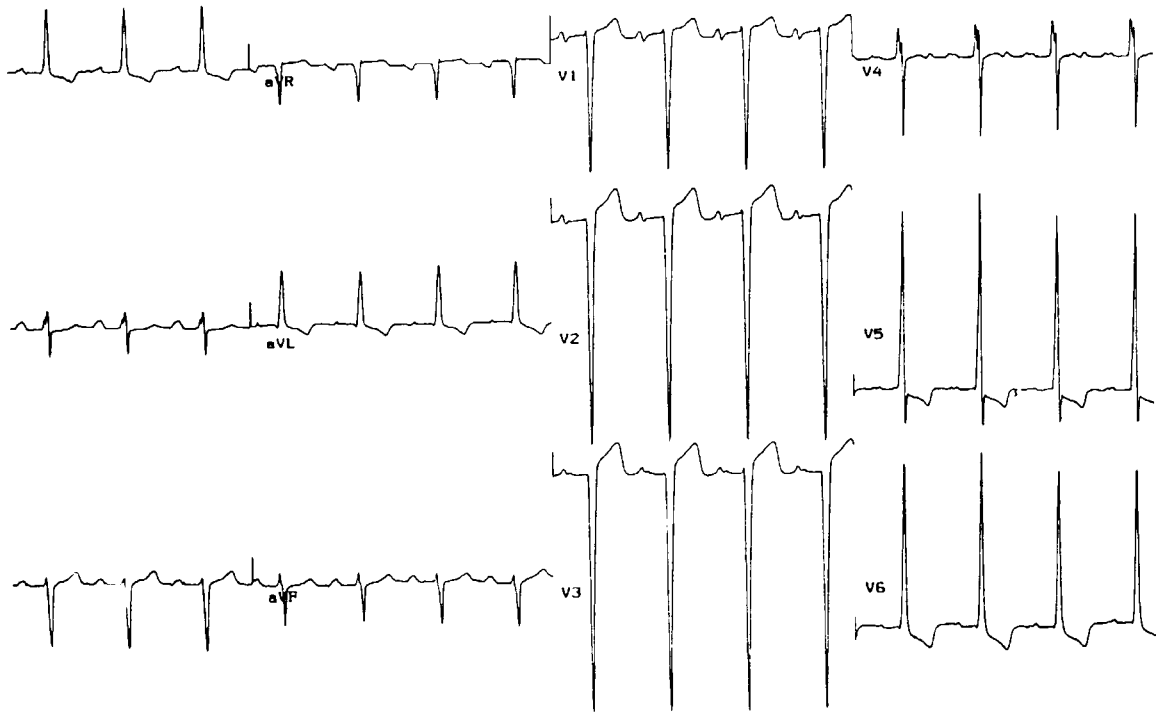

Figure 6. Left ventricular hypertrophy without acute myocardial infarction.

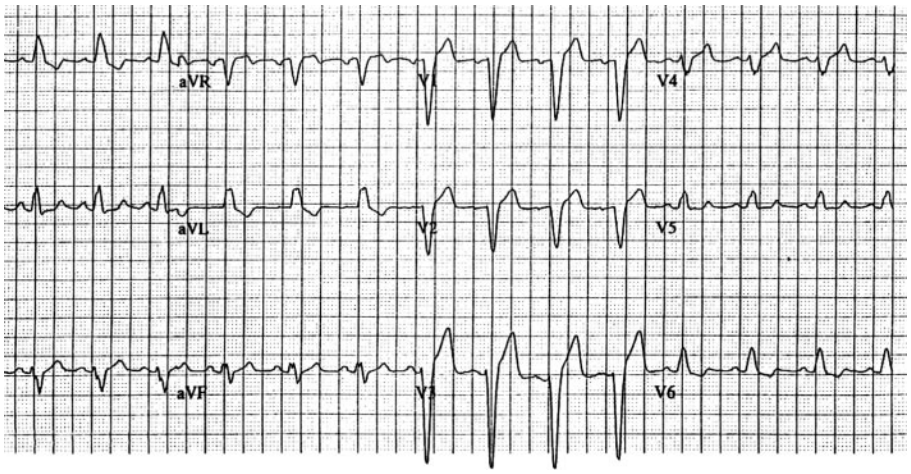

Figure 7. Left bundle branch block without acute myocardial infarction.

rior descending artery thrombus and anterior-wall motion abnormality.

• Figure 11—Benign early repolarization without AMI: Negative rule-out myocardial infarction protocol (negative serial creatinine phosphokinase and troponin and serial 12-lead ECG); static appearance of pattern with serial ECGs; and negative, adequate exercise thallium test.

Measurements and Definitions. The listed electrocardiographic diagnosis for each of the 11 ECGs was recorded for the study EPs. Study participants were queried as to the cause of the electrocardiographic STE; they were asked to list the electrocardiographic diagnosis. Correct diagnoses were recorded as such; incorrect diagnoses were noted as incorrect with the addition of the incorrect interpretation relative to the correct diagnosis. A correct diagnosis required a full, complete correct answer. For example, the notation "left bundle branch block" for case scenario 1 (correct answer —LBBB with AMI) was considered incorrect. Discrepancies in answer nomenclature were reviewed by the authors.

In a separate analysis, rates of thrombolytic therapy administration were determined and recorded as either appropriate or inappropriate; appropriate administration was defined as that oc-

curring in an AMI patient, while inappropriate thrombolytic therapy administration was defined as occurring in the non-AMI patient.

The physician level of training was noted in the following manner: postgraduate year (PGY) 2 (the second year of emergency medicine residency training), PGY 3 (the third year of emergency medicine residency training), and postgraduate (completion of residency training/practicing EP).

Data Analysis. Comparisons were made between physician groups in terms of the rate of correct interpretation. Proportions were analyzed with the chi-square or Fisher's exact test where appropriate. Means and standard deviations were compared with Student's t-test. A p-value <0.05 was considered statistically significant.

RESULTS

Four hundred fifty-eight EPs completed the questionnaire; levels of medical experience included the

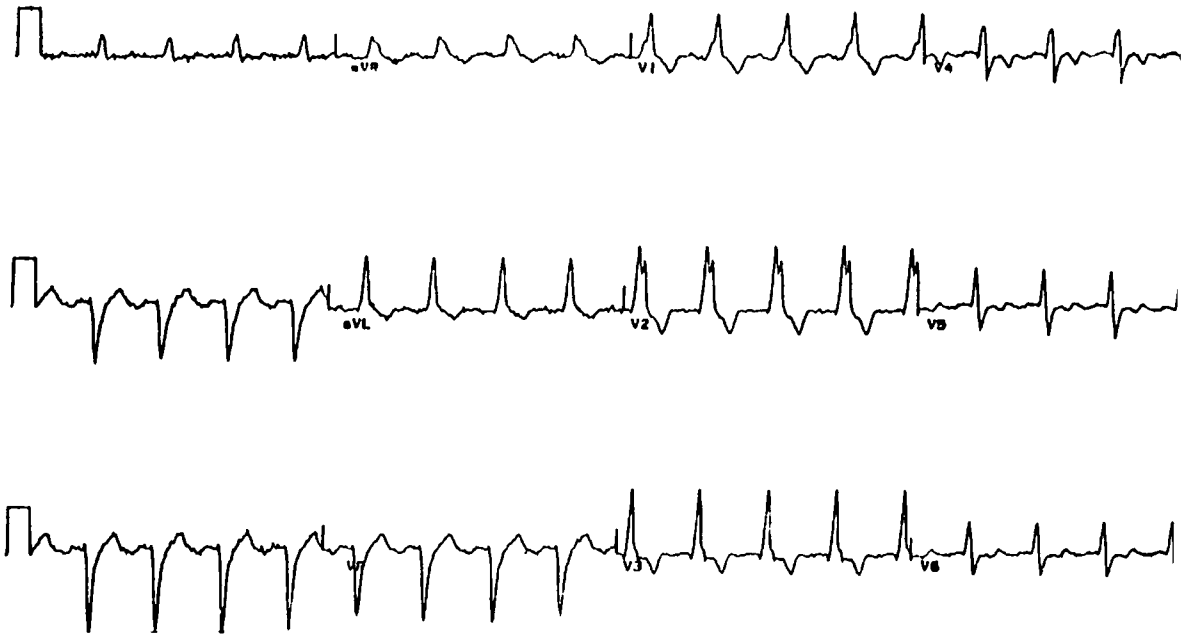

Figure 8. Right bundle branch block without acute myocardial infarction.

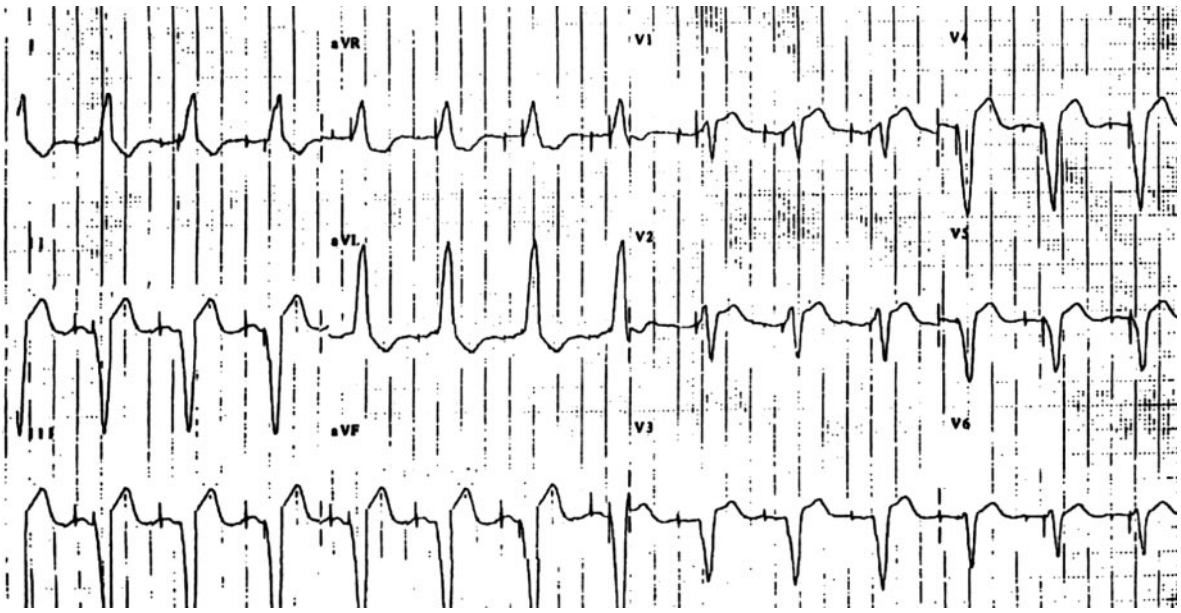

Figure 9. Right ventricular paced rhythm without acute myocardial infarction.

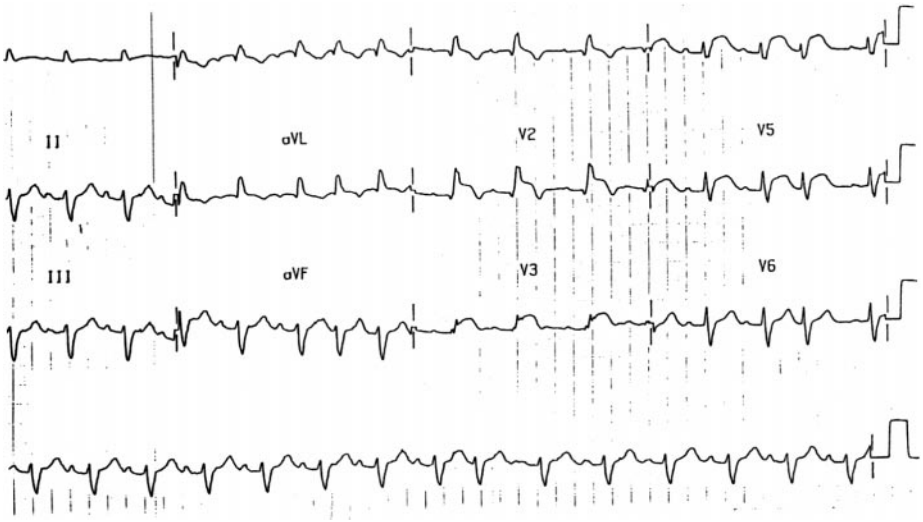

Figure 10. Right bundle branch block with acute myocardial infarction.

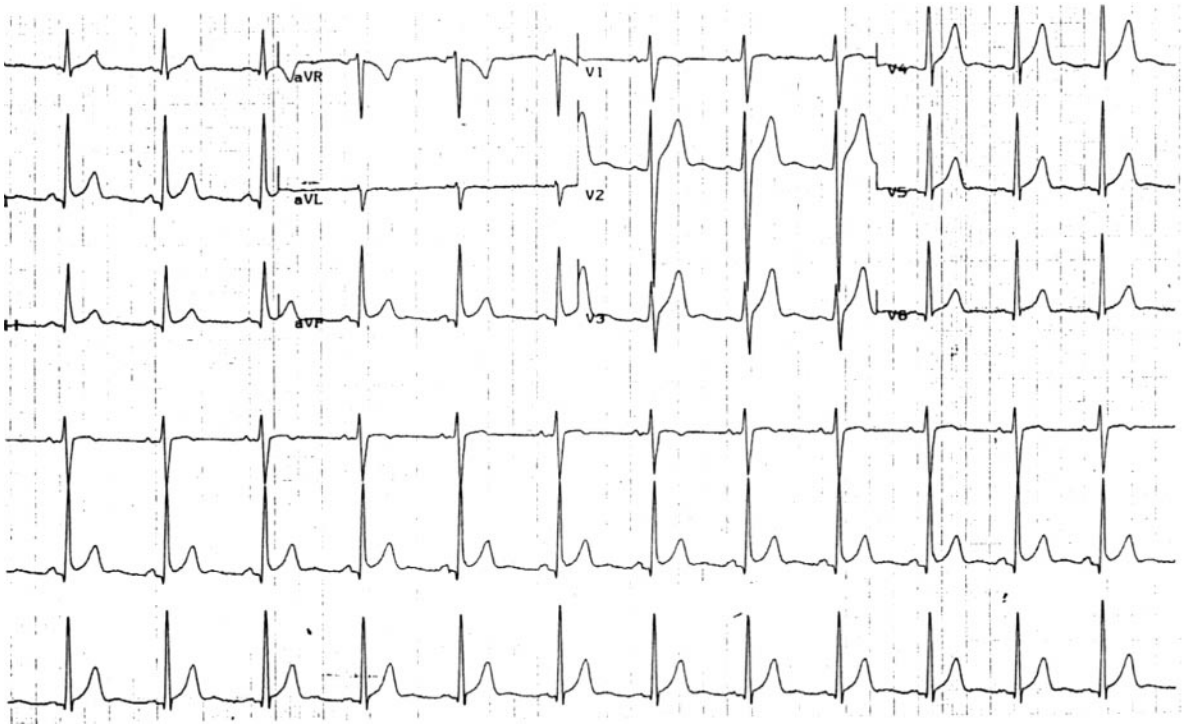

Figure 11. Benign early repolarization without acute myocardial infarction.

following: PGY 2–3, 193 (42%); and attending, 265 (58%) (Fig. 12). The rates of incorrect diagnosis ranged from 9% (LBBB with AMI [52 misinterpretations] and without AMI [52 misinterpretations]) to 72% (left ventricular aneurysm); Table 1 and Figure 13 show the rates of incorrect diagnosis by ultimate correct electrocardiographic diagnosis. Interpretations of RBBB without AMI, RBBB with AMI, inferoposterior AMI, and right ventricular paced rhythms were correct in all instances. The overall rate of correct interpretation of the study ECGs was 94.9% (4,782 correct interpretations out of 5,038 instances). Acute myocardial infarction with typical STE, ventricular paced rhythm, and RBBB were never misinterpreted. The remaining conditions were misinterpreted with rates ranging between 9% (LBBB, 52 misinterpretations) and 72% (LVA, 330 misinterpretations).

The overall rate of appropriate thrombolytic agent administration was 83% (1,525 correct administrations out of 1,832 indicated administrations). The leading diagnosis for which thrombolytic agent was given inappropriately was LVA (28%), followed by BER (23%), pericarditis (21%),

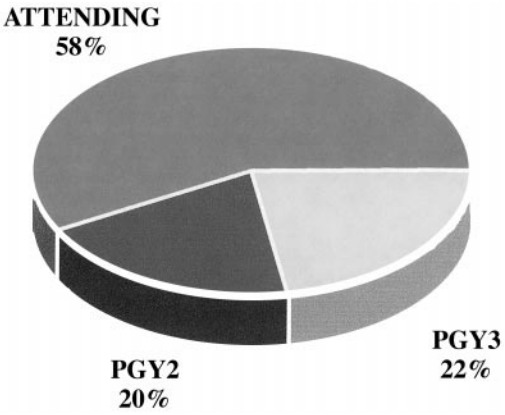

Figure 12. Level of physician training within the study population: 458 emergency physicians completed the questionnaire; levels of medical experience included the following: postgraduate year (PGY) 2 emergency medicine resident 90, PGY 3 emergency medicine resident 103, and attending physician 265.

and LBBB without electrocardiographic AMI (5%). Thrombolytic agent was appropriately given in all cases of AMI except when associated with atypical STE, where it was inappropriately withheld 67% of the time; Figures 14 and 15 show the rates of correct thrombolytic therapy administration and incorrect thrombolytic therapy administration by ultimate correct electrocardiographic diagnosis, respectively.

DISCUSSION

ST-segment elevation is perhaps the "most demanding" of the electrocardiographic features seen in the chest pain patient; it is "demanding" in that its presence must be explained and, if the etiology involves AMI, urgent therapeutic decisions must be made. Unfortunately, STE is a not uncommon finding on the ECG of the chest pain patient; its cause infrequently involves AMI. The occurrence of numerous other noninfarctional STE syndromes only reinforces the point that STE is a nonspecific marker for AMI.[6] One out-of-hospital study of adult chest pain patients demonstrated that the majority of patients manifesting STE on the ECG did not have AMI as a final hospital diagnosis; rather, LVH and LBBB accounted for the majority of the cases.[7] Further, in a review of adult ED chest pain patients with STE on the ECG, STE resulted from AMI in only 15% of this population; LVH, seen in 30% of adult chest pain patients, was the most frequent cause of this STE.[8] In the coronary care unit population, Miller et al.[9] demonstrated that STE was diagnostic for acute infarction in only half of patients with a past history of ischemic heart disease who present with chest pain and such ST-segment changes.

The observation that STE less often results from AMI in the adult chest pain patient in the ED[8] only reinforces the contention that the EP must be an expert in the interpretation of the ECG. The ECGs in this study represent challenging electrocardiograms. We believe that the EP should be able to determine the etiology of the STE in most, if not all, of these scenarios. It is likely that in a real scenario, other clinical clues from the history, examination, past medical records, and other investigations would have assisted the EP in determining the correct etiology of the STE. In this contrived, hypothetical situation, arriving at the correct diagnosis was more difficult.

Unfortunately, errors in patient evaluation and management do occur in this area of emergency medicine, at times with significant consequences for the patient and the EP. The various syndromes

TABLE 1. Incorrect Electrocardiographic Interpretations Relative to the Actual Cause of ST-segment Elevation (n = 458)

	Number of Incorrect Interpretations (%)								
Correct Interpretation	Incorrect Interpretations	LBBB	LBBB w/ AMI	LVH	BER	Pericarditis	LVA	AMI w/ Atypical STE	AMI
LBBB	52 (11)		24 (5)	19 (4)	0	0	0	0	9 (2)
LBBB w/ AMI	52 (11)	43 (9)		9 (2)	0	0	0	0	0
LVH	60 (13)	29 (6)	0		0	0	0	0	31 (7)
BER	243 (53)	0	0	27 (6)		101 (22)	0	0	115 (25)
Pericarditis	170 (37)	0	0	0	141 (31)		0	0	29 (63)
LVA	330 (72)	0	0	21 (5)	0	0		0	309 (46)
AMI w/ Atypical STE	289 (63)	0	0	169 (37)	99 (22)	21 (46)	0		0

LBBB = left bundle branch block; AMI = acute myocardial infarction; LVH = left ventricular hypertrophy; BER = benign early repolarization; LVA = left ventricular aneurysm; STE = ST-segment elevation.

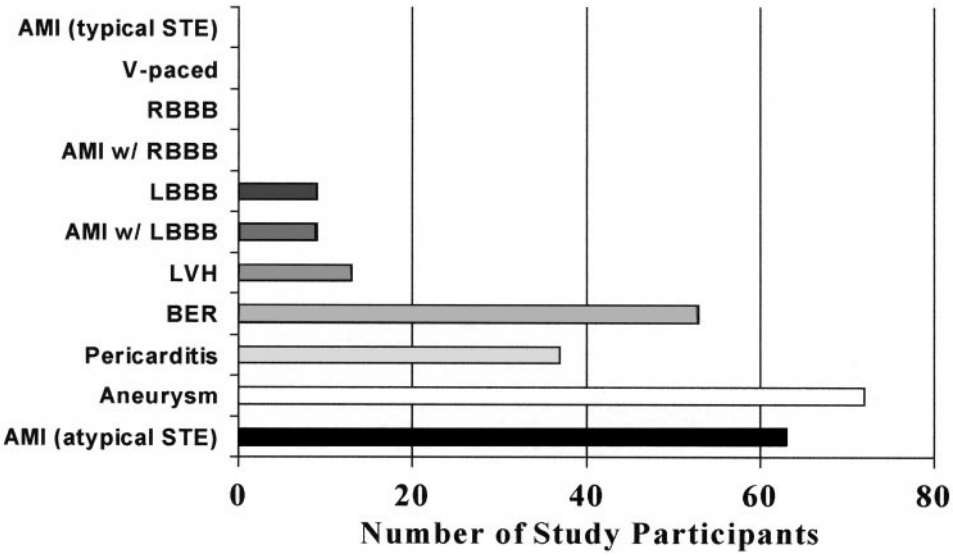

Figure 13. Rates of incorrect electrocardiographic diagnosis with respect to correct interpretation. AMI = acute myocardial infarction; STE = ST-segment elevation; V-paced = ventricular paced rhythms; RBBB = right bundle branch block; LBBB = left bundle branch block; LVH = left ventricular hypertrophy; BER = benign early repolarization.

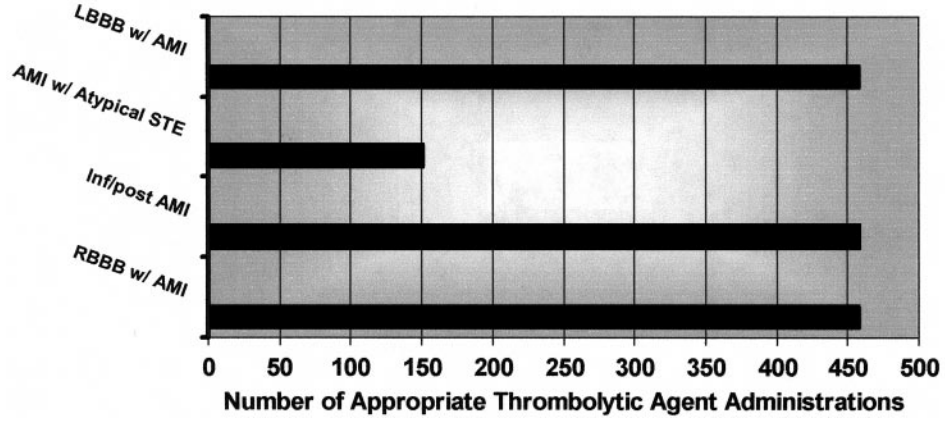

Figure 14. Rate of appropriate administration of thrombolytic agent by electrocardiographic diagnosis. LBBB = left bundle branch block; AMI = acute myocardial infarction; STE = ST-segment elevation; RBBB = right bundle branch block.

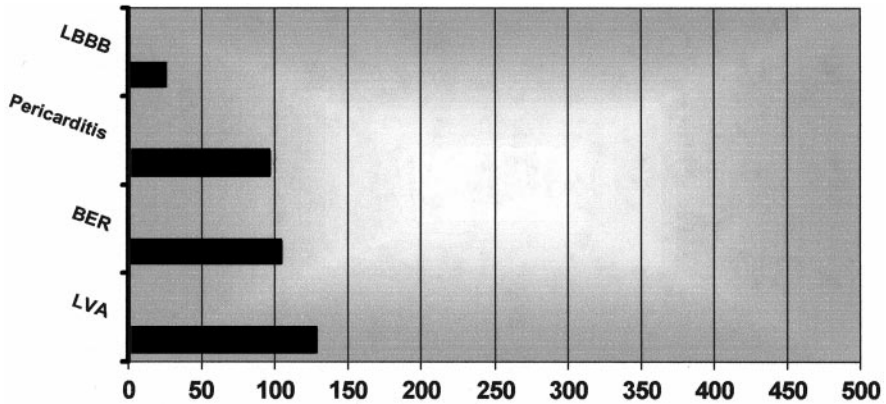

Figure 15. Rate of inappropriate administration of thrombolytic agent by electrocardiographic diagnosis. LBBB = left bundle branch block; BER = benign early repolarization; LVA = left ventricular aneurysm.

causing non-AMI STE may be misdiagnosed as acute infarction, which then may subject the patient to unnecessary and potentially dangerous therapies and procedures. For example, a report by Sharkey et al. noted that 11% of patients receiving thrombolytic agent were not experiencing AMI. The electrocardiographic syndromes producing this pseudo-infarct STE included benign early repolarization (30%), LVH (30%), and various intraentricular conduction abnormalities (30%).[10] In a recent investigation,[11] we reported that the rate of real-time misinterpretation of STE by EPs is quite low. In this study,[11] 202 patients had STE with 12 cases of electrocardiographic STE misinterpretation—a rate of 5.9% in this ED population. The most frequently misdiagnosed form of STE was LVA, which accounted for two of the cases and was initially believed to represent AMI. The BER pattern was the second most frequently misinterpreted STE entity, accounting for a total of three cases of which two were initially believed to represent pericarditis and one AMI. ST-segment elevation resulting from actual AMI was initially misidentified and incorrectly attributed to non-infarct etiology in two cases—one patient with BER and one with LVH. No instance of STE misinterpretation resulted in unnecessary acute reperfusion, while one patient with BER initially diagnosed as having AMI was admitted to the coronary care unit. This incorrect electrocardiographic interpretation by EPs has been noted in other reviews as well.[12–14]

These STE patterns may also confound the ED evaluation of the chest pain patient with electrocardiographic AMI and ST-segment abnormality. These patterns may either reduce the ECG's ability to detect STE or suggest to unwary physicians an incorrect cause of the electrocardiographic abnormality when, in fact, the patient is experiencing AMI. In the first instance, the classically reported confounding patterns include LBBB, LVH, and ventricular paced rhythms. These patterns produce noninfarctional ST-segment–T-wave changes that frequently mask the actual electrocardiographic abnormalities usually seen in AMI. In the less common case, the ECG of the patient actually experiencing AMI is confounded by changes such as atypical STE, which, if not recognized by the EP, may lead to the missed diagnosis of AMI. In the second case, the AMI-mimicking STE patterns may be incorrectly diagnosed as the responsible entity when acute infarction is present and responsible for the STE. Such patterns include BER, pericarditis, and LVA.

The issue of LBBB and AMI deserves special attention. The presence of LBBB markedly reduces the diagnostic power of the ECG. Common medical opinion holds that the electrocardiographic diagnosis of AMI is impossible in the presence of LBBB when, in fact, this diagnosis is often straightforward and considered "disarmingly easy" by others. A clinical decision rule has been developed to assist in the electrocardiographic diagnosis of AMI in the setting of LBBB using specific electrocardiographic findings.[5] Reportedly, three specific electrocardiographic criteria are independent predictors of myocardial infarction. The ECG criteria suggesting a diagnosis of AMI, ranked with a scoring system based on the probability of such a diagnosis, include 1) STE greater than 1 millimeter that is concordant with the QRS complex (Fig. 1, leads V5 and V6; a score of 5); 2) ST-segment depression greater than 1 millimeter in leads V1, V2, or V3 (a score of 3); and 3) STE greater than 5 millimeters that is discordant with the QRS complex (Fig. 1, leads V2, V3, and V4; a score of 2). A total score of 3 or more suggests that the patient is likely experiencing an AMI, while a score of less than 3 possibly indicates acute infarction and requires additional evaluation. In this study, Figure 1 depicts AMI in the setting of LBBB pattern with changes strongly suggestive of acute infarction. Figure 7 demonstrates a LBBB pattern without electrocardiographic AMI; this patient could be experiencing an AMI yet no electrocardiographic criteria are supportive of this diagnosis.

Incorrect interpretation of the ECG, particularly with respect to STE, may also impact the rate of missed AMI in the ED. A minority of AMIs (2% to 4%)[15–17] are inappropriately sent home from the ED. The vast majority of these cases involve young patients with unsuspected/atypical AMI and elder patients with atypical presentation. Misinterpretation of the ECG, however, is not infrequently implicated as a significant reason for inappropriate discharge of the AMI patient from the ED. According to the 1996 Physician Insurers Association of America questionnaire of the "missed myocardial infarction" malpractice claim,[18] the ECG was misinterpreted and/or incorrectly used in 25% of such cases. One large ED-based study investigating the missed AMI found that ECG misinterpretation accounted for 25% of undiagnosed cases.[15] A recent multicenter study researching this issue found a 2.1% rate of inappropriate discharge from the emergency department; 11% of these missed AMI cases reportedly manifested STE that was not noted.[16]

In this study, the most frequently misinterpreted STE patterns were LVA, AMI with concave (atypical) STE morphology, BER, and acute pericarditis; less frequently misinterpreted patterns included LVH and LBBB with and without AMI. The LVA pattern (Fig. 5) is most often noted after large anterior-wall myocardial infarctions but may also be encountered status-post inferior and pos-

terior wall myocardial infarctions. In most cases, the LVA is manifested electrocardiographically by varying degrees of STE, which may be difficult to distinguish from ST-segment changes due to AMI —particularly in the chest pain patient with known past myocardial infarction.[19] In this hypothetical model, it is not surprising that this particular example of LVA is misidentified as AMI. Of particular concern, many EPs in the study would have offered a thrombolytic agent to the patient for a presumed AMI. In a real-time interpretation of the ECG, the EP is able to use additional tools as to etiology of the STE—such as other historical features, the examination, and other diagnostic studies; this issue is true for all instances of misinterpretation in this study. This additional information likely explains the relative infrequency of this particular pattern among reports of incorrect electrocardiographic diagnosis of acute myocardial infarction.

The second most frequently misinterpreted STE pattern in this study involved AMI (Fig. 2) of the lateral wall with an atypical, or concave, STE morphology. In the majority of patients with AMI, the initial upsloping portion of the ST segment usually is either convex or flat; if the STE is flat, it may be either horizontally or obliquely so. Conversely, patients with non-infarctional STE tend to have a concave morphology of the waveform. This morphologic observation should only be used as a guideline. As with most guidelines, it is not infallible; patients with STE due to AMI may demonstrate concavity of this portion of the waveform.[8] This morphologic guide would also have not assisted the EPs in this particular case due to the concavity of the STE.

Benign early repolarization (Fig. 11) and acute pericarditis (Fig. 3) patterns were the next most commonly misdiagnosed STE patterns in this study EP population. Both electrocardiographic entities may present with pronounced changes, including prominent T waves and obvious STE. In fact, these two electrocardiographic diagnoses are often difficult to distinguish from one another as well as from AMI.[19,20] In this study, these patterns were treated with thrombolytic agent frequently—which can certainly cause significant morbidity particularly in the pericarditis patient scenario. Left ventricular hypertrophy and LBBB produced STE, which incorrectly suggested the electrocardiographic diagnosis of AMI to the study physicians. These two patterns are well known to hinder the diagnosis of AMI via ECG—both as masquerading and obscuring factors—and have been noted to cause similar diagnostic confusion in other situations.[8,10,19] Unfortunately, the LBBB pattern with electrocardiographic AMI was also misdiagnosed as non-AMI.

LIMITATIONS AND FUTURE QUESTIONS

This study is limited by several issues, primarily involving study design. First, the questionnaire structure of the study itself is a hypothetical, contrived situation—highly artificial—and very much unlike the actual ED encounter. In a real-time interpretation of the ECG, the EP has numerous other diagnostic tools that may assist in arriving at the correct etiology of the STE, such as an expanded history, past medical history, the physical examination, both prior and serial ECGs, various other diagnostic studies, and consultants. Clearly, the patient's history and physical examination are of particular value in reaching the appropriate diagnosis, both clinical and electrocardiographic. The chest pain patient with STE who appears pale and diaphoretic is likely experiencing an acute coronary ischemic event. Conversely, a complaint of chest pain, fully investigated by additional questions in the history and suggestive of a non-ischemic diagnosis, also assists the EP in the electrocardiographic interpretation. Essentially, the ECG is a test that must be interpreted in the context of that particular patient event. The study design clearly removed this option from the participants. Furthermore, past ECGs are invaluable, if available, in the evaluation of the chest pain patient with STE. The EP who evaluates the chest pain patient with a history of LVA and electrocardiographic STE clearly is assisted by past ECGs. Perhaps the lack of past ECGs in this study model accounts for the high rate of misinterpretation in the LVA patient scenario. Last, serial ECGs, whether via repeat ECGs or ST-segment trend monitoring, would also have assisted the EP in distinguishing between ischemic and non-ischemic causes of STE.

The EP study population comprised a convenience sample of physicians. In that these physicians were attending a lecture on the electrocardiographic diagnosis of AMI, they may have represented either a group of physicians with a particular interest in the ECG or, alternatively, a subset of EPs with a knowledge deficit in the topic. In either instance, this group of EPs may not represent the EP pool in general.

The major future issue involves educational programs aimed at EP instruction regarding electrocardiography. The fact that attending-level EPs fared better in terms of correct diagnosis with certain patterns may suggest that additional education (and experience) may improve a physician's diagnostic ability with respect to the ECG and STE. Structured educational programs may have an impact on correct diagnosis in resident-level EPs. Emergency physician electrocardiographic education must focus on the proper identification of

these STE syndromes so that appropriate therapies may be utilized. The AMI-mimicking and -confounding electrocardiographic patterns such as BER, LVA, acute pericarditis, LVH, and LBBB all represent significant diagnostic challenges. Furthermore, atypical ST-segment morphology associated with AMI must also be reviewed. Educational efforts, both at the resident level and during postgraduate continuing medical education for the attending physician, must focus on these issues, hopefully assisting in the correct identification of these electrocardiographic syndromes.

CONCLUSIONS

In this hypothetical model, certain electrocardiographic syndromes with STE were frequently misdiagnosed. These patterns included AMI with atypical ST-segment morphology, LVA, BER, acute pericarditis, LBBB, and LVH. In certain instances, the diagnosis of AMI was made when, in fact, a non-infarction etiology was responsible for the STE; the physicians indicated that thrombolysis was appropriate for these non-AMI STE syndromes. In other cases, AMI was present yet the EPs incorrectly attributed the STE to non-infarction causes.

The EP must be an expert in the interpretation of the ECG. Patients with chest pain and STE on the 12-lead ECG who are experiencing AMI may be candidates for urgent coronary revascularization via either thrombolysis or primary angioplasty. Such therapy, to maximize benefit, must be delivered as early as possible after the onset of infarction. Other such chest pain patients with electrocardiographic STE may be suffering from a noncoronary chest discomfort syndrome. The correct identification of these patients—both clinically and electrocardiographically—must be made in order to offer the most appropriate treatments and to avoid potentially dangerous therapies.

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