Additional Electrocardiographic Leads in the ED Chest Pain Patient: Right Ventricular and Posterior Leads

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In the evaluation of the patient with chest pain, the 12-lead electro cardiogram is a less-than-(ECG) perfect indicator of acute myocardial infarction (AMI), particularly when used early in the course of the acute ischemic event; this relative insensitivity for AMI results from many different issues, including a less-than-optimal imaging of certain areas of the heart. It has been suggested that the sensitivity of the 12-lead ECG can be improved if 3 additional body surface leads are used in selected individuals. Acute posterior (PMI) and right ventricular myocardial infarctions are likely to be underdiagnosed, because the standard lead placement of the 12-lead ECG does not allow these areas to be assessed directly. Additional leads frequently used include leads V_8 and V_9, which image the posterior wall of the left ventricle, and lead V_{4R}, which reflects the status of the right ventricle. The standard ECG coupled with these additional leads constitutes the 15-lead ECG, the most frequently used additional lead ECG in clinical practice. The use of the additional leads might not only confirm the presence of AMI, but also provide a more accurate reflection of the true extent of myocardial damage. (Am J Emerg Med 2003;21:563-573. © 2003 Elsevier Inc. All rights reserved.)

In the evaluation of the patient with chest pain, the electrocardiogram (ECG) is an invaluable tool, assisting the physician in establishing certain diagnoses, selecting both appropriate therapies and inpatient disposition locations, and predicting risk of complication and death. The 12-lead ECG, however, is a less-than-perfect indicator of acute myocardial infraction (AMI), particularly when used early in the course of the acute ischemic event. The sensitivity of a single 12-lead ECG for the diagnosis of AMI is relatively poor. For example, one report[1] examined the initial ECG in a group of adult patients with chest pain, all of whom were subsequently proven to have had an AMI. Less than 50% of these patients demonstrated ST-segment elevation (STE) on their initial ECG; the remaining patients manifested a variety of ischemic changes, including ST-segment depression and T-wave inversion. In 30% of these patients, the initial ECG was normal or showed nonspecific changes only. This same study demonstrated that ST-segment depression was a poor indicator of AMI; less than 50% of those patients who presented with isolated precordial ST-segment depression were subsequently shown to have had an AMI.

It has been suggested that the sensitivity of the 12-lead ECG could be improved if three additional body surface leads are used in selected individuals.[2,3] Acute posterior (PMI) and right ventricular myocardial infarctions are likely to be underdiagnosed, because the standard lead placement of the 12-lead ECG does not allow these areas to be assessed directly.[4] Additional leads frequently used include leads V_8 and V_9, which image the posterior wall of the left ventricle, and lead V_{4R} (alternatively termed R_{V4}), which reflects the status of the right ventricle.[5] The following case histories demonstrate the use of the additional electrocardiographic leads and the 15-lead ECG in clinical practice.

CASE PRESENTATIONS

Case No. 1

A 68-year-old man presented with chest pain radiating to the left arm of 3 hours' duration. He was diaphoretic and pale; the examination was otherwise unremarkable. The ECG (Fig. 1) revealed ST-segment depression, prominent R wave, and upright T waves in leads V_1 to V_3 in addition, ST-segment depression was seen in the inferior and lateral leads. Because of the anterior ST-segment depression and related findings, posterior wall AMI was suspected. Additional posterior leads (Fig. 2) were used, which demonstrated ST-segment elevation in leads V_8 and V_9 confirming acute posterior wall myocardial infarction. The patient was emergently transferred to the catheterization laboratory where a distal right coronary artery subtotal occlusion with thrombus was successfully opened through angioplasty and stenting. Ventriculography revealed posterior wall hypokinesis. Cardiac enyzmes were elevated establishing the diagnosis of AMI. The patient was admitted to the coronary care unit with an isolated posterior wall AMI.

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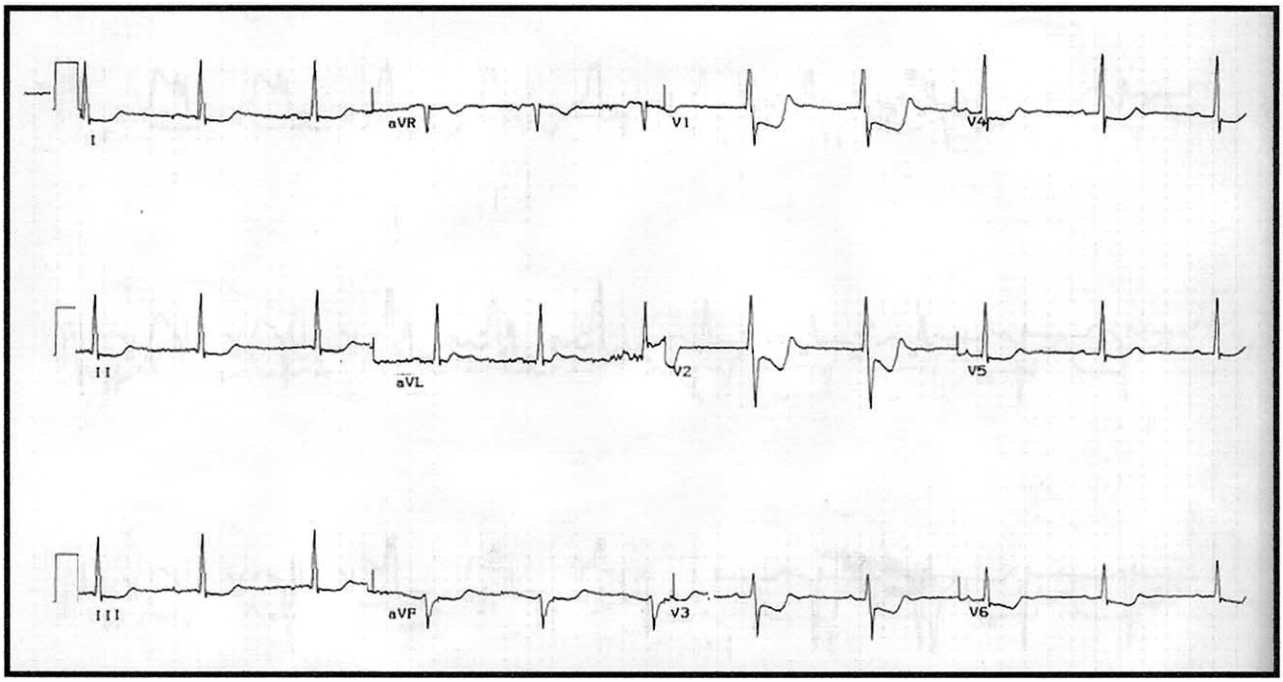

FIGURE 1. Case No. 1: The ECG revealed ST-segment depression, prominent R wave, and upright T waves in leads V_1 to V_3 in addition, ST-segment depression in the inferior and lateral leads was observed. This ECG is consistent with an isolated posterior wall AMI. Additional posterior electrocardiographic leads can be used to more completely investigate this issue.

Case No. 2

A 56-year-old man with a history of hypertension developed epigastric pain while working. On ED arrival he was pale, diaphoretic, and hypotensive with a blood pressure of 70 mm/Hg by palpation. An ECG was performed (Fig. 3), revealing sinus tachycardia with inferior and lateral ST-segment elevation as well as ST-segment depression, prominent R wave, and upright T waves in leads V_1 to V_3. These electrocardiographic findings were felt to be consistent with inferolateral wall AMI with possible posterior wall acute infarction. Additionally, the hypotension occurring in the setting of inferior wall infarction suggested the possibility of right ventricular infarction. Additional electrocardiographic leads (Fig. 4) including posterior and right ventricular leads were then recorded, revealing ST-segment elevation in leads V_8 and V_9 (indicating acute posterior wall infarction) as well as ST-segment elevation in lead V_{4R} (indicating right ventricular AMI). Fluid resuscitation was initiated, which corrected the systemic hypoperfusion. The patient received a thrombolytic agent with resolution of the chest discomfort and ST-segment elevation. Elevated cardiac enzymes confirmed the clinical diagnosis of inferoposterior AMI with right ventricle infarction.

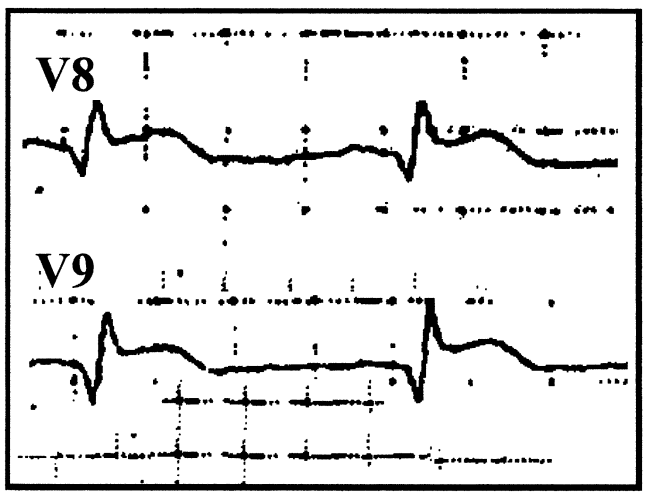

FIGURE 2. Case No. 1: Additional posterior leads were used that demonstrated ST-segment elevation in leads V_8 and V_9 confirming acute, isolated posterior wall myocardial infarction. Note that the magnitude of the ST-segment elevation in these leads is less pronounced.

Case No. 3

A 54-year-old woman with a history of hypertension presented to the ED with chest pain. The examination was remarkable for diaphoresis. While the ECG was being performed, the patient received nitroglycerin; subsequently, the patient developed emesis accompanied by hypotension. The 15-lead ECG (Fig. 5) demonstrated normal sinus rhythm with ST-segment elevation in the inferior leads as well as leads V_1 and V_{4R} consistent with an inferior wall AMI with right ventricular infarction. Normal saline was administered through a vein while a thrombolytic agent was given. The patient did not experience improvement; she was then transferred to the interventional suite where angioplasty was performed on multiple lesions (with thrombus) in the right coronary artery. Creatinine phosphokinase elevation (with positive MB fraction) confirmed the diagnosis of AMI;

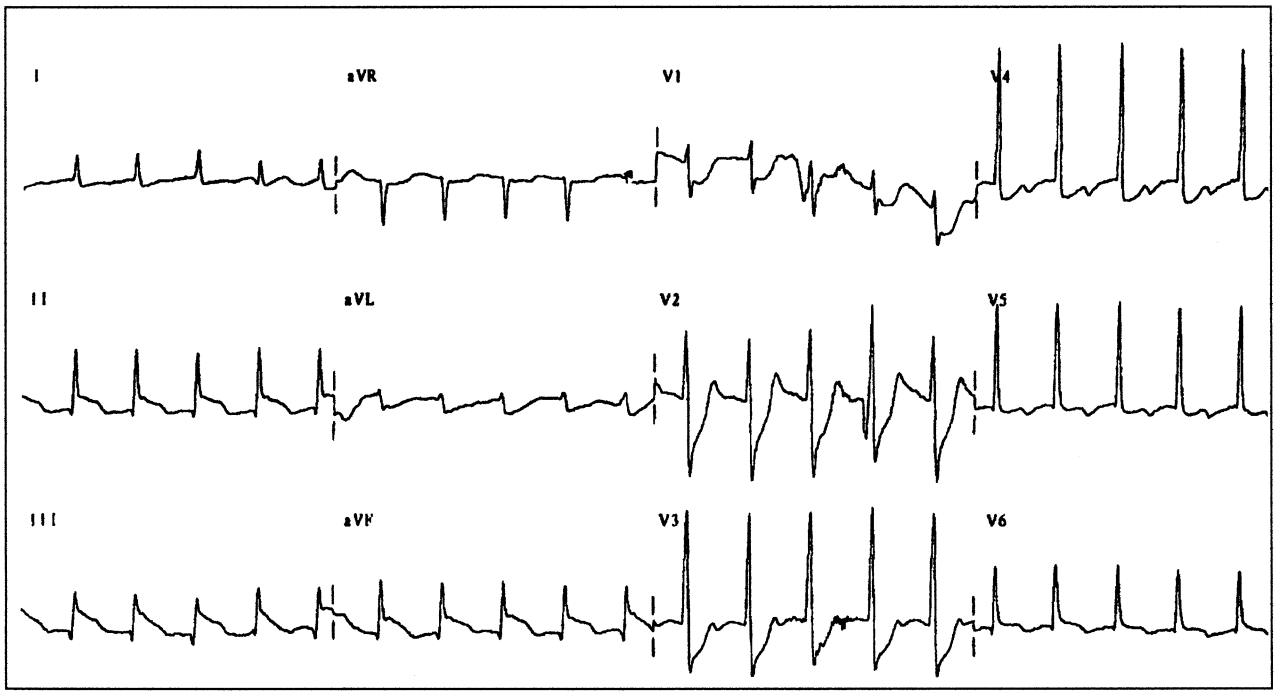

FIGURE 3. Case No. 2: An ECG revealed sinus tachycardia with inferior and lateral ST-segment elevation as well as ST-segment depression, prominent R wave, and upright T waves in leads V_1 to V_3 electrocardiographic findings consistent with inferolateral wall AMI with possible posterior wall acute infarction.

echocardiographic examination revealed hypokinesis of the inferior wall of the left and right ventricles.

Case No. 4

A 54-year-old women presented with substernal chest pain. The patient appeared pale and anxious. A 15-lead ECG (Fig. 6) demonstrated ST-segment elevation in leads V_8 and V_9 as well as minimal ST-segment depression with large R waves in the right precordial leads (V_1 to V_3), confirming posterior wall AMI. Heart catheterization revealed 90% distal right coronary artery occlusion with acute thrombus. The ventriculogram showed hypokinesis of the posterior walls; cardiac serum markers were elevated, confirming the diagnosis of acute, isolated posterior myocardial infarction.

Case No. 5

A 64-year-old man presented with chest pain. The symptoms had started while he was playing golf. He noted associated nausea, dyspnea, diaphoresis, and dizziness. He was a heavy tobacco user with a history of diabetes mellitus and hypertension. He was diaphoretic and pale; the examination was also remarkable for systemic hypotension. A 15-lead ECG (Fig. 7) revealed pronounced ST-segment elevation in leads II, III, and AVF, leads V8 and V9, and lead RV4, consistent with an inferoposterior AMI with right ventricular acute infarction. While awaiting transfer to the angioplasty suite, the patient developed ventricular fibrillation, which responded to a single electrical defibrillation at 200 watt-seconds. Cardiac serum markers were elevated, establishing the diagnosis of AMI. Ventriculography demonstrated akinesis of the inferior and posterior walls of the left ventricle and marked hypokinesis of the right ventricle.

DISCUSSION

The electrocardiogram is an essential tool for evaluating the patient with chest pain. The standard 12-lead ECG assists the physician in making certain diagnoses, selecting both appropriate therapies and inpatient disposition locations, and establishing prognoses. The 12-lead ECG, however, is an imperfect indicator of AMI. The 12-lead ECG has many limitations, a significant one being the detection of acute infarction of either the right ventricle or posterior wall of the left ventricle. This regional limitation can be reduced or entirely overcome by using additional electrocardiographic leads. The most widely used additional electrocardiographic leads include lead V_{4R} and leads V_8 and V_9. These additional leads more directly image areas of the heart that are relatively "ignored" by the standard 12-lead ECG; lead V_{4R} images the right ventricle and leads V_8 and V_9 image the posterior wall of the left ventricle. When used in conjunction with the 12-lead ECG, these additional leads comprise the 15-lead ECG.[4,6] Refer to Figure 8 for a depiction of the standard precordial leads and the previously mentioned additional leads and their relation to the cardiac structures.

The Right Ventricular Leads

Right ventricular (RV) infarction was initially identified in autopsy studies.[7] Cohn et al. were the first to describe RV infarction as the syndrome of hypotension, elevated jugular venous pressure, and shock in the presence of clear lung fields.[8] The incidence of RV infarction varies depending on

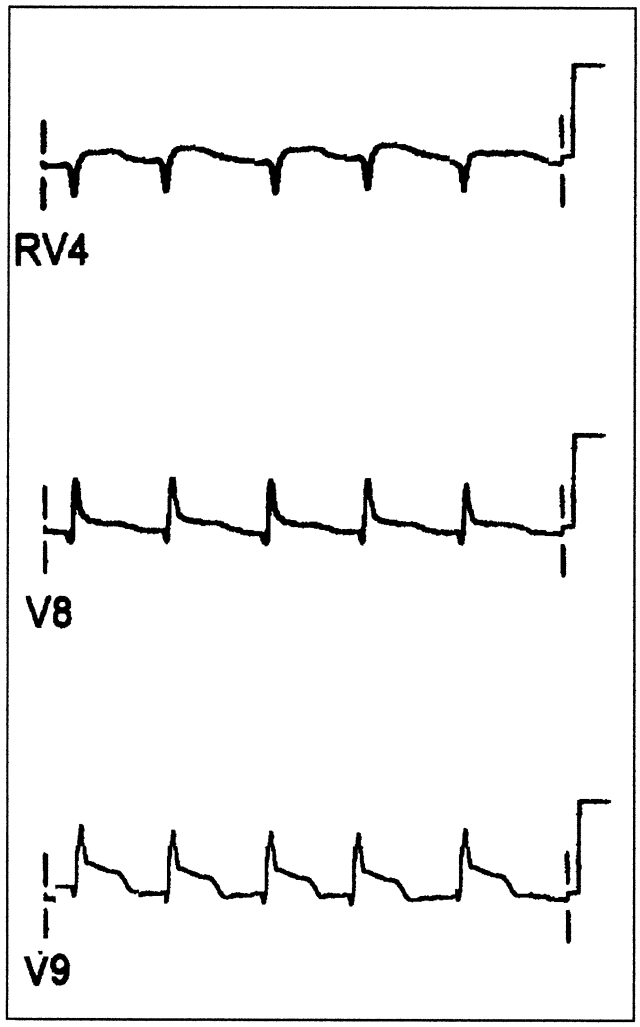

FIGURE 4. Case No. 2: Additional electrocardiographic leads, including posterior and right ventricular leads, were then recorded, revealing ST-segment elevation in leads V_6 and V_9 (indicating acute posterior wall infarction) as well as ST-segment elevation in lead V_{4R} (indicating right ventricular AMI). Clinical confirmation of a right ventricular infarction included hypotension likely resulting from reduced preload as a result of right-sided heart failure.

the method of detection, i.e., autopsy, invasive studies, or noninvasive imaging, including electrocardiography.[7] Electrocardiographic studies have consistently shown that RV infarction occurs in approximately one-third of inferior wall AMIs.[9-11] Isolated RV infarction is a rare phenomenon. The reason for these anatomic observations is that most RV infarctions result from occlusion of the right coronary artery proximal to the right ventricular branch. The left circumflex artery supplies the right ventricle in approximately 10% of patients. In this setting, RV infarction will present in the setting of a lateral wall AMI.

Several findings on the standard 12-lead ECG are suggestive of RV AMI, including ST-segment elevation (STE) in the inferior leads (II, III, and AVF)[12] or in the right precordial chest leads, particularly V_1, perhaps the only one of the standard 12 leads that reflects the right ventricle.[10,13-15] On occasion, coexisting AMI of the left ventricle's posterior wall can obscure the STE resulting from RV infarction in lead V_1 as seen in patients with the acute inferoposterior myocardial infarction with right ventricular involvement,[16-18] although Correale et al. propose that the opposite can also occur: extension of inferior AMI to the RV masking the ECG markers of posterior wall involvement.[19] A pattern of the relative magnitudes of the STE in the inferior leads suggest RV AMI. If STE is greatest in lead III compared with the other inferior leads, then RV infarction is suggested. Other suggestive findings with include the presence of a right bundle branch block,[20] second- and third-degree AV blocks,[21] and STE in lead V_2 greater than 50% the magnitude of ST-segment depression (STD) in lead aVF.[22]

The addition of lead V_{4R} provides additional objective evidence of RV involvement than can be noted on the 12-lead ECG. RV infarction can be diagnosed with 80% to 100% sensitivity by STE greater than 1 mm in lead V_{4R},[7,10,11] Robalino et al. found that STE greater than 1 mm in V_{4R} has 100% sensitivity, 87% specificity, and 92% predictive accuracy in detecting acute infarction of the RV resulting from occlusion of the right coronary artery above its first ventricular branch.[20] Figure 9 demonstrates STE in the right-sided leads. In all instances, the magnitude of the STE is less pronounced than is usually seen in the standard 12-leads of the ECG. This finding results from the fact the right ventricle is composed of considerably less muscle when compared with the left ventricle; with less myocardium manifesting a current of injury, the degree of STE is less. In Figure 9A, the entire right-sided leads V_{1R} to V_{6R} demonstrate an acute RV infarct; use of the single lead V_{4R} has demonstrated similar rates of RV infarct diagnosis when compared with the entire array of right-sided electrocardiographic leads.

Identifying RV involvement is important, because patients with coexisting inferior infarction have a large amount of jeopardized myocardium. Consequently, they are at increased risk for life-threatening complications, including high-grade AV blocks,[10] atrial fibrillation, symptomatic sinus bradycardia, atrial infarction (PR-segment displacement, elevation, or depression in leads II, III, or aVF), cardiogenic shock, cardiopulmonary arrest, or death.[7] In fact, the complication rate for this type of infarction is similar to the complication rate of anterior AMIs.[23] Aggressive therapy is often warranted in these patients to limit adverse events. Zehender et al. showed that patients with RV AMI diagnosed by STE in V_{4R} had significantly reduced mortality and inhospital complications if they received thrombolysis.[24] Nitrate-induced hypotension has also been well described in patients with RV AMI and can be prevented by using the additional V_{4R} lead.[25] Therefore, STE in lead V_{4R} should prompt the EP to restrict the use of nitrates and other vasodilating agents while judiciously administering crystalloid infusions to avoid systemic hypotension.

The Posterior Leads

The term posterior myocardial infarction (PMI) refers to AMI of the posterior wall of the left ventricle. This region is supplied by the left circumflex artery or by a dominant

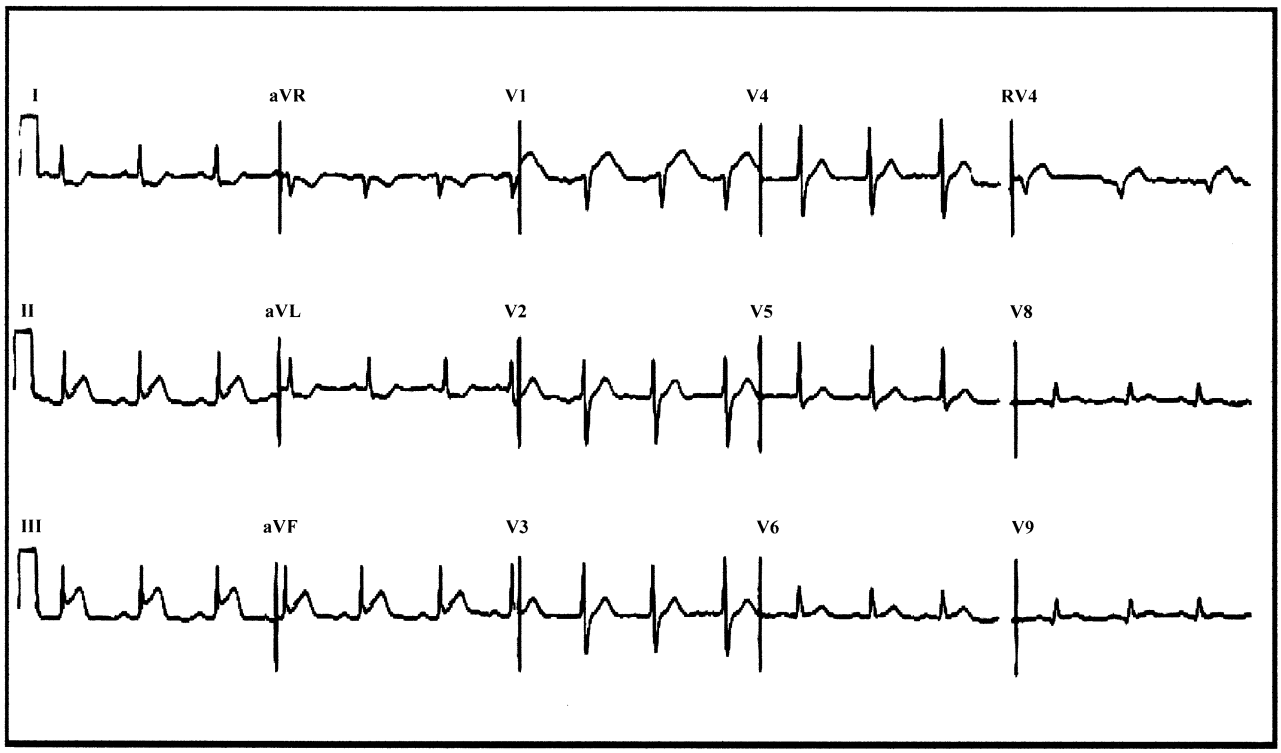

FIGURE 5. Case No. 3: A 15-lead ECG demonstrated normal sinus rhythm with ST-segment elevation in the inferior leads as well as leads V_1 and V_{4R} consistent with an inferior wall AMI with right ventricular infarction. Minimal ST-segment elevation is also seen in leads V_8 and V_9 potentially suggestive of early posterior wall AMI. The lack of appropriate findings in leads V_1 to V_3 suggestive of a posterior wall AMI is easily explained by the presence of an active right ventricular acute infarction and related ST-segment elevation in the right precordial leads.

right coronary artery with prominent posterolateral or posterior descending branches. Consequently, infarctions involving the posterior wall usually occur in conjunction with inferior or lateral AMIs and are estimated to occur in 15% to 21% of all infarctions.[26] Isolated PMIs occur less commonly. The rapid recognition of PMI is clinically important for several reasons. Patients with inferior or lateral wall AMI with concomitant PMI are experiencing a larger infarction, in essence, 2 walls of the left ventricle are involved. The risks of dysrhythmia, left ventricular dysfunction, and death increase proportionally with the size of the infarct. Therefore, it is reasonable that accompanying PMI is a factor for worsened prognosis with inferior or lateral AMI.[27-29] Also, urgent revascularization therapy might benefit patients with PMI accompanying inferior wall AMI[30] or isolated PMI.[5] A recent survey of cardiologists and EPs indicated that many of them would be more likely to administer thrombolytic agents if they were provided with electrocardiographic evidence suggestive of PMI.[31] However, the current literature on thrombolytic treatment for AMI has not specifically addressed isolated PMI, and therefore this caveat should be remembered when decisions to use thrombolysis are considered.

Using the standard 12-lead ECG to identify PMI can be both challenging and unrewarding. Electrocardiographic changes occur primarily in leads V_1 and V_2, occasionally extending to lead V_3, including: (1) horizontal STD; (2) a tall, upright T wave; (3) a tall, wide R wave; and (4) an R/S wave ratio greater than 1.0.[26] These changes seem more familiar when thought of as the reversal of the typical electrocardiographic findings indicative of transmural AMI. They are reversed for PMI because it is the endocardial surface of the posterior wall that faces the anterior precordial leads. In other words, STD, prominent R waves, and tall, upright T waves in leads V_1 to V_3 "when reversed," represent STE, Q waves, and T wave inversions, respectively, of PMI. When viewed in this manner, the electrocardiographic changes on the 12-lead ECG in PMI assume a more familiar, ominous significance. Coexisting infarctions to the inferior or lateral walls of the left ventricle are other electrocardiographic features that should stimulate consideration of acute PMI, particularly if these findings are accompanied by STD or prominent R waves in leads V_1 to V_3.

Evaluating the posterior wall through the 15-lead ECG is often more rewarding than the 12-lead evaluation. STE greater than 1 mm in leads V_8 and V_9 confirms the diagnosis of PMI; such a finding is more indicative of AMI than the anterior lead findings described here.[3,4,16] In fact, the sensitivity could be as high as 90% for identifying posterior AMIs[32] with a predictive accuracy up to 93.8%.[4] Also, false-positive STE in leads V_8 and V_9 occurs at the same frequency encountered in the 12-lead ECG.[26] However, the degree of STE could be significantly less in the posterior leads compared with the anterior leads because of the greater distance between the epicardial surface the surface leads as well as the amount of interposed tissues (Fig. 10).

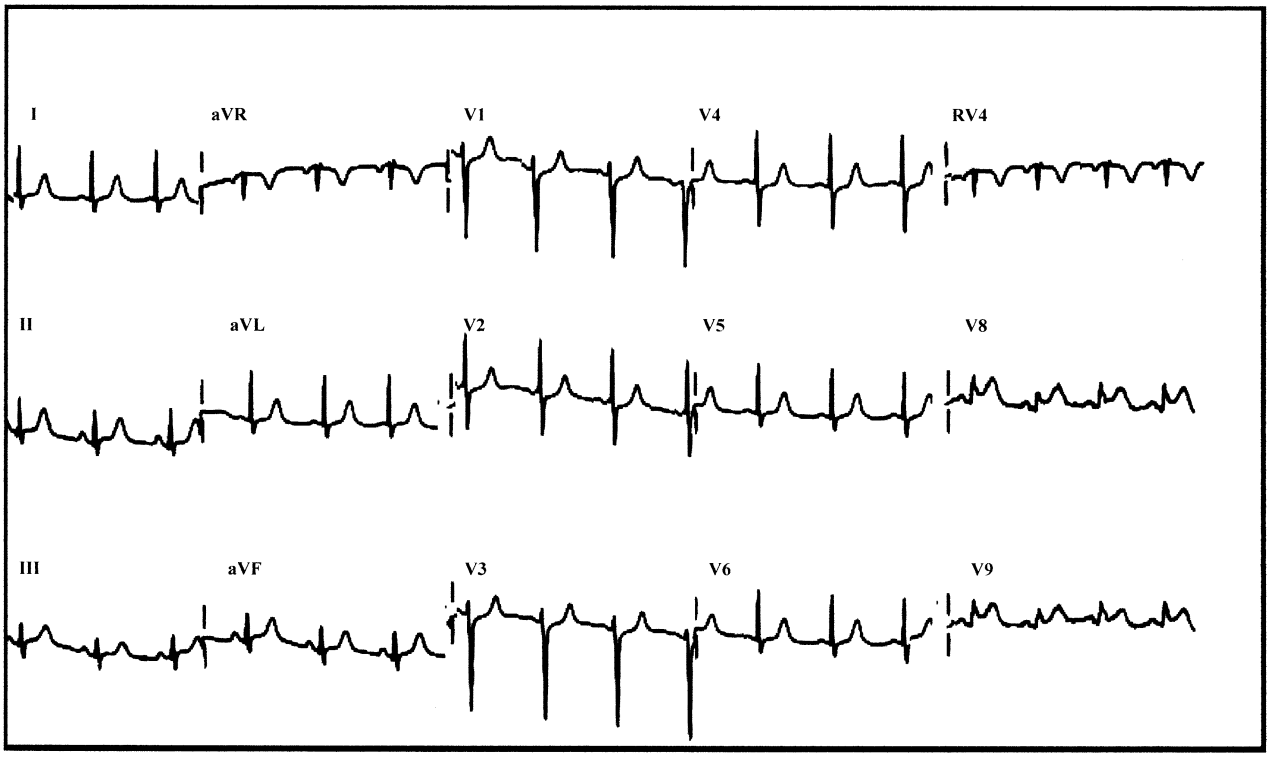

FIGURE 6. Case No. 4: A 15-lead ECG demonstrated ST-segment elevation in leads V_8 and V_9 as well as minimal ST-segment depression with large R waves in the right precordial leads (V_1 to V_3), confirming posterior wall AMI. The clinician should note the rather minimal ST-segment depression in leads V_1 to V_3.

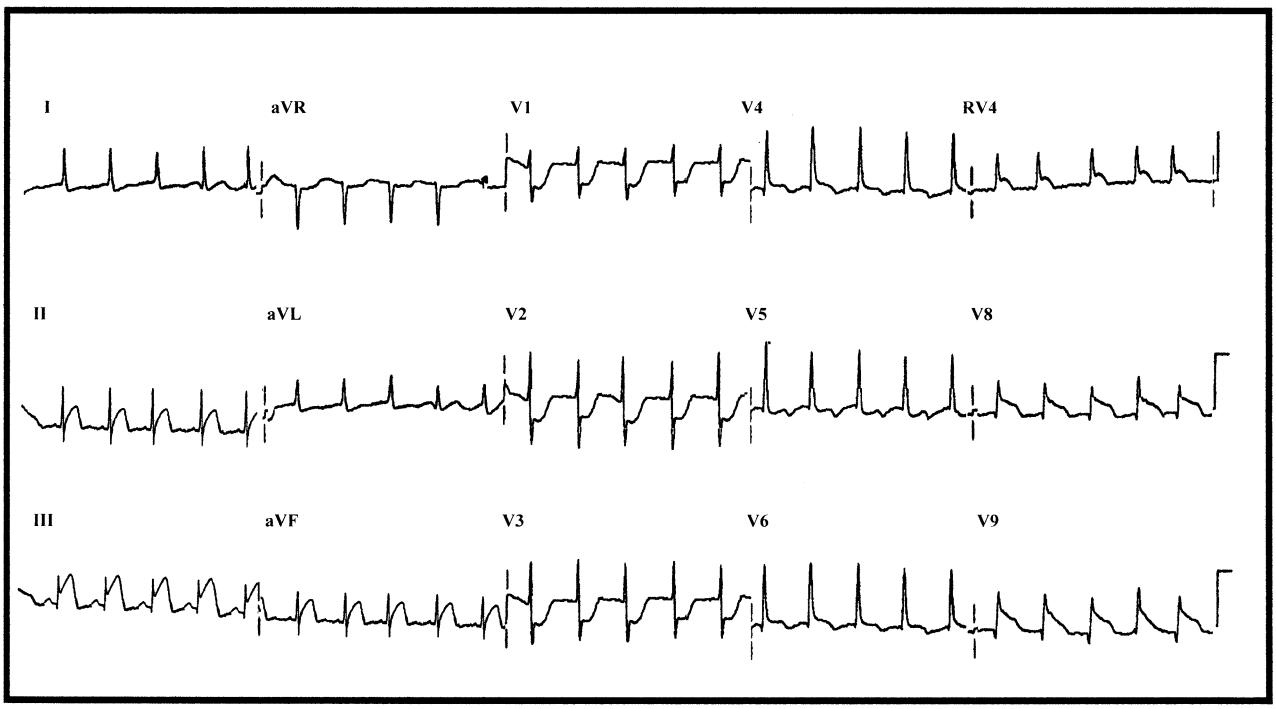

FIGURE 7. Case No. 5: A 15-lead ECG revealed pronounced ST-segment elevation in leads II, III, and AVF, leads V_8 and V_9 and lead V_{4R} consistent with an inferoposterior AMI with right ventricular acute infarction.

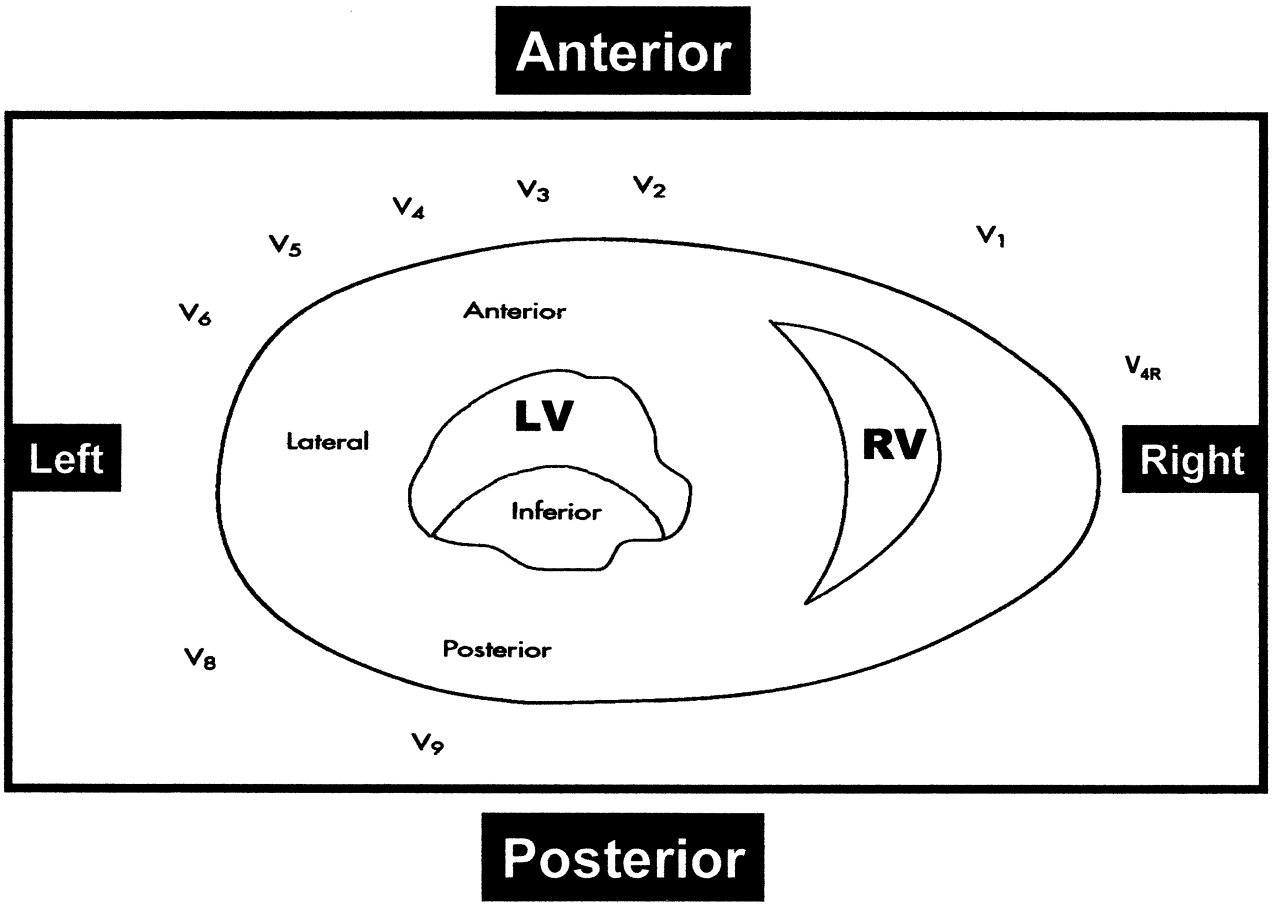

FIGURE 8. A depiction of the standard precordial leads and the additional leads (V_8, V_9 and V_{4R}) and their relation to the cardiac structures. Note that the posterior leads directly image the posterior wall of the left ventricle (LV), whereas leads the standard precordial leads V_1 to V_6 indirectly view the posterior wall of the LV. Also note that right ventricle (RV) is directly imaged only by the right-sided lead V_{4R} and, to a lesser extent, standard precordial lead V_1.

A recent increase in the use of posterior chest leads has indicated that the incidence of PMI is higher than once thought. The incidence, which was originally thought to be very rare, has been reported as high as 11% by detection with posterior chest leads.[2,3,5,33] Similar to RV AMIs and lead V_{4R}, electrocardiographic changes in leads V_8 and V_9 might provide information about the amount of myocardium involved and potential complications for both isolated PMI and PMI associated with inferior and/or lateral AMI. Oraii et al. demonstrated that patients with PMI who exhibit posterior lead STE have more frequent inhospital complications when compared with matched control subjects (odds ratio of 7 with confidence intervals of 1.28-28.43).[33]

Recently, valuable information regarding the pathophysiology and natural history of PMI has been revealed through electrocardiographic studies performed during coronary angiography. Khaw et al. demonstrated improved detection of PMI using the 15-lead ECG during single-vessel percutaneous transluminal coronary angioplasty of the right, circumflex, and left anterior descending coronary arteries.[34] Overall sensitivity during circumflex occlusion was 32% for the 12-lead ECG versus 69% for the 15-lead ECG when maximal STD in leads V_2 to V_3 was considered secondary to posterior wall injury. In a similar study, Wung et al. showed that adjusting the ischemic threshold from 1 to 0.5 mm of STE in leads V_7 to V_9 improved sensitivity for diagnosing PMI from 49% with the 12-lead ECG to 94% with the 15-lead ECG.[35] Future use of this lower criterion could increase the reported incidence of PMI from that previously mentioned greater than 11%.[2,3,5,33] Correale et al. suggest that higher inhospital mortality and complication rates found with RV involvement are actually related more to posterior extension that is masked by RV involvement rather than RV involvement itself.[19] They evaluated patients with inferior AMI using extra-lead ECGs (V_{3R} to V_{5R} and V_8 & V_9), cardiac enzymes, cardiac catheterization, and other methods. Comparing ECG markers, enzyme peaks, ejection fractions, and coronary scores, they concluded that the masking of ECG markers of posterior extension by RV involvement is likely the result of electrical balancing. All of these studies affirm the usefulness of the 15-lead ECG for diagnosing and predicting prognosis of PMI.

Although it is beyond the concentration of this report, it is worthwhile to at least mention the use of body surface mapping (BSM), the application of numerous additional leads. BSM has yielded a higher rate of diagnosing AMI than other additional lead ECGs, particularly in the "difficult-to-image" areas of the right ventricle and posterior

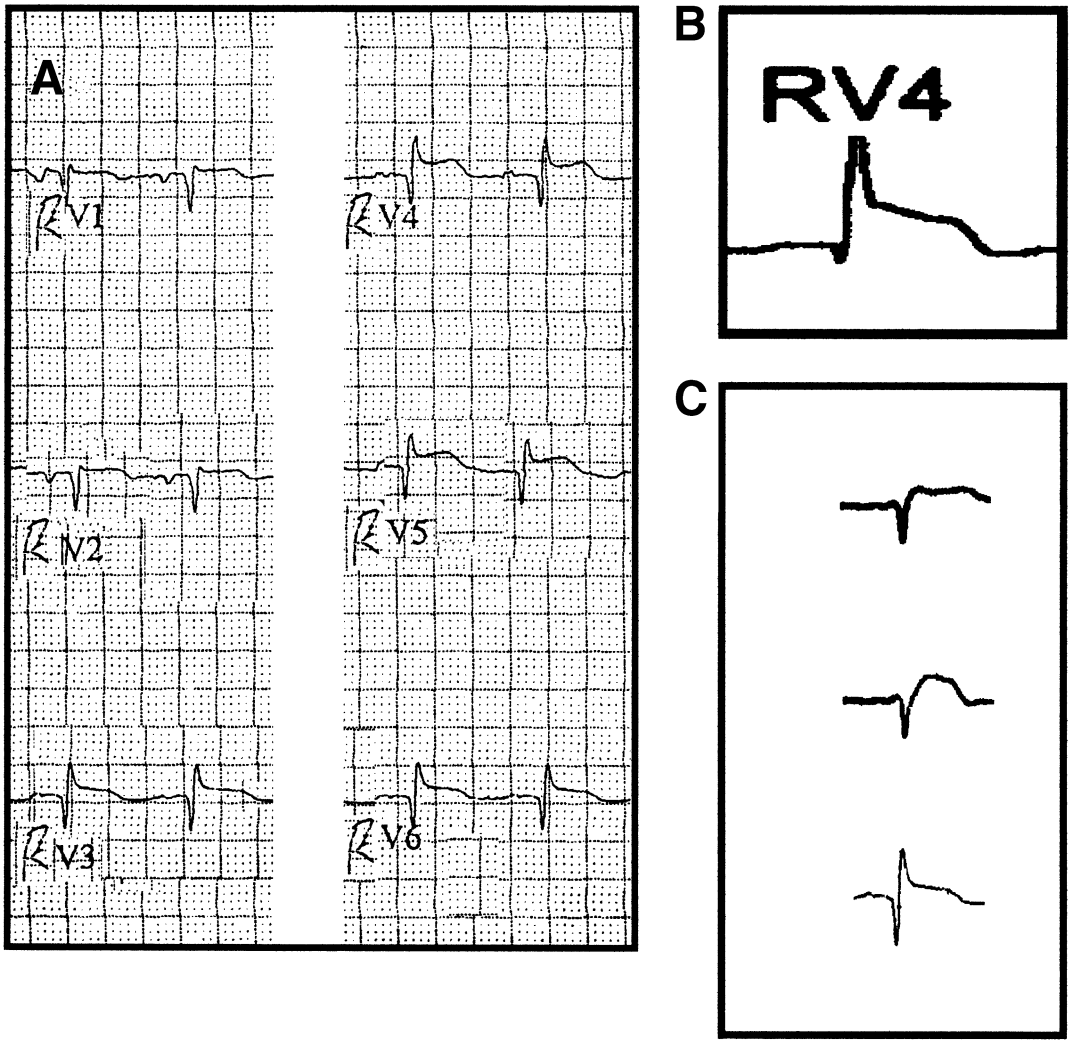

FIGURE 9. Right-sided electrocardiographic leads in patients with acute RV myocardial infarction manifested by ST-segment elevation. In all instances, the magnitude of the STE is less pronounced than is usually seen in the standard 12-leads of the ECG. This finding results from the fact the right ventricle is composed of considerably less muscle when compared with the left ventricle: with less myocardium manifesting a current of injury, the degree of STE is less. (A) The entire right-sided leads V_{1R} to V_{6R} demonstrate an acute RV infarct; use of the single lead V_{4R} has demonstrated similar rates of RV infarct diagnosis when compared with the entire array of right-sided electrocardiographic leads. (B and C) Various examples of STE in the patient with acute RV myocardial infarction.

wall.[26] For example, Menown et al compared the use of BSM with other additional leads, including V_7 to V_9 [36] They found higher rates of STE in the posterior region of the BSM compared with the number of posterior leads with STE. Additionally, they noted larger-sized infarcts in patients with abnormal posterior regions of the BSM compared with patients with STE in the "standard" posterior leads.

Indications

It is important to maintain a high index of suspicion for RV or posterior wall involvement when evaluating at-risk patients with the 12-lead ECG, particularly when only subtle abnormalities are present. The 15-lead ECG should be obtained when there is evidence of inferior AMI, lateral AMI, or STD in V_1 V_2 and/ or V_3 [18,37] The established specific indications for using 15-lead ECGs include the following: (1) STD or suspicious isoelectric ST-segments in leads V_1–V_3; (2) borderline STE in leads V_5 and V_6 or in leads II, III, and aVF; (3) all STE inferior AMIs (STE in leads II, III, and aVF); and (4) STE in leads V_1 and V_2 or isolated STE in lead V_1. Although they are less established, the other previously mentioned 12-lead ECG findings suggesting RV AMI should prompt V_{4R} investigation: right bundle branch block, second- and third-degree AV blocks, and STE in lead V_2 less than 50% the magnitude of STD in aVF. Additionally, inferior AMI presenting with hypotension is a strong indicator of coexistent RV infarction, although hemodynamic instability presents in the minority RV AMI cases.[7]

Limitations

STE in lead V_{4R} is represented by a rightward and anteriorly oriented vector. Consequently, leftward ST-segment

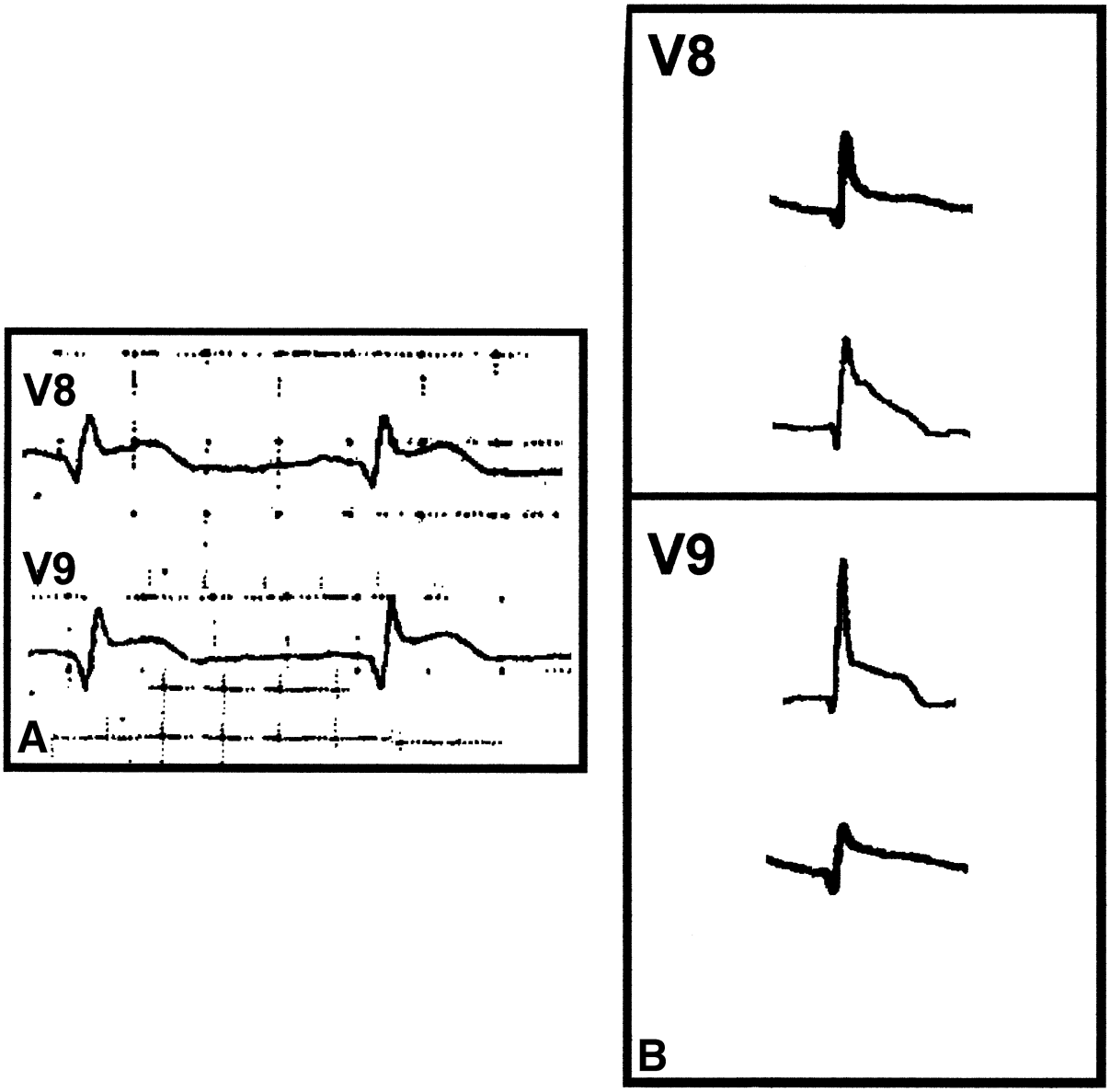

FIGURE 10. Posterior electrocardiographic leads directly image the posterior wall of the left ventricle. ST-segment elevation in these leads indicates acute infarction of the posterior wall of the left ventricle. Note that the magnitude of the elevation is less pronounced than is typically seen; this findings results from the relative distance of the posterior leads from the posterior wall of the LV. (A and B) Various examples of ST-segment elevation resulting from posterior wall ST-segment elevation AMI.

deviation, as is seen in leads V_5 and V_6 during a lateral AMI, could cancel the right-lead STE and obscure the diagnosis. Additionally, if STE is not prominent in the inferior leads, then it will be less prominent in V_{4R}.

There are also cases of inferior wall AMI with simultaneous RV infarction, but false-negative STE in lead V_{4R}. A study by Kosuge et al. suggests that this phenomenon results from concomitant posterior wall ischemia with a resultant current of injury that attenuates STE otherwise observed in the right precordial leads.[38] This study involved patients with first-time inferior wall AMI and total occlusion of the right coronary artery proximal to its first ventricular branch. Patients without STE greater than 1.0 mm in lead V_{4R} had a higher frequency of dominant right coronary arteries in addition to greater levels of STE in leads V_7, V_8, and V_9.

Several studies have suggested that the 15-lead ECG has greater sensitivity for identifying AMIs than the standard 12-lead ECG.[2-4,16,17] Two of these studies are particularly noteworthy. Zalenski et al, compared 12- and 15-lead ECGs in a prospective study that demonstrated an 11.7% increase in sensitivity of AMI diagnosis by STE from 47.1% to 58.8% with the use of additional leads.[16] Additionally, specificity remained unchanged and a 6-fold increase in the odds of meeting ECG thrombolytic therapy criteria was shown. Later work by Zelenski et al. revealed that additional lead analysis modestly increased diagnostic accuracy

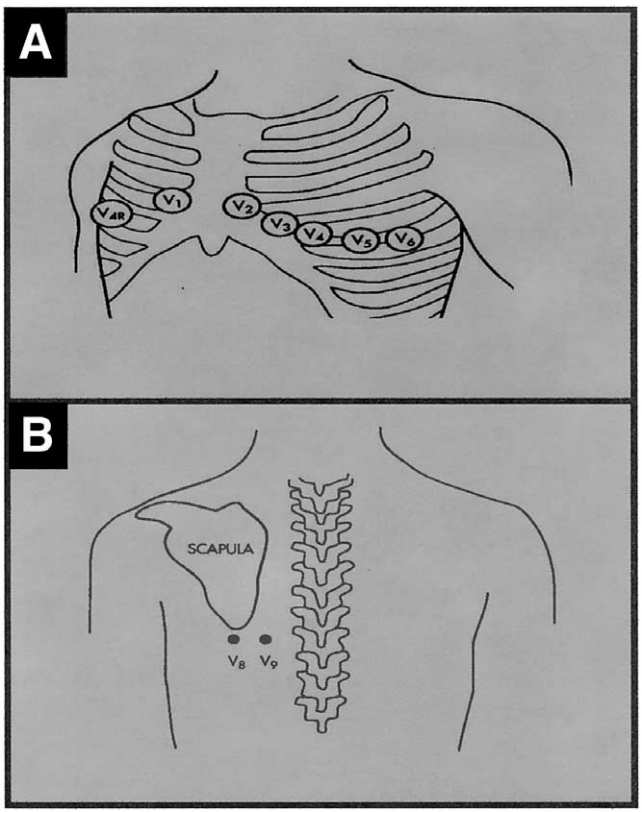

FIGURE 11. (A) Placement of the right ventricular ("right-sided") lead RV_4 on the right side of the chest in a position analogous to the left-sided lead V_4 (B) Placement of the posterior leads V_8 and V_9 on the patient's left back at the tip of the scapula (V_8) and one-half the distance between lead V_8 and the paraspinal muscles (V_9).

while it impacted therapy by increasing the administration of fibrinolytic agents.[17] The results of Zalenski et al.'s work are promising, although these results were obtained from a patient population with a high suspicion for acute coronary disease.

More recent work included patients with general chest pain in the ED, not just those with suspected AMI or unstable angina, and therefore a higher pretest probability for diagnosing AMI.[25] This prospective study with real-time physician survey showed that the 15-lead ECG did not alter diagnosis and management of acute coronary ischemic syndromes (AMI and unstable angina). However, physicians felt that the 15-lead ECG provided a more complete anatomic picture of the ischemic events; such opinion was also expressed in Zalenski et al.'s earlier work.[16,17] The authors concluded that all patients with chest pain in the ED do not require 15-lead ECG use, and therefore more studies are needed to identify the patient populations that will truly benefit from 15-lead ECG analysis. These populations are likely patients with right precordial lead STD and inferior or lateral AMIs, particularly those with hypotension.

Agarwal et al. performed posterior chest lead (V_7–V_9) on patients with chest pain consistent with a cardiac etiology, but without STE on the 12-lead ECG (they do not mention how many total patients were screened).[39] Of 58 such patients, 18 had posterior-lead STE greater than 1 mm or Q waves, and all of these 18 were confirmed to have had AMI by creatine phosphokinase criteria or cardiac catheterization. They concluded that all patients with suspected AMI, but nondiagnostic 12-lead ECG, should receive posterior lead analysis. This conclusion can be extrapolated to routine use of 15-lead ECGs for suspected AMI despite a nondiagnostic 12-lead ECG.

Performing the 15-lead ECG

The 15-lead ECG can be performed using the standard 12-lead ECG with movement of leads or through a specialized 15-lead ECG machine using 15 simultaneous electrocardiographic leads. The least expensive method involves use of the 12-lead ECG with subsequent lead manipulation and imaging of the additional cardiac areas. After performance of the standard 12-lead ECG, the additional 3 leads are placed as follows: placement of the right ventricular ("right-sided") lead RV_4 on the right side of the chest in a position analogous to the left-sided lead V_4 as seen in Figure 11B; and placement of the posterior leads V_8 and V_9 on the patient's left back at the tip of the scapula (V_8) and one-half the distance between lead V_8, and the paraspinal muscles (V_9) as seen in Figure 11A.

CONCLUSIONS

The 15-lead ECG is a valuable tool used to identify RV and posterior wall infarctions. Its primary benefits include information about the extent, severity, and potential complications of some AMIs, particularly in areas that might be missed by the standard 12-lead ECG. This can result in the identification of more patients who would benefit from more aggressive therapeutic regimens. It could also prevent the deleterious administration of nitrates in the patient with RV AMI.

The 15-lead ECG is easily performed and readily available in most EDs. Simply put, the generally accepted indications for use of the 15-lead ECG are inferior wall AMI, lateral wall AMI, and STD in leads V_1 to V_3 Other 12-lead ECG findings suggesting RV AMI (right bundle branch block, second- and third-degree AV blocks, and lead V_2 STE greater than 50% the magnitude of STD in lead aVF) should also prompt 15-lead ECG investigation. The role of the 15-lead ECG in routine ED practice remains questionable, and further clinical research is required to solve this controversy.

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