

# Assessing the Diagnostic Value of an ECG Containing Leads $V_{4R}$ , $V_8$ , and $V_9$ : The 15-Lead ECG

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**Study objectives:** To assess sensitivity, specificity, and odds ratios of ECG findings on leads  $V_{4R}$ ,  $V_8$ , and  $V_9$  for acute myocardial infarction.

**Design:** Prospective, two-stage cohort study.

**Setting:** A 660-bed university-affiliated community hospital.

**Type of participants:** One hundred forty-nine admitted patients with suspected myocardial infarction or unstable angina.

**Interventions:** Standard 12-lead ECG followed immediately by  $V_{4R}$ ,  $V_8$ , and  $V_9$ .

**Measurements:** Initial ECG findings of ST-segment displacement, Q waves, T-wave inversion, and eligibility for thrombolytic therapy.

**Results:** Major abnormalities (ST-segment deviation, T-wave inversion, Q waves) were found on the extra three leads in 28.9% (43 of 149) of patients. Sensitivity of ST-segment elevation for acute myocardial infarction on 12 versus 15 leads increased from 47.1% to 58.8%, respectively, with no decrease in specificity. McNemar's pair-matched analysis for ST-segment elevation on myocardial infarction subgroup showed an association of ST elevation with the 15-lead ECG ( $P < .05$ ). An eightfold increase in the odds of detecting ST elevation was found (90% confidence interval, 1.42 to 14.58); 22% of patients negative for ST elevation on 12 leads were positive on 15 leads. Analysis of ECG criteria for thrombolytic therapy presenting uniquely on extra leads showed an increased sensitivity from 35.3% to 44.1% on 12 versus 15 leads, respectively; there was a sixfold increase in the odds of meeting ECG thrombolytic therapy criteria (90% confidence interval, 0.34 to 11.66); 13.5% of patients not meeting criteria on 12 leads did so on 15 leads.

**Conclusion:** The 15-lead ECG provides increased sensitivity and odds of detecting ST-segment elevation in acute myocardial infarction patients with no loss of specificity; its use may expand the selection of thrombolytic therapy candidates and provide a

fuller ECG description of the extent of myocardial injury and necrosis.

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## INTRODUCTION

The ECG is essential to the evaluation of the patient with suspected myocardial ischemia. Patterns of injury and necrosis are highly specific,<sup>1</sup> provide essential information for the decision to use thrombolytic therapy,<sup>2-4</sup> and distinguish patients at high and low risk for complications from myocardial infarction.<sup>5,6</sup> The 12-lead ECG, however, is an imperfect predictor of acute myocardial infarction. Its sensitivity is poor; nonspecific or normal ECGs have been found in as many as 30% of patients with proven infarction.<sup>7</sup> Fewer than half of patients with a confirmed infarct have an initial ECG demonstrating ST-segment elevation.<sup>7</sup> ST-segment depression, in particular, is a poor predictor of infarction and up to half of the patients with isolated precordial ST-segment depression on presentation do not demonstrate evidence of infarction.<sup>7</sup>

These limitations of the 12-lead ECG may be explained partly by its poor detection of posterior walls<sup>8</sup> and right ventricular infarctions. These areas are not assessed directly by standard lead placement but are examined by posterior leads  $V_8$  and  $V_9$ <sup>9</sup> and the right ventricular lead  $V_{4R}$ .<sup>10</sup> Posterior myocardial infarction is one of the most commonly missed ECG findings,<sup>9</sup> and this may be explained by this lack of direct ECG examination.

This study was designed to determine whether the sensitivity, specificity, predictive value, and other diagnostic characteristics of the ECG for detection of myocardial infarction in the emergency department could be improved by expanding the ECG from 12 to 15 leads.

## MATERIALS AND METHODS

From August 8, 1986, to March 31, 1987, patients presenting to the ED of a 660-bed suburban, university-affiliated teaching hospital were eligible for the study. Patients presenting with chest pain, shortness of breath, diaphoresis, or weakness were eligible for screening with an expanded 15-lead ECG if they presented between the hours of 5 AM and 11 PM (the hours during which ECG technicians were available). All patients who were screened with a 15-lead ECG and admitted to the coronary care unit or telemetry unit with a diagnosis of myocardial

infarction or unstable angina were studied. Patients were not included if they were younger than 18 years, admitted to the hospital with provisional diagnoses other than myocardial infarction or unstable angina (such as syncope, congestive heart failure, gastrointestinal bleed), admitted to a non-cardiac-monitored bed, or discharged. The study received expedited review and was approved by the institutional review board of Lutheran General Hospital, with a provision for deferred consent on patients after the extra leads were recorded.

A standard 12-lead ECG was performed, followed immediately by a recording of leads  $V_{4R}$ ,  $V_8$ , and  $V_9$  (15-lead ECG). ECG technicians performed all studies after receiving instruction on lead placement. The posterior leads were placed at the level of the anterior 5th intercostal space,  $V_8$  under the midscapular line and  $V_9$  at the left paraspinal border.  $V_{4R}$  was placed on the right anterior chest opposite to the corresponding left chest placement of lead  $V_4$ .

ED attending and resident physicians completed prospective data collection sheets at the time of their initial patient evaluations. Data collected included age, gender, race, presenting symptom, history of myocardial infarction or angina, duration of pain, presence of pain during the ECG, degree of suspicion of myocardial infarction, and disposition.

Hospital charts were reviewed for complications and interventions. Myocardial infarction was recorded as positive if the attending physician recorded a discharge diagnosis of myocardial infarction and if this was confirmed by chart review that documented either a rise of creatinine kinase MB/CK of 5% or more or ECG findings of new pathologic Q waves (0.04 seconds or more) or existing Q waves if accompanied by ST-segment elevation. Otherwise, myocardial infarction was recorded as negative. For unstable angina, the attending physician's discharge diagnosis was accepted.

Total creatinine kinase measurements were performed on Kodak's Ektachem Analyzer (Rochester, New York), an enzymatic rate method; normal range was 50 to 150 IU/mL. Creatinine kinase isoenzymes were performed either immediately by electrophoresis (August 1986 to January 1987) or by initial immunometric screening measurements (January 1987 to March 1987) on the analyzer followed by confirmatory electrophoresis on all positive MB/CK screens. The usual schedule of creatinine kinase measurements on admission to the hospital is every eight hours repeated three times. A random sample of 10% (15) of the study cohort found that 86.7% had enzymes drawn

at least three times in the first 24 hours and 93.5% within 36 hours.

The initial ED ECGs, including the three added leads, were interpreted by a cardiologist from whom the study

patient clinical outcomes were withheld. ST-segment deviation, T-wave inversion (symmetric), and pathologic Q waves (40 msec or more) were recorded for each of seven possible infarct sites (anteroseptal, anterior, inferior,

**Table 1.**

Measurements of test validity; presence of any ST-segment elevation versus final diagnosis of acute myocardial infarction (AMI)\*

	15 Leads				15-Lead Calculations		
	+AMI	-AMI	Total				
+STE <sup>†</sup>	20	8	28	SE <sup>‡</sup> =	20/34 =	58.8	
-STE	14	107	121	SP =	107/115 =	93.0	
<b>Total</b>	<b>34</b>	<b>115</b>	<b>149</b>	PV+ =	20/28 =	71.4	
				PV- =	107/121 =	88.4	
				AC =	127/149 =	85.2	
				LR =	.588 (1-.93)	8.4	
				PTO =		67.7%	

	12 Leads				12-Lead Calculations		
	+AMI	-AMI	Total				
+STE	16	8	24	SE =	16/34 =	47.1	
-STE	18	107	125	SP =	107/115 =	93.0	
<b>Total</b>	<b>34</b>	<b>115</b>	<b>149</b>	PV+ =	16/24 =	66.7	
				PV- =	107/125 =	85.6	
				AC =	123/149 =	82.6	
				LR =	.471 (1-.93) =	6.7	
				PTO =		62.7%	

\*All subjects (AMI prevalence = 34/149 = 22.8%).

<sup>†</sup>+STE, positive for ST-segment elevation; -STE, negative for ST-segment elevation; +AMI, confirmed AMI; -AMI, negative AMI.

<sup>‡</sup>SE, sensitivity; SP, specificity; PV+, positive predictive value; PV-, negative predictive value; AC, accuracy; LR, likelihood ratio; PTO, post-test odds.

**Table 2.**

Measurements of test validity; thrombolytic therapy criteria versus final diagnosis of acute myocardial infarction (AMI)\*

	15 Leads				15-Lead Calculations		
	+AMI	-AMI	Total				
+TRX <sup>†</sup>	15	1	16	SE <sup>‡</sup> =	15/34 =	44.1	
-TRX	19	114	133	SP =	114/115 =	99.1	
<b>Total</b>	<b>34</b>	<b>115</b>	<b>149</b>	PV+ =	15/16 =	93.8	
				PV- =	114/133 =	85.7	
				AC =	129/149 =	86.6	
				LR =	.44 (1-.99)	44.1	

	12 Leads				12-Lead Calculations		
	+AMI	-AMI	Total				
+TRX	12	1	13	SE =	12/34 =	35.3	
-TRX	22	114	136	SP =	114/115 =	99.1	
<b>Total</b>	<b>34</b>	<b>115</b>	<b>149</b>	PV+ =	12/13 =	92.3	
				PV- =	114/136 =	83.8	
				AC =	126/149 =	84.6	
				LR =	.353 (1-.99)	35.5	

\*All subjects (AMI prevalence = 34/149 = 22.8%).

<sup>†</sup>TRX, ECG-eligible for thrombolytic therapy; -TRX, not ECG-eligible for thrombolytic therapy; +AMI, confirmed AMI; -AMI, negative AMI.

<sup>‡</sup>SE, sensitivity; SP, specificity; PV+, positive predictive value; PV-, negative predictive value; AC, accuracy; LR, likelihood ratio.

lateral, posterior [reciprocal], posterior indicative, right ventricular). ST-segment elevation was recorded as present if 1.0 mm (0.1 mV) or more in at least one lead, but 0.5 mm or more was accepted in the posterior leads if the R waves were of low (less than 10 mm) amplitude. Previous ECGs were not used for comparison.

All tracings with ST-segment elevation on the extra leads were evaluated to determine if they met ECG criteria for thrombolytic therapy. For this, patients were required to have at least 1.0 mm of ST-segment elevation in two anatomically contiguous leads. Posterior leads were considered contiguous to inferior or lateral leads, and  $V_{4R}$  was considered contiguous to inferior leads. For purposes of this analysis, patients were classified as 12-lead ECG-eligible or not. Patients not eligible on 12-lead ECG were classified further as uniquely eligible on 15-lead ECG. Uniquely eligible on 15 leads occurred if the patient did not meet the ECG criteria on 12 leads but did so when all 15 leads were considered.

During the study period, all admissions for "rule-out myocardial infarction" to the coronary care and telemetry units were reviewed from a computer-generated list from the hospital's mainframe computer. The rate of infarction in the admitted "rule-out myocardial infarction" group patients who were not studied was compared with the rate of myocardial infarction in the subgroup of studied

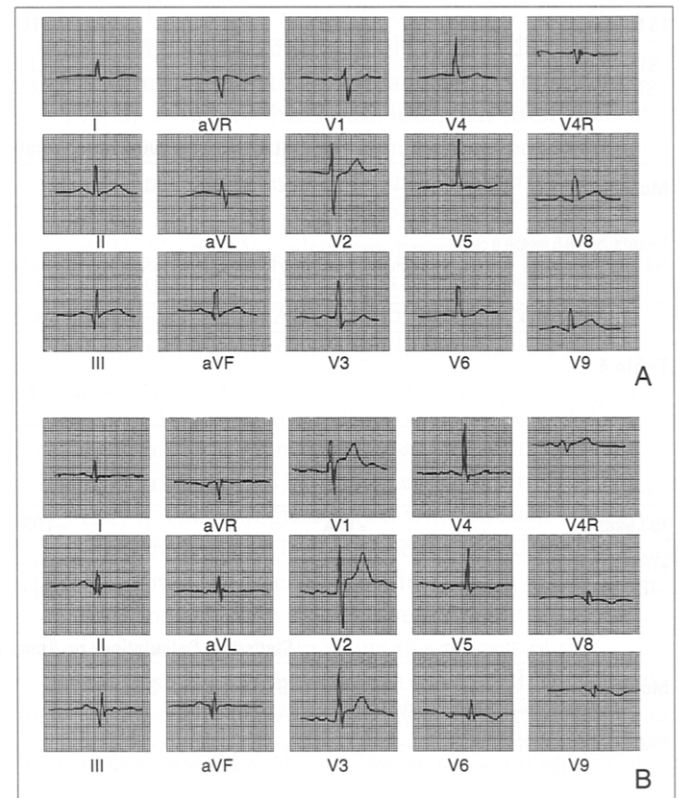
patients with the admission diagnosis of "rule-out myocardial infarction" to address potential sampling bias.

On the full study population (Tables 1 and 2), measurements such as sensitivity and specificity for the 12- and 15-lead ECGs were obtained by comparing two types of ECG findings with the diagnosis of acute myocardial infarction. These are "ST-segment elevation" and "unique eligibility for thrombolytic therapy." For each criterion, we calculated sensitivity, specificity (with 95% confidence intervals), positive and negative predictive values, false-positive rates, false-negative rates, accuracy of correct classification (true-positives and true-negatives/total sample), and the likelihood ratio (true-positive/false-positive rate).

Using a hypothetical 20% pretest probability of myocardial infarction detection before the ECG, we compared the post-test probability of myocardial infarction detection

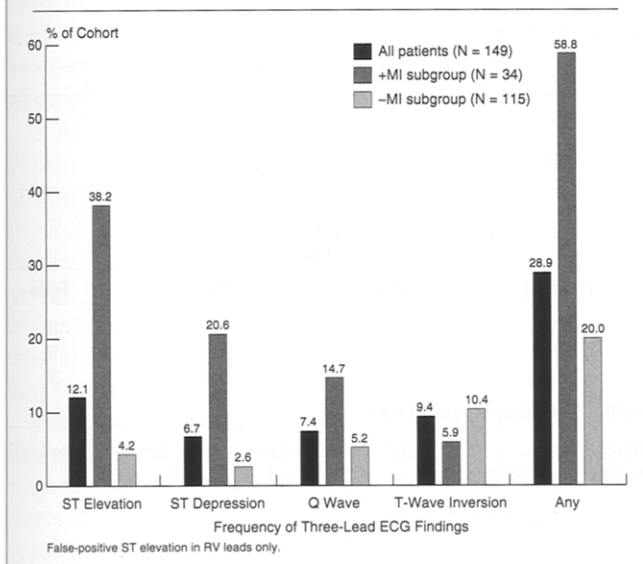
**Figure 2.**

Example of 15-lead tracing A, which shows ST-segment elevation (ECG thrombolytic criteria) solely on posterior leads. Patient had peak MB/CK of 159 of 1,817, three-vessel disease on cardiac catheterization with inferior-posterior regional hypokinesia; B, follow-up three-lead tracing 26 hours later shows evolution of Q waves with T-wave inversion.



**Figure 1.**

Histogram showing frequency of abnormal ECG findings on  $V_{4R}$ ,  $V_8$ , and  $V_9$  as a percentage of total study population, acute myocardial infarction subgroup, nonmyocardial infarction subgroups.



for the 15- and 12-lead tests using the likelihood ratio and Bayesian methods as described by Radack et al.<sup>11</sup> The likelihood fraction is a ratio of the true-positive to the false-positive proportion of test results. If this ratio is more than 1, the patient has a higher probability of the condition.

On the subsample of myocardial infarction patients (Tables 3 and 4), a conventional evaluation of the 12-versus 15-lead ECG was made using the McNemar's  $\chi^2$  test for pair-matched samples. The formula is given by  $\chi^2_m = (b - c)^2 / (b + c)$ , without the Edwards correction factor.<sup>12,13</sup>

Using discordant pairs analytic procedure,<sup>14</sup> we generated the odds ratio ( $b/c$ , substituting 0.5 for zero cells) and the standard error of the odds ratio with 90% confidence intervals. We also calculated a percentage improvement fraction, given by the formula  $(b - c) / (b + d)$ .<sup>12-15</sup> The improvement fraction reports as a percentage those who are negative on one test outcome and are positive on the

second (eg, negative on 12 leads for ST elevation but positive on 15 leads).

RESULTS

During the study period, 149 patients underwent a 15-lead ECG and were admitted to a cardiac-monitored unit with a provisional diagnosis of myocardial infarction (109) or unstable angina (40). The mean age was 63.9 (SD  $\pm$  12.9 years); 56.4% were men and 97.3% white. In the 149 patients admitted as "rule-out myocardial infarction" or "unstable angina," acute myocardial infarction was diagnosed in 22.8% (34 of 149). In the subgroup of patients admitted as "rule-out myocardial infarction," 28.4% (31 of 109) had a confirmed myocardial infarction; in the subgroup admitted as "unstable angina," 7.5% (three of 40) had a confirmed myocardial infarction.

When the 109 patients admitted as "rule-out myocardial infarction" who were studied are compared with the "rule-out myocardial infarction" not entered into the study

Table 3.

Comparison of the presence of any ST-segment elevation on 12 versus 15 leads in myocardial infarction subgroup using McNemar's  $\chi^2$

Data Array 12 Leads			Actual Data 12 Leads			
15 Leads	+STE*	-STE	15 Leads	+STE	-STE	Total
+STE	CELL a	CELL c	+STE	16	4	20
-STE	CELL b	CELL d	-STE	0	14	14
			<b>Totals</b>	16	18	34

**Statistical Calculations on Acute Myocardial Infarction Subset (N = 34)**

<b>McNemar's <math>\chi^2</math></b>	$\chi^2_m = (b - c)^2 / (b + c) = (4 - 0)^2 / (4 + 0) = 4.0$ ( $df = 1, P < .05$ )	<b>Odds Ratio</b>	$b/c = 4/0.5^* = 8.0$ ( $SE = 4.0$ ; 90% CI, 1.42 to 14.58)
		<b>% Improvement Fraction</b>	$(b - c) / (b + d) = (4 - 0) / (4 + 14) = 22.2\%$

\*Substituting 0.5 for the 0 cell.  
†+STE, positive for ST-segment elevation; -STE, negative for ST-segment elevation; +AMI, confirmed AMI; -AMI, negative AMI.

Table 4.

Comparison of thrombolytic therapy eligibility on 12 versus 15 leads in myocardial infarction subgroup using McNemar's  $\chi^2$

Data Array 12 Leads			Actual Data 12 Leads			
15 Leads	+TRX*	-TRX	15 Leads	+TRX	-TRX	Total
+TRX†	CELL a	CELL b	+TRX	12	3	15
-TRX	CELL c	CELL d	-TRX	0	19	19
			<b>Totals</b>	12	22	34

**Statistical Calculations on Acute Myocardial Infarction Subset (N = 34)**

<b>McNemar's <math>\chi^2</math></b>	$\chi^2_m = (b - c)^2 / (b + c) = (3 - 0)^2 / (3 + 0) = 3.0$ ( $df = 1, P < .10$ )	<b>Odds Ratio</b>	$b/c = 3/0.5^* = 6.0$ ( $SE = 3.45$ ; 90% CI, 0.34 to 11.66)
		<b>% Improvement Fraction</b>	$(b - c) / (b + d) = (3 - 0) / (3 + 19) = 13.0\%$

\*Substituting 0.5 for the 0 cell.  
†+TRX, ECG-eligible for thrombolytic therapy; -TRX, not ECG-eligible for thrombolytic therapy; +AMI, confirmed AMI; -AMI, negative AMI.

during the eight-month study period, there was no difference in the discharge diagnosis infarct rate between those studied (31 of 109, 28.4%) and those not studied (62 of 209, 29.7%). This suggests no recruitment bias toward patients with acute myocardial infarction.

The abnormal ECG findings of either ST-segment displacement, primary T-wave inversion, or pathologic Q waves on the three study leads were detected in 28.9% of patients (43 of 149). ST-segment elevation was the most frequently detected abnormal finding (Figure 1). ST-segment elevation occurred solely on three extra leads in four of 34 infarct patients; all four patients had myocardial infarction confirmed by MB/CK enzymes. No patients without infarction had ST-segment elevation on posterior leads (zero of 115). In patients with myocardial infarction and inferior injury pattern, posterior ST elevation occurred in 50% (six of 12) of patients; 25% (three of 12) of patients with inferior injury had right ventricular injury (nine of 12 with either finding). Of the subset of 71 patients having no ST-segment displacement, Q waves, or T-wave inversion on 12 leads, none (zero of 71) had these findings on 15 leads.

The results of ST elevation against acute myocardial infarction for the 15- and 12-lead ECG are displayed (Table 1). The 15-lead ECG had a sensitivity of 58.8% (95% confidence interval [CI], 42.3 to 75.3) compared with 47.1% (95% CI, 30.3 to 63.9) for the 12-lead ECG; both had a specificity of 93% (95% CI, 88.3 to 97.7). The predictive value of a positive and a negative test and the accuracy were increased. Analysis of the likelihood ratio shows that use of the expanded ECG would lead to a small increase (5%) in a clinician's ability to conclude that a patient is probably having a myocardial infarction (likelihood ratio 6.73 versus 8.40; see Table 1). This increase is caused by the greater true-positive rate for myocardial infarction with ST-segment elevation on the 15-lead ECG.

In the myocardial infarction subgroup, there was a significant increase in the association of the 15-lead (versus 12-lead) ECG with ST-segment elevation (McNemar's  $\chi^2 = 4.0$ ,  $df = 1$ ,  $P < .05$ ; Table 3). There was an eightfold increase in the odds of detecting ST elevation on 15-lead (versus 12-lead) ECG (SE, 4.0; 90% CI, 1.42 to 14.58). Of myocardial infarction patients negative for ST-segment elevation on 12 leads, 22.2% were positive on 15 leads (percentage improvement fraction, Table 3).

The results of "unique eligibility for thrombolytic therapy" against acute myocardial infarction discharge diagnosis for the 15- and 12-lead ECGs are shown (Table 2). The findings indicate increased sensitivity, positive and negative predictive values, and accuracy;

specificity was unchanged. The pair-matched analysis (Table 4) showed an association of the 15-lead ECG with unique ECG thrombolytic criteria that approached statistical significance ( $P < .10$ ). There was a sixfold odds of meeting ECG criteria for thrombolytic therapy on 15- rather than 12-lead ECG (SE, 3.45; 90% CI, 0.34 to 11.66). For myocardial infarction patients, 13.5% who are not ECG-eligible for thrombolysis on 12-lead ECG are so on the 15 leads (Table 4 and Figure 2).

## DISCUSSION

Our study shows that the addition of leads  $V_{4R}$ ,  $V_8$ , and  $V_9$  to the standard 12 leads produces information about injury, necrosis, and ischemia (Figure 1). Our analysis focused on ST-segment elevation, for it is the most specific marker of acute infarction and is often a criterion for use of thrombolytic therapy.

For ST-segment elevation in the diagnosis of myocardial infarction, there was an increase of 11.7% in sensitivity and a small increase in positive predictive value with no loss of specificity (no increase in false-positives). Although 95% confidence intervals for sensitivity overlap, the more powerful pair-matched analysis (McNemar's) showed a significant increase in the proportion of myocardial infarctions with ST elevation on 15 versus 12 leads. This suggests that similar associations would be found in other acute myocardial infarction populations. The findings of ST elevation by use of these extra leads can strengthen the ED diagnosis of acute myocardial infarction on the initial tracing and may provide an indication for thrombolytic treatment.

Leads  $V_8$ ,  $V_9$ , and  $V_{4R}$  also may provide a better ECG characterization of inferior infarcts than is possible on the standard 12-lead ECG. Associations of posterior with inferior injury patterns may provide information about the extent of myocardial infarction. For example, the summation of ST-segment elevation is correlated with myocardial necrosis<sup>16</sup> and infarct salvage.<sup>17</sup> In inferior myocardial infarction, ST-segment elevation in  $V_8$  and  $V_9$  may imply a worse prognosis than without such changes. By quantitating the sum of ST-segment elevation accurately, which is known to correlate with final infarct size,<sup>16</sup> the extra leads may provide an improved severity classification of infarctions and help refine the process of risk benefit assessment for the administration of thrombolytic and invasive therapies. Also, right ventricular infarction as predicted by ST elevation in  $V_{4R}$  may be a valid predictor of nitrate-induced hypotension.<sup>18</sup>

Findings other than ST elevation in the three additional leads, like such findings on 12-lead tracings, were of limited diagnostic value. No patient showed an abnormality in the three leads when the 12-lead ECG was normal or with minimal abnormalities. If additional data bear out this finding, posterior and right ventricular leads could be applied selectively (eg, in the presence of ST-segment depression in leads  $V_1$  to  $V_4$  or ST-segment elevation in  $V_6$ ). This finding suggests, however, that additional leads will not solve the problem of patients presenting with myocardial infarction and a normal ECG.

On 12-lead ECGs, posterior myocardial infarction is known to be difficult to diagnose, and its true incidence for this reason is unknown.<sup>9</sup> A study by Cabin and Roberts<sup>19</sup> showed that a group with unrecognized infarction had a significantly higher prevalence of posterior infarcts (compared with other locations) than a control group with recognized infarcts. This is probably due to the late depolarization of the posterior wall, the subtleties of identifying prolonged R waves in  $V_1$  (more than 0.04 seconds), and the nonspecificity of R more than S and upright T waves in  $V_1$ .<sup>20</sup> Also, recent research indicates that epicardial occlusion of the circumflex vessel often may be "silent" on standard 12 leads.<sup>21</sup>

An additional limitation to the 12-lead approach is the impossibility of distinguishing anterior "subendocardial" from posterior "transmural" myocardial infarction.<sup>20</sup> Also, in the setting of inferior myocardial infarction, concomitant right ventricular infarction obscures the vectorcardiographic findings of posterior myocardial infarction<sup>22</sup> and may obscure the ECG findings of reciprocal changes as well.

Our findings are comparable with what is reported in the sparse literature on this subject. Melendez et al<sup>23</sup> reported posterior lead findings in 117 patients admitted to a monitored unit for suspected infarction. Three of 46 (6.5%) confirmed acute myocardial infarction patients demonstrated ST-segment elevation solely on the posterior leads.

Toyama et al<sup>24</sup> looked at posterior leads  $V_7$  to  $V_9$  in the context of body surface isopotential mapping. When body surface isopotential mapping was used as diagnostic of posterior wall myocardial infarction in the retrospective cohort, the addition of posterior leads increased the proportion of diagnostic ECGs from five of 20 to 11 of 20.<sup>24</sup> Ikeda et al<sup>25</sup> showed that  $V_1$  (R/S of more than 1) is afflicted with a high false-negative rate in detection of old posterior infarction.

Rich et al<sup>8</sup> found that  $V_9$  alone was superior to standard 12-lead ECG for the detection of posterior myocardial infarction. Perloff<sup>9</sup> found the sensitivity of posterior leads

for posterior myocardial infarction (defined by vectorcardiogram criteria) in ten patients to be 90%. However, anterior ST depression (a common criterion for posterior reciprocal injury pattern) was only 46% predictive of posterior wall myocardial infarction in context of the "diltiazem in reinfarction study."<sup>20</sup> These studies suggest that leads  $V_8$  and  $V_9$  are superior in the diagnosis of posterior myocardial infarction to the reciprocal findings in leads  $V_1$  to  $V_3$ .

Our study is limited by several factors. Our sample of hospital admissions was not consecutive. Thus, it is possible that bias entered into the selection, and it either over- or under-represents the true proportion of patients with findings in posterior leads at this hospital. These results should be interpreted with caution because of the small sample size. Replication of this study with a large sample would help to confirm these results.

The estimates of sensitivity and specificity were based on the sample of patients who were admitted to the hospital with suspected acute ischemic heart disease. Patients discharged from the ED or admitted with nonischemic diagnoses were not evaluated for acute myocardial infarction. Therefore, the calculated diagnostic parameters do not apply to the larger population of patients who present with chest pain, dyspnea, diaphoresis, or weakness and who are either discharged or admitted without a diagnosis of acute ischemic heart disease. Inclusion of discharged patients may alter absolute determinations of sensitivity and specificity but probably would not affect the relative improvement in 12 versus 15 leads.

Posterior leads frequently have low-amplitude QRS complexes (less than 10 mm), and ST-segment elevation can be subtle and require careful interpretation. Follow-up 15-lead ECGs, coronary angiography, and ventricular function studies were not performed systematically to verify right or posterior left infarction in patients having ST-segment elevation in those regions. Thus, assessing the full clinical value of the expanded ECG will require such investigations; they currently are being addressed by a multicenter follow-up study.

## CONCLUSION

The 15-lead ECG provides increased sensitivity and odds of detecting injury pattern with no loss of specificity. It may lead to improved selection of thrombolytic therapy candidates and provide a fuller description of the extent of myocardial injury and necrosis.

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