

Right ventricular infarction: diagnostic accuracy of electrocardiographic right chest leads V3R to V7R investigated prospectively in 43 consecutive fatal cases from a coronary care unit

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SUMMARY The accuracy of ST elevation ≥ 1 mm in right chest leads V3R to V7R in the diagnosis of right ventricular infarction was investigated in a clinical and necropsy study of 43 consecutive patients who died in a coronary care unit. Thirty six patients had left ventricular myocardial infarction and in 27 the right ventricle was also affected. Seven patients had normal hearts. The specificity and positive predictive value of ST elevation in V3R were 81% and 77%, respectively. These increased to 100% when combined with ST elevation in one or more leads V4R-V7R. The diagnostic accuracy was poor for anterior infarcts (sensitivity $\leq 27\%$), but high for inferior/posterior infarcts (sensitivity $\geq 64\%$) in which the specificity and positive predictive value reached 100% in V6R and V7R.

Inferior/posterior infarction affecting the right ventricle can be diagnosed reliably by examination of electrocardiograms from right chest leads V6R and V7R.

The first pathological report on right ventricular infarction was published in 1930 by Sanders.¹ In 1974, Erhardt suggested that ST segment elevation in right chest lead CR4R was a good indicator of extensive right ventricular infarction.² Since then, many studies have evaluated the diagnostic value of various right chest leads in right ventricular infarction. Most electrocardiographic studies have examined two leads only (V3R and V4R) and conflicting results have been reported.³⁻⁸ A few studies have focused on more lateral right chest leads⁹⁻¹³ and in two of these studies the lateral right chest leads V5R and V6R were claimed to be most accurate.^{9,11} Right ventricular infarct size and location, however, were not confirmed at necropsy in these two studies.

The aim of this prospective study was to evaluate the diagnostic value of right chest leads V3R to V7R in right ventricular infarction in consecutive patients from a coronary care unit whose hearts were examined at necropsy.

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Patients and methods

STUDY PATIENTS

From November 1984 to January 1986, consecutive patients with clinically suspected ischaemic heart disease were prospectively included in a study of right ventricular infarction. The hearts of patients who died in hospital were examined for evidence of right ventricular infarction.¹⁴

Patients who died were included in the present study if (a) there was no previous myocardial infarction (recognised clinically or electrocardiographically); (b) an electrocardiogram was recorded within 12 hours of the onset of symptoms, and (c) the electrocardiogram did not show bundle branch block. Forty three patients fulfilled these criteria (15 women and 28 men; median age 67 years, range 44-84 years). Thirty six patients died from acute myocardial infarction and seven from non-cardiac disease.

ANATOMICAL INVESTIGATION

The necropsy technique has been described in detail elsewhere.¹⁴ In brief, we performed postmortem coronary arteriography followed by cross sectioning

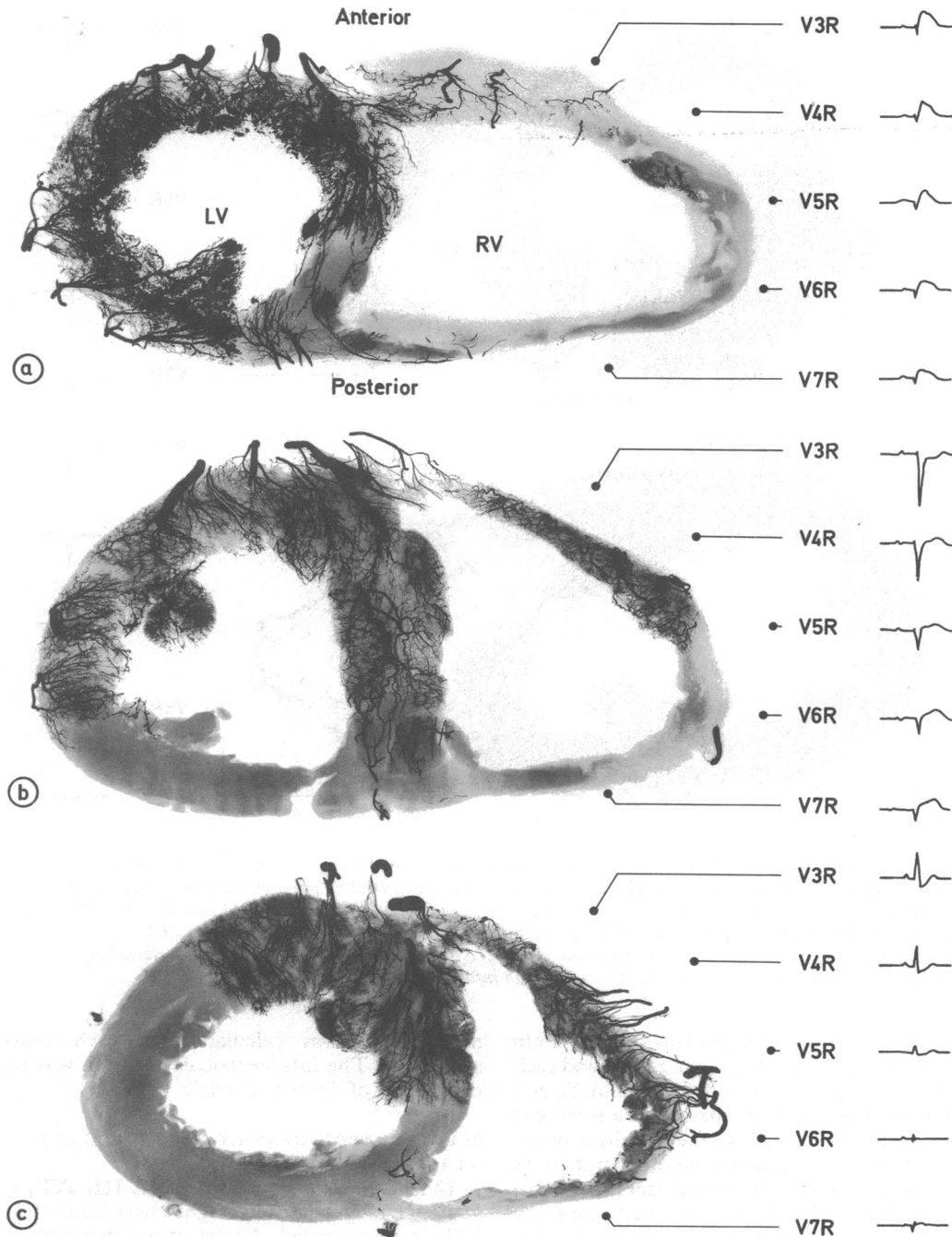


Fig 1 Inferior/posterior infarction. Right chest electrocardiogram (V3R to V7R) with corresponding necropsy angiograms of 1 cm thick transventricular slices showing poor contrast filling in infarcted myocardium. (a and b) Posterior infarction caused by occluded right coronary artery with extensive right ventricular (RV) infarction and concomitant minor (a) and larger (b) left ventricular (LV) infarction. (c) Posterolateral infarct caused by occluded circumflex coronary artery with extensive left ventricular infarction and minor right ventricular infarction. Corresponding electrocardiograms show ST elevation in five leads (a), three leads (b), and one lead (c).

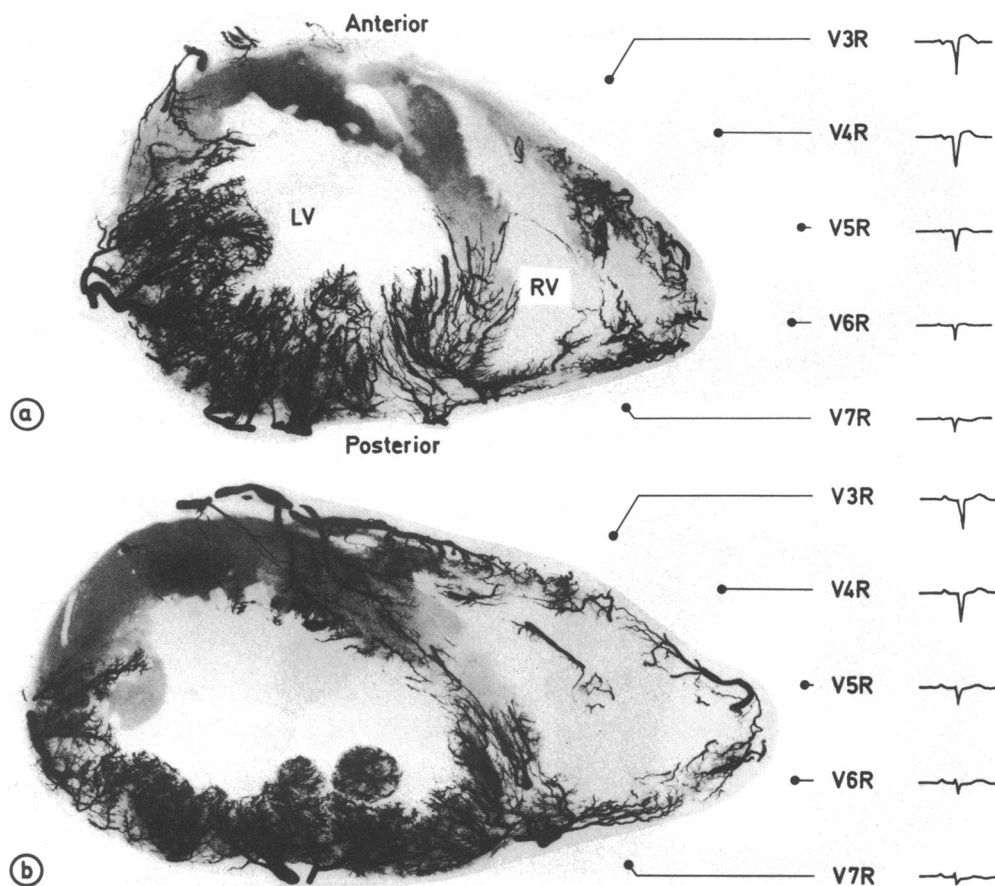


Fig 2 Anterior infarction. Right chest electrocardiogram (V3R to V7R) with corresponding necropsy angiograms of 1 cm thick transventricular slices showing poor contrast filling in infarcted myocardium after an occlusion of the left anterior descending coronary artery. (a) Right ventricular (RV) involvement with electrocardiographic Q wave in V3R and ST elevation in V3R to V5R. (b) Left ventricular (LV) infarction without right ventricular involvement. There is a Q wave in V3R but there is no ST elevation in V3R to V7R. Much of the septum was affected by both infarcts.

of the arteries. The myocardium was cut into 1 cm slices parallel to the atrioventricular groove and each slice was photographed, x rayed (figs 1 and 2), and photographed again after incubation in a nitroblue tetrazolium solution to improve the definition of the infarct. New and old infarcts were identified as regional myocardial necrosis or regional myocardial scar tissue (fibrosis) distal to an appropriate coronary artery lesion,¹⁵ and were classified as anterior, lateral, or posterior.¹⁶ The right septal wall was taken on the boundary between the right and the left ventricle.¹⁴ Computer assisted planimetry was performed on colour prints of each myocardial slice, and the total weight of ventricular myocardium and infarcted

myocardium was calculated for each ventricle separately. The interventricular septum was regarded as part of the left ventricle.

ELECTROCARDIOGRAPHIC RECORDING AND MEASUREMENTS

A 12 lead electrocardiogram (I, II, III, aVR, aVL, aVF, V1 to V6) and five right chest leads (V3R to V7R) were recorded. The electrocardiograph was a three channel ink-jet Siemens Elema Mingograph 34 galvanometer recorder (Siemens Elema AB). The paper speed was 25 mm/s and manual calibration (1 mV = 10 mm) was adjusted before the recordings. Measurements were obtained according to the

Minnesota code criteria.¹⁷ The ST segment was measured 40 ms after the last nadir in the QRS complex in leads II, III, aVF, and V3R–V7R. Three consecutive QRS complexes were evaluated with the PQ level as the isoelectrical line. Elevation of the ST segment was estimated to the nearest ¼ mm by careful visual inspection. The mean value for three consecutive ST segments was calculated. Abnormal ST elevation was defined as a deviation >0.6 mm in V3R, >0.5 mm in V4R to V6R, and >0.4 mm in V7R.¹⁸ Because it is difficult for the human eye to detect ST elevation of 0.4 to 0.6 mm with precision, we also evaluated ST elevation ≥1 mm, which has been reported to be a useful clinical marker of right ventricular infarction.^{3,5-7,13}

The electrocardiographic site of the infarct was classified as anterior or posterior if characteristic changes were seen in at least two leads in the 12 lead electrocardiogram.¹⁹ Changes in V1–V3 (anteroseptal), V1–V6 (extensive anterior), or V4–V6, I, aVL, and possibly II (anterolateral) were all classified as anterior. Changes in II, III, aVF (inferior), inverse changes in V1–V3 (true posterior), and II, III, aVF, V5, V6, and sometimes also I and aVL (inferolateral) were classified as posterior. If no such changes were seen the infarct location was unclassified.

INFARCT GROUPS

Patients were grouped according to the presence or absence of anatomical right ventricular infarction. Furthermore, infarct locations determined by anatomical and electrocardiographic criteria were considered separately, particularly anterior and posterior locations.

STATISTICAL ANALYSIS

Pathological, anatomical, and electrocardiographic data are presented as median and range. We used the Mann-Whitney U test to compare median values in two groups and the Kruskal-Wallis test to compare median values in more than two groups. We used Fisher's exact test to compare frequencies. Coefficients of correlation (Spearman rho) were calcu-

Table 1 Anatomical and electrocardiographic (ECG) location of 36 acute myocardial infarcts in 36 patients

ECG infarct location	Anatomical infarct location		
	Anterior 18 (15)	Posterior 12 (11)	Lateral 6 (1)
Anterior 17	16 (14)	1 (1)	0
Posterior 16	1 (0)	11 (10)	4 (1)
Unspecified 3	1 (1)	0	2 (0)

Figures in parentheses are infarcts with anatomical right ventricular involvement.

lated. The level of significance was $p < 0.05$, and two sided tests were performed. The diagnostic accuracy of ST elevation and Q wave was calculated.²⁰

Results

ANATOMICAL FINDINGS

Seven patients died from non-cardiac disease and had normal hearts without appreciable disease of the coronary arteries, valves, or myocardium. Three of these patients died from pneumonia; arrhythmia, pulmonary embolism, perforation of a peptic ulcer, and unspecified haematological disease was the cause of death in the remaining four patients. Thirty six patients died from acute myocardial infarction, and necropsy showed old infarcts in 17 although they had had no clinical or electrocardiographic evidence of previous infarction. Thirteen patients died from myocardial rupture, 12 from cardiogenic shock, seven from arrhythmia, two from ischaemic brain damage after cardiac arrest, one from acute bronchial asthma, and one from heart failure (within days).

Table 1 lists the anatomical site and corresponding electrocardiographic location of the 36 acute myocardial infarcts and whether or not the right ventricle was affected. In 27 cases (75% of infarcts) it was—about half were anterior infarctions and half were posterior infarctions, and only one was a left lateral infarction.

Table 2 shows the amount of infarcted myocardium in the left and right ventricle in anterior,

Table 2 Size, site, and distribution of acute myocardial infarction in 36 patients

	Anatomical infarct location			<i>p</i> value
	Anterior <i>n</i> = 18	Posterior <i>n</i> = 12	Lateral <i>n</i> = 6	
Total infarct size (gm)	40 (4–91)	40 (11–99)	38 (12–113)	NS*
Left ventricular infarct size (gm)	39 (4–84)	19 (3–72)	38 (8–103)	<0.05*
Right ventricular infarct size (gm)	2 (0.1–7)	15 (5–32)	(<i>n</i> = 1)	<0.05**
Percentage of left ventricle infarcted	28 (4–84)	16 (2–33)	29 (6–51)	<0.05*
Percentage of right ventricle infarcted	5 (1–21)	53 (25–72)	—	<0.05**
Percentage of septum infarcted	48 (8–87)	11 (0–32)	—	<0.05**

*Posterior infarction compared with anterior or lateral infarction (Kruskal-Wallis test).

**Posterior infarction compared with anterior infarction but not with lateral infarction (Mann-Whitney test).
Septum included in the left ventricle.

Table 3 Diagnostic accuracy (%) of right chest leads in 43 patients with or without right ventricular infarction by necropsy

	Sensitivity	Specificity	Positive predictive value	Negative predictive value	Efficiency
ST elevation ≥ 1 mm in leads:					
V3R	37 (44)	81 (81)	77 (80)	43 (46)	53 (58)
V4R	33 (41)	86 (76)	82 (85)	44 (47)	53 (58)
V5R	26 (44)	94 (81)	88 (80)	43 (46)	51 (58)
V6R	33 (41)	100 (88)	100 (85)	47 (47)	58 (58)
V7R	41 (44)	100 (81)	100 (80)	50 (50)	63 (58)
Q wave in V3R	37	87	83	45	56

Numbers in parentheses are percentages with abnormal ST elevation (V3R > 0.6 mm, V4R to V6R > 0.5 mm, V7R > 0.4 mm^{1b}).

posterior, and lateral infarcts. Total infarct size was not significantly different in these sites, but there was quite a different pattern of infarct distribution between the ventricles in anterior and posterior infarcts. The right ventricular infarct component was much larger in posterior infarction than in anterior infarction. Lateral infarcts could not be considered further because there was only one with right ventricular involvement.

ELECTROCARDIOGRAPHIC FINDINGS

The 12 lead electrocardiogram and the right chest leads were normal in the seven patients without heart disease. Tables 3 and 4 show the sensitivity, specificity, positive and negative predictive value, and efficiency for ST elevation and Q wave.

ST ELEVATION AND Q WAVE

The diagnostic accuracy of ST elevation of ≥ 1 mm in right chest leads was higher for lateral than for medial leads and reached 100% specificity and positive predictive value in leads V6R and V7R (table 3). The diagnostic accuracy of ST elevation varied with different infarct sites. The sensitivity was < 28% in anatomical anterior infarction, but > 63% in posterior infarcts. The same differences were found for electrocardiographic anterior and posterior infarction. The sensitivity was > 63% in electrocardiographic posterior infarction where specificity and positive predictive value reached 100% in V5R, V6R, and V7R (table 4). The positive predictive

value of ST elevation in V6R and V7R fell from 100% to 80–85% when abnormal ST elevation rather than ≥ 1 mm was used as the discriminator (table 3).

The positive predictive value of a Q wave in lead V3R was 83% for all infarcts (table 3). The positive predictive value was 86% for anatomical anterior infarcts, but 100% for anatomical posterior infarcts. Use of a combination of Q wave in V3R and ST elevation in leads V5R–V7R did not improve the diagnostic accuracy.

NUMBER OF LEADS WITH ST ELEVATION

There was a positive correlation between the number (1–5) of right chest leads with ST elevation ≥ 1 mm and the size (g) of right ventricular infarction ($\rho = 0.41$, $p < 0.05$). An inverse and non-significant correlation was found between the number of leads with ST elevation ≥ 1 mm and the size of left ventricular infarction ($\rho = -0.23$, NS). ST elevation ≥ 1 mm in all five right chest leads (V3R–V7R) was always associated with extensive right ventricular infarction (median 62%, range 36–72% of the right ventricle infarcted) with concomitant small left ventricular infarction (median 14%, range 7–16%) of the left ventricle infarcted (fig 1a). ST elevation of ≥ 1 mm in both of the two medial leads (V3R and V4R) was found in 10 patients and was associated with right ventricular infarction in eight. The other two patients had left (anterior) ventricular infarction without right ventricular involvement (fig 2b). ST

Table 4 Diagnostic accuracy (%) of right chest leads in 16 patients with electrocardiographic posterior infarction. Infarct location was examined at necropsy

	Sensitivity	Specificity	Positive predictive value	Negative predictive value	Efficiency
ST elevation ≥ 1 mm in leads:					
V3R	37 (44)	81 (81)	77 (80)	43 (46)	53 (58)
V4R	33 (41)	86 (76)	82 (85)	44 (47)	53 (58)
V5R	26 (44)	94 (81)	88 (80)	43 (46)	51 (58)
V6R	33 (41)	100 (88)	100 (85)	47 (47)	58 (58)
V7R	41 (44)	100 (81)	100 (80)	50 (50)	63 (58)
Q wave in V3R	37	87	83	45	56

Numbers in parentheses are percentages with abnormal ST elevation (V3R > 0.6 mm, V4R to V6R > 0.5 mm, V7R > 0.4 mm^{1b}).

elevation of ≥ 1 mm in the three lateral leads (V5R–V7R) was always associated with posterior infarction with right ventricular involvement. Furthermore, ST elevation of ≥ 1 mm in all five right chest leads was always associated with occlusion of the right coronary artery proximal to the marginal (acute) branch.

Discussion

ANATOMICAL FINDINGS

Anterior right ventricular infarcts were always small and mainly located in the apical third of the heart far from the right chest leads. Posterior right ventricular infarcts were often large and located near the atrioventricular groove, closer to the right chest leads. Therefore we expected that electrocardiographic infarct signals from the right ventricle would be stronger and easier to detect in posterior infarction than in anterior infarction.

DIAGNOSTIC ACCURACY OF ST ELEVATION

ST elevation of ≥ 0.5 mm in right chest leads has been reported to be diagnostic for right ventricular infarction.^{5–7} Recently, normal values for ST elevation were published; these indicated that ST elevation of 0.4–0.6 mm in various right chest leads should be accepted as normal.¹⁸ Therefore, to secure interpretation of a valid abnormal signal in practice, we recommend ST ≥ 1 mm as the discriminatory level. Using this criterion, we found that positive predictive value and specificity were 100% in leads V6R and V7R. This is even higher than the results obtained by Funk who evaluated ST elevation of ≥ 1 mm in V6R.⁹

DIAGNOSTIC ACCURACY OF Q WAVE

Loss of the rightwardly directed electrical vector generated from activation of the right ventricle and the septum¹⁹ may be responsible for the abnormal Q wave in V3R. So a Q wave in V3R may reflect septal necrosis as well as right ventricular necrosis. Therefore in anterior infarction extensive septal necrosis (loss of a rightwardly directed vector) with small right ventricular involvement (fig 2a) may be responsible for the smaller positive predictive value for this infarct site. In posterior infarction (fig 1), a Q wave in V3R was more likely to reflect right ventricular necrosis.

NUMBER OF LEADS

Erhardt *et al* used only one right chest lead (CR4R).²⁷ Since then, most studies were limited to two leads (V3R and V4R) and very few studies investigated multiple right chest leads.^{9–13} Braat *et al* found the best diagnostic result with lead V4R¹⁰ whereas Croft

et al found that a combination of V4R, V5R, and V6R was the most reliable.¹¹ Funk, however, found the highest diagnostic sensitivity and specificity for V6R; diagnostic efficiency was not improved by combining V6R with changes in V4R and V5R.⁹ The present study confirms the observations of Funk⁹ and Carson¹³ that diagnostic accuracy is greatest for lateral right chest leads, and is not significantly improved by combining these leads with the more medial leads.

The number of right chest leads showing ST elevation ≥ 1 mm decreased as the size of left ventricular infarction increased. Patients with extensive right ventricular infarction and large left ventricular infarction may be without ST elevation in V3R and V4R while leads V5R to V7R show ST elevation ≥ 1 mm (fig 1b). This may be caused by the abolition of ST elevation in leads V3R and V4R by the large left ventricular infarction. Furthermore, large infarcts of the left ventricular lateral wall with minor right ventricular involvement may cause ST depression in most of the right chest leads (fig 1c).

CLINICAL IMPLICATIONS

The electrocardiographic diagnosis of acute myocardial infarction is normally based upon changes in the 12 lead electrocardiogram. The right chest leads do not add much to the diagnostic accuracy of the 12 lead electrocardiogram. Therefore, the risk of missing a diagnosis of acute myocardial infarction if the right chest leads are not examined is very small. But the right chest leads are useful for diagnosing the distribution of infarct between the right and the left ventricle. In patients with electrocardiographic inferior/posterior infarction, the right chest leads can identify those without ST elevation in V3R–V7R (predominantly left ventricular infarction), who have a similar prognosis to patients with anterior infarction, and those with ST elevation in V3R–V7R (extensive infarction of the right ventricle and minor infarction of the left ventricle) who have a much better prognosis.²¹ None the less, cardiogenic shock can develop in such patients despite only slight impairment of the left ventricle. Early recognition of this infarct distribution by electrocardiography may be needed to ensure that these patients are treated appropriately.²²

We conclude that involvement of the right ventricle during inferior/posterior infarction can be diagnosed with high accuracy by right chest leads V6R and V7R.

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