

Value of electrocardiogram in diagnosing right ventricular involvement in patients with an acute inferior wall myocardial infarction

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SUMMARY To study the value of the electrocardiogram in diagnosing right ventricular involvement in acute inferior wall myocardial infarction, the electrocardiographic findings were analysed in 67 patients who had had scintigraphy to pin-point the infarct. All 67 patients were consecutively admitted because of an acute inferior wall infarction. A 12 lead electrocardiogram with four additional right precordial leads (V3R, V4R, V5R, and V6R) was routinely recorded on admission and every eight hours thereafter for three consecutive days.

Thirty-six to 72 hours after the onset of chest pain a 99m technetium pyrophosphate scintigraphy and a dynamic flow study were performed to detect right ventricular involvement, which was found in 29 of the 67 patients (43%). ST segment elevation ≥ 1 mm in leads V3R, V4R, V5R, and V6R is a reliable sign of right ventricular involvement. ST segment elevation ≥ 1 mm in lead V4R was found to have the greatest sensitivity (93%) and predictive accuracy (93%). The diagnostic value of a QS pattern in lead V3R and V4R or ST elevation ≥ 1 mm in lead V1 was much lower. ST segment elevation in the right precordial leads was short lived, having disappeared within 10 hours after the onset of chest pain in half of our patients with right ventricular involvement. When electrocardiograms are recorded in patients with an acute inferior wall infarction within 10 hours after the onset of chest pain, additional right ventricular infarction can easily be diagnosed by recording lead V4R.

Until recently, the diagnosis of right ventricular infarction was only possible at necropsy. In 1948, in their review of 2000 consecutive necropsies, Wartman and Hellerstein¹ described 22 instances of right ventricular infarction out of 164 cases of myocardial infarction. At necropsy on 19 hearts with right ventricular infarction, Wade² found that the major damage was on the posterior wall of the heart. In all 19 patients the electrocardiogram had shown an inferior wall myocardial infarction.

In 1974 Cohn *et al.*³ reported characteristic haemodynamic changes in six patients with an inferior wall myocardial infarction, who also had right ventricular infarction. Sharpe *et al.*⁴ observed that six out of 15 patients with an inferior wall infarction showed abnormal technetium pyrophosphate uptake in the right ventricle. In a study of patients with an acute

inferior wall infarction Wackers *et al.*⁵ found that in 37% the technetium pyrophosphate uptake also showed involvement of the right ventricle. Erhardt *et al.*⁶ in 1976 described the value of a right precordial lead V4R in diagnosing right ventricular involvement. They compared their data with necropsy findings and found right ventricular infarction in nine out of 18 patients (50%). These data have subsequently been confirmed by other investigators.⁷ Recently, Chou *et al.*⁸ described the value of lead V1 in diagnosing right ventricular infarction in 11 patients. The diagnosis of right ventricular infarction was proven at necropsy and supported by haemodynamic findings. Morgera *et al.*⁹ in a preliminary report described the value of a QS pattern in lead V3R and V4R in diagnosing right ventricular infarction.

The incidence of abnormal haemodynamic findings suggesting right ventricular infarction is much lower than findings pointing to right ventricular involve-

ment at necropsy or scintigraphy. Therefore we decided to compare the value of the different electrocardiographic criteria in diagnosing right ventricular infarction in patients with inferior wall infarction when additional right ventricular involvement was shown by ^{99m}Tc pyrophosphate scintigraphy.

Patients and methods

Studies were made on 67 consecutive patients (56 men, 11 women), admitted because of an acute inferior wall infarction. Four had a documented myocardial infarction in the past, in two on the anterior and in two on the inferior wall. Patients were admitted half an hour to 30 (mean five) hours after the onset of chest pain. Ages ranged from 39 to 80 (mean 57 ± 9.4) years. The diagnosis of acute inferior wall infarction was based on the clinical history, a characteristic enzyme pattern of CK and AST values, and the appearance of new pathological Q waves in the inferior leads (II, III, and aVF). On admission and every eight hours during the next three days a 12 lead electrocardiogram and four additional right precordial

leads were recorded (Fig. 1). The amount of ST elevation in lead V1, and in leads V3R, V4R, V5R, and V6R (these leads are the mirror image of leads V3, V4, V5, and V6), was measured and the duration of its presence noted. We also looked for the presence of a QS pattern in leads V3R and V4R.

At the time of the electrocardiographic recordings blood was taken to determine the values for CK and AST. Normal values for CK and AST in our laboratory are, respectively, less than 240 and 40 U/l. None of the 67 patients had clinical signs of right ventricular infarction on physical examination. No haemodynamic monitoring was performed in any of the patients. Thirty-six to 72 hours after the onset of chest pain ^{99m}Tc pyrophosphate scintigraphy was performed. A Philips scintillation camera with a general all purpose parallel hole collimator interfaced to a PDS computer system or a Ohio Nuclear Sigma 420 mobile gamma camera with a general all purpose parallel hole collimator interfaced to a MCS 560 mobile computer system was used for all studies. One hour after the injection of 15 to 20 mCi ^{99m}Tc pyrophosphate, the anterior, the left lateral, and the

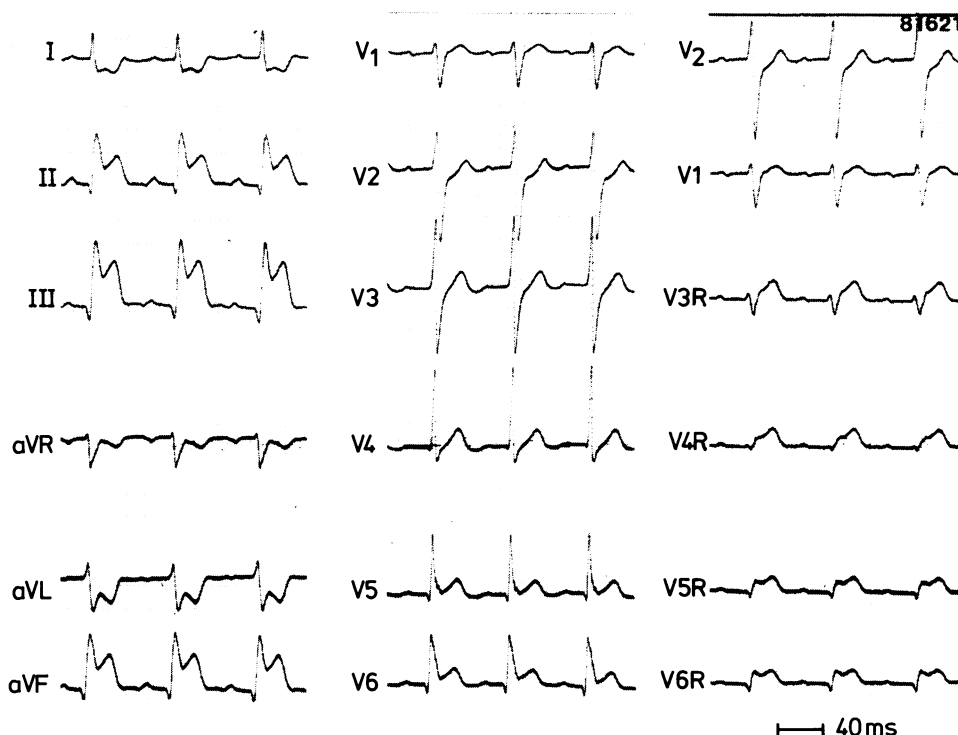


Fig. 1 Leads I, II, III, aVR, aVL, aVF, leads V1, V2, V3, V4, V5, V6, and leads V2R, V1R, V3R, V4R, V5R, and V6R are recorded simultaneously. This electrocardiogram shows an acute inferoposterior wall myocardial infarction. The right precordial leads show ST segment elevation in leads V3R, V4R, V5R, and V6R, in the absence of a QS pattern in lead V3R or V4R or ST segment elevation in lead V1.

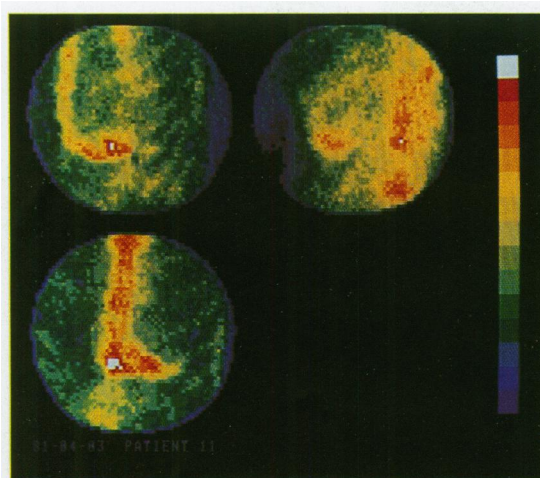


Fig 2

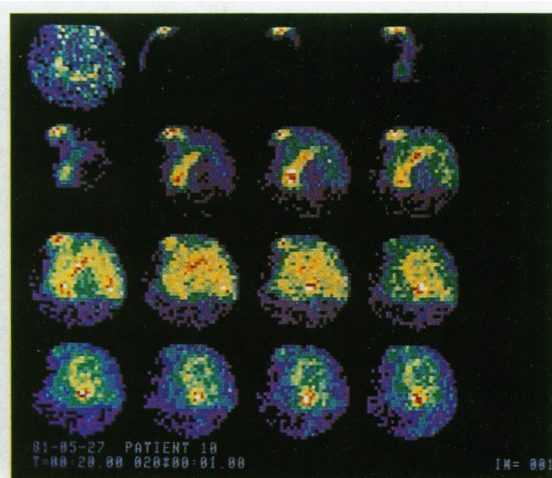


Fig 3

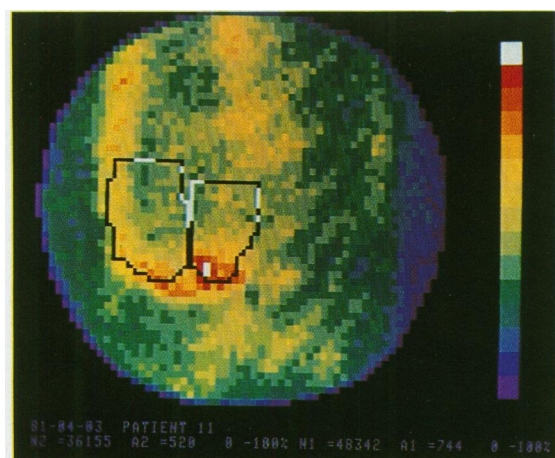


Fig 4

Fig. 2 An example of a ^{99m}Tc pyrophosphate scan in three different views. Top left the 45° left anterior oblique view. On the right, the left lateral, and below the anterior view. Apart from uptake in the sternum, spine, and ribs, pathological ^{99m}Tc pyrophosphate uptake is seen in the inferior wall of the myocardium.

Fig. 3 An example of a dynamic flow study. The activity can be followed from the superior caval vein to the right ventricle, the lungs, the left ventricle, and the aorta.

Fig. 4 The 45° left anterior oblique view on which is superimposed the region of interest drawn around the right and left ventricle. Definite involvement of the right ventricle is seen.

45° left anterior oblique views were recorded with a general all purpose parallel hole collimator (Fig. 2). Each view contained at least 600 000 counts. After the last view was recorded, which was always the 45° left anterior oblique, a small bolus of ^{99m}Tc was injected without moving the patient or the collimator. Simultaneously a dynamic flow study was performed, using frames of one second to visualise separately the right and left ventricle (Fig. 3). A region of interest was placed around the right and left ventricle and these regions of interest were superimposed on the left anterior oblique view to verify whether there was right ventricular involvement (Fig. 4).

The radionuclide data were analysed by two independent observers without knowledge of the clinical data. The ^{99m}Tc pyrophosphate scintigraphy was judged to be positive when there was myocardial

uptake. Right ventricular involvement was considered to be present if myocardial uptake was seen in the region of interest of the right ventricle.

Results

All 67 patients had pathological uptake of ^{99m}Tc pyrophosphate in the inferior wall. Twenty-nine patients (43%) also had right ventricular involvement. Twenty-one patients had ST segment elevation ≥ 1 mm in lead V3R. Only in one patient did the scan not show right ventricular involvement. Twenty-nine patients had ST segment elevation in lead V4R ≥ 1 mm. In two of this group there was no right ventricular involvement on the ^{99m}Tc scan. In only three of the 29 patients with ST segment elevation in lead V4R ≥ 1 mm did this elevation per-

Value of lead V4R

sist for more than 72 hours. In 14 patients the duration of significant ST elevation lasted less than 10 hours after the onset of chest pain. In the two patients without ST elevation in lead V4R but with a positive ^{99m}Tc scan, 15 to 30 hours, respectively, had elapsed before arrival in hospital. In addition, in 29 patients there was ST segment elevation in lead V5R. Three of them had no pathological $^{99m}\text{technetium}$ uptake in the right ventricle. Twenty-seven patients had ST elevation ≥ 1 mm in lead V6R. As shown in Table 1, in three cases there was disagreement between the electrocardiographic and the scintigraphic data.

Sixteen patients with a QS pattern in leads V3R and V4R had right ventricular involvement on the scan. Of the 29 patients with scintigraphic right ventricular infarction only eight had ST elevation in lead V1 ≥ 1 mm. A false positive QS pattern in lead V3R and V4R, or ST elevation in lead V1, was seen in five and three patients, respectively.

Table 2 shows the sensitivity, specificity, and predictive accuracy of ST elevation ≥ 1 mm in leads V1, V3R, V4R, V5R, V6R, and of a QS pattern in leads V3R and V4R.

The mean peak AST value of all the 67 patients was 255 ± 120 U/l. In the 29 patients with right ventricular infarction the mean AST was 271 ± 120 U/l and in the

38 patients without right ventricular infarction 243 ± 118 U/l. This difference has no statistical significance. The mean peak CK value of all the patients was 2262 U/l and here also there was no statistically significant difference between the patients with and without right ventricular infarction (respectively 2321 ± 1548 U/l and 2177 ± 1418 U/l).

Only one of the patients died in hospital, suddenly on the third day after admission, and cardiac tamponade was found at necropsy. This patient had a positive scintigraphy for right ventricular involvement and ST elevation > 1 mm in lead V3R, V4R, V5R, and V6R, but no QS pattern in lead V3R or V4R, nor ST elevation in V1. At necropsy apart from an inferoposteroseptal infarction right ventricular involvement was also found.

Discussion

Since Cohn *et al.*³ in 1974 reported on the value of recognising additional right ventricular infarction in patients with inferior wall infarction, interest in diagnosing this abnormality has grown. Since then it has become clear, however, that the clinical and haemodynamic features of right ventricular infarction are found in only a small percentage of patients showing right ventricular infarction at necropsy or during cardiac scintigraphy.¹⁰ In our series 43% of patients with an inferior wall myocardial infarction had right ventricular involvement. Maximal enzyme values and clinical course during admission were not different in patients with and without additional right ventricular infarction. Because of the high incidence of atrioventricular nodal conduction disturbances in patients with inferior wall myocardial infarction with associated right ventricular infarction,¹¹ we feel that it is desirable to have an easy and cheap tool to detect right ventricular involvement. Our results indicate that lead V4R is the single most valuable electrocardiographic lead to detect right ventricular involvement. It is of importance to stress, however, that the duration of ST segment elevation is short, disappearing in less than 10 hours in 48% of our patients with right ventricular infarction. This indicates the necessity for early recording of the right precordial leads. Our data indicate that ST elevation in lead V1 or a QS pattern in V3R and V4R does not have the same diagnostic value as ST segment elevation in leads V3R, V4R, V5R, and V6R. In addition, a combination of V4R and V1 or V4R and a QS pattern in V3R and V4R does not improve the diagnostic accuracy of the electrocardiogram in making the diagnosis of right ventricular infarction.

The most consistent finding in our patients with right ventricular infarction was the ST segment elevation in the right precordial leads. Necropsy data have

Table 1 Presence of ST segment elevation in leads V3R, V4R, V5R, and V6R in patients with acute inferior wall infarction with and without right ventricular involvement

	V3R	V4R	V5R	V6R
<i>ST elevation ≥ 1 mm</i>				
Positive pathological ^{99m}Tc uptake in right ventricle	20	27	26	24
Negative pathological ^{99m}Tc uptake in right ventricle	1	2	3	3
<i>No ST elevation > 1 mm</i>				
True negative	37	36	35	35
False negative	9	2	3	5

Table 2 Sensitivity, specificity, and predictive accuracy of ST segment elevation ≥ 1 mm in leads V1, V3R, V4R, V5R, and V6R and a QS complex in leads V3R or V4R in diagnosing right ventricular involvement in patients admitted because of an acute inferior myocardial infarction

ST segment elevation > 1 mm	Sensitivity	Specificity	Predictive accuracy
V1	28	92	73
V3R	69	97	95
V4R	93	95	93
V5R	90	92	90
V6R	83	92	89
<i>QS pattern</i>			
V3R	55	87	76
V4R	55	87	76

shown that right ventricular infarction is seen almost exclusively in patients with inferior wall infarction in combination with posteroseptal involvement. Perhaps the ST segment elevation in the right precordial leads is an expression of posteroseptal involvement. The finding of a QS pattern in V4R and V3R requires further study. Again this could be the result of the loss of septal forces from the infarcted posteroseptal area. The incidence of a QS pattern in right precordial leads in patients without myocardial infarction is, however, not known. From our data and those of others^{6,7,12} we conclude that the single recording of lead V4R in order to see an ST segment elevation equal to or more than 1 mm is the most reliable way to diagnose right ventricular involvement in patients admitted because of an acute inferior myocardial infarction.

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