

Electrocardiographic Findings in Acute Right Ventricular Infarction: Sensitivity and Specificity of Electrocardiographic Alterations in Right Precordial Leads V₄R, V₃R, V₁, V₂ and V₃

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To determine the sensitivity, specificity, predictive value and diagnostic efficiency of electrocardiographic alterations in the diagnosis of acute right ventricular infarction, 43 autopsy patients with acute myocardial infarction and an electrocardiogram including 12 leads plus leads V₃R and V₄R were studied. Group A included 21 patients with right ventricular infarction, of whom 14 (group AI) had posterior and 7 (group AII) had anterior right ventricular infarction. Group B included 22 patients without right ventricular infarction. Excluding group AII patients, the sensitivity of the presence of a Q wave reached 78.6% in lead V₄R and decreased in leads V₁ to V₃; its specificity was low in all the leads. The sensitivity of ST segment elevation reached 100% in lead V₄R and decreased in leads V₁ to V₃; its specificity was highest (68.2%) in leads V₄R and V₃R, its negative

predictive value was 100% and its diagnostic efficiency was 80.6%. The criterion of ST segment elevation in lead V₄R being higher than that in leads V₁ to V₃ was less sensitive (78.6%) than ST segment elevation in lead V₄R alone, but its specificity reached 100%, its positive predictive value 100% and its diagnostic efficiency 91.7%.

In conclusion, there are no electrocardiographic criteria to identify anterior right ventricular necrosis, but posterior right ventricular necrosis may be identified by the presence of a Q wave or ST segment elevation in the right precordial leads, reaching the highest sensitivity and specificity in lead V₄R. The criterion of ST segment elevation in lead V₄R being higher than that in leads V₁ to V₃ offers the highest specificity and efficiency in the diagnosis.

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Although electrocardiography is probably the most useful, simple and objective tool for the diagnosis of acute myocardial infarction, there are no well defined electrocardiographic criteria to properly identify right ventricular involvement in acute myocardial infarction. Several alterations, including the presence of a Q wave and ST segment elevation in some of the right precordial leads (CR₄R, V₄R, V₃R, V₁, V₂, V₃), have been described (1-29) as sensitive and specific for acute right ventricular infarction. However, these findings could not be corroborated in other studies (30-36). In addition, the majority of electrocardiographic studies conducted to identify simple and useful criteria for the diagnosis of right ventricular infarction excluded patients with anatomic or electrocardiographic criteria of anterior

left ventricular infarction (17-19,25,26,29) on the basis that it may be accompanied by the same electrocardiographic alterations (37,38), introducing a bias that decreases the validity of the aforementioned diagnostic criteria.

To further evaluate the diagnostic accuracy of abnormalities in right precordial leads (V₄R, V₃R, V₁, V₂ and V₃) as diagnostic criteria for right ventricular infarction, the presence of a Q wave and ST segment elevation in these electrocardiographic leads was investigated in a group of patients with acute myocardial infarction whose location was identified in postmortem studies.

Methods

Study patients. Forty-three autopsy patients with acute myocardial infarction form the basis of this study. Thirty were men and 13 women, and their ages ranged from 40 to 85 years (mean 65.3).

The following patients were excluded: patients with cardiomyopathy, valvular disease or associated pericardial dis-

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ease, those admitted after 12 hours of evolution of symptoms, patients with left bundle branch block, patients without a record of leads V_4R and V_3R and those with right ventricular infarction secondary to acute pulmonary embolism because of possible electrocardiographic changes occurring after this event.

Anatomic study. Serial sections of the heart perpendicular to its longitudinal axis were obtained. The different sections were incubated in tetrazolium tetraclorure solution (39) to identify possible necrotic or ischemic areas. These areas were then examined to perform the histologic diagnosis of acute myocardial infarction.

Necrosis of the right ventricle was classified according to the method of Isner and Roberts (40) into the following types (Fig. 1): I = necrosis of less than 50% of the posterior wall; II = necrosis confined to the posterior wall, involving more than 50% of it; III = necrosis of the posterior wall and of less than 50% of the anterolateral wall; and IV = necrosis of the posterior wall and of more than 50% of the anterolateral wall. A grade V was added to this classification, it corresponding to necrosis of a portion of the anterolateral wall without involvement of the right ventricular posterior wall (41). This division of the right ventricular wall does not correspond to the anatomic location of the heart within the thorax. In vivo, the anterolateral wall of the right ventricle is placed anteriorly, behind the sternum. The posterior wall is right and inferior.

Necrosis of the left ventricle was classified as anterior or posterior independent of the possible lesions of the lateral wall. Necrosis of the interventricular septum was also classified as anterior or posterior (Fig. 1).

Electrocardiographic study. An electrocardiogram containing the 12 conventional leads and leads V_3R and V_4R was obtained in every patient at 12 hour intervals during the first 24 hours of evolution of symptoms and then at least each 24 hours thereafter. An electrocardiogram with the maximal ST segment elevation in leads V_4R and V_3R

throughout the evolution of the patient's infarction was selected for analysis. The presence of a pathologic Q wave in leads V_4R , V_3R , V_1 , V_2 and V_3 and elevation of the ST segment (measured in millivolts) in the same leads were analyzed in each case. Elevation of the ST segment greater than 0.05 mV was considered positive.

Infarction groups. The patients were classified into two groups according to the presence or absence of right ventricular infarction in the postmortem study. Group A included 21 patients with acute myocardial infarction involving the right ventricle. According to the location of the necrosis in the right ventricle, group A was classified in two subgroups: group AI, 14 patients with type I to IV right ventricular infarction, and group AII, 7 patients with type V or multifocal nontransmural (1 patient) right ventricular infarction. Group B included 22 patients with acute myocardial infarction without right ventricular involvement.

Statistical analysis. The sensitivity and specificity of two electrocardiographic criteria (presence of a Q wave and ST segment elevation) were calculated in each lead (V_4R , V_3R , V_1 , V_2 and V_3). The sensitivity, specificity, predictive value and diagnostic efficiency of ST segment elevation in lead V_4R being higher than that in leads V_1 to V_3 , as well as the predictive value and diagnostic efficiency of ST segment elevation in lead V_4R , were also calculated according to the following definitions (42):

Sensitivity: percent of cases with each of the three mentioned electrocardiographic criteria in group AI.

Specificity: percent of patients without each of the mentioned electrocardiographic criteria in group B.

Predictive value of the presence of ST segment elevation in lead V_4R and of elevation of the ST segment in lead V_4R being higher than that in leads V_1 to V_3 : percent of patients in group AI among all patients with these electrocardiographic criteria.

Predictive value of the absence of ST segment elevation in lead V_4R and of elevation of the ST segment in lead V_4R being higher than that in leads V_1 to V_3 : percent of patients

Figure 1. Schematic representation of a section of the heart perpendicular to its longitudinal axis, where the necrosis of the right ventricle is classified.

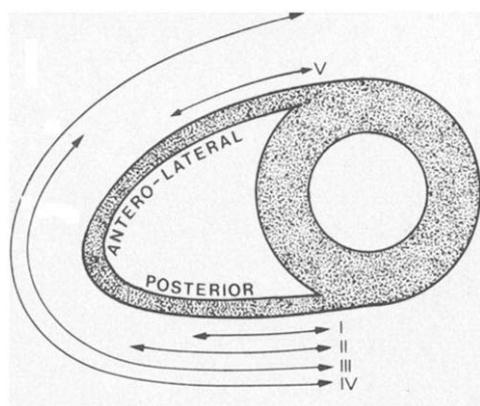
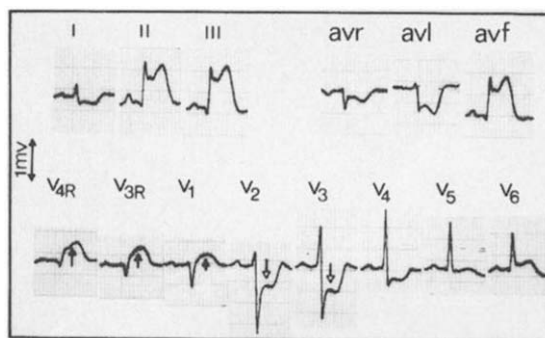


Figure 2. Electrocardiogram of a patient with acute inferior myocardial infarction and right ventricular involvement. Although the ST segment is elevated in lead V_1 , the elevation in lead V_4R is higher than in lead V_1 .



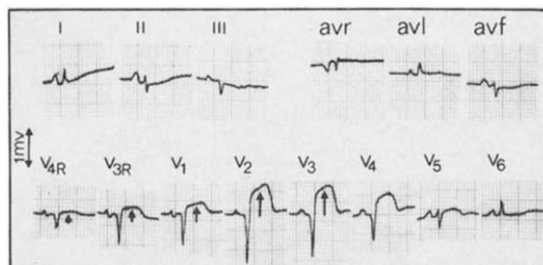


Figure 3. Electrocardiogram of a patient with acute anterior myocardial infarction, without right ventricular involvement. The ST segment is elevated in leads V_1 to V_4 and the elevation in leads V_2 to V_3 is higher than that in leads V_3R to V_4R .

in group B among all patients without these electrocardiographic criteria.

Diagnostic efficiency of ST segment elevation in lead V_4R and of elevation of the ST segment in lead V_4R being higher than that in leads V_1 to V_3 : percent of patients correctly diagnosed with or without right ventricular infarction according to the mentioned electrocardiographic criteria.

Results

Myocardial necrosis. In group AI, by definition, right ventricular necrosis was of type I to IV in all 14 patients. Left ventricular necrosis was located in the posterior wall in 11 patients and in the anterior as well as the posterior wall in 3. Necrosis of the interventricular septum was present in 13 patients; in 7 of these, it was confined to the posterior half of the septum and in 6 the necrosis extended to the anterior and posterior portions of the septum.

In group AII, right ventricular necrosis was of type V in six patients and nontransmural with focal necrosis of the lateral right ventricular wall in one patient. Left ventricular necrosis was located in the anterior wall in four patients, in both anterior and posterior walls in one patient, in the posterior wall in one patient and subendocardial in the remaining patient. Necrosis of the interventricular septum was

present in six patients, in three of these, it was located in the anterior half of the septum and in three it involved the anterior and posterior portions of the septum.

In group B (without right ventricular involvement), 15 patients had necrosis of the anterior left ventricular wall, 6 had necrosis of the posterior wall and in 1 patient the necrosis was subendocardial. Necrosis of the interventricular septum was present in 20 patients; in 6 it was located in the posterior half of the septum, in 10 it was anterior and in 4 it involved both the anterior and posterior portions of the septum.

Electrocardiographic findings. In group AI (14 patients), a Q wave was present in lead V_4R in 11 patients, in lead V_3R in 10, in lead V_1 in 7, in lead V_2 in 4 and in lead V_3 in 5. ST segment elevation was found in all 14 patients in lead V_4R , in 11 in lead V_3R , in 6 in lead V_1 , in 5 in lead V_2 and in 7 in lead V_3 . ST segment elevation in leads V_1 to V_3 greater in magnitude than that found in lead V_4R was present only in 3 of the 14 patients with involvement of the right ventricular posterior wall. Left ventricular necrosis was anterior and posterior, with complete necrosis of the interventricular septum in two of these three; infarction located in the posterior left ventricular wall and posterior portion of the interventricular septum was present in the other. In the 11 remaining patients, either there was no ST segment elevation in leads V_1 to V_3 or it was less in magnitude than that found in lead V_4R (Fig. 2).

In group AII (seven patients), a Q wave was present in four patients in leads V_4R , V_3R , V_1 and V_2 , and in five in lead V_3 . ST segment elevation was found in one patient in leads V_4R and V_3R , in two in lead V_1 and in five in leads V_2 and V_3 .

In group B (22 patients), a Q wave was present in lead V_4R in 13 patients, in lead V_3R in 14, in leads V_1 and V_2 in 17 and in lead V_3 in 16. Upward displacement of the ST segment was found in leads V_4R and V_3R in 7 patients, in lead V_1 in 12, in lead V_2 in 16 and in lead V_3 in 15. When present, the magnitude of ST segment elevation in lead V_4R was always less than that found in leads V_1 to V_3 (Fig. 3).

Table 1. Statistical Difference Among Groups

Group	V_4R	V_3R	V_1	V_2	V_3
Q wave					
AI/B	NS	NS	NS	0.02†	0.03†
AII/B	NS	NS	NS	NS	NS
↑ ST					
AI/B	0.001*	0.01*	NS	0.03†	0.03†
AII/B	NS	NS	NS	NS	NS
↑ ST V_4R > ↑ ST V_1 - V_3					
AI/B		0.001*			
AII/B		NS			

*Greater incidence in group AI; †greater incidence in group B. AI = posterior right ventricular infarction; AII = anterior right ventricular infarction; B = no right ventricular infarction; ↑ ST = presence of ST segment elevations.

Table 2. Sensitivity and Specificity of Electrocardiographic Alterations

Lead	Sensitivity		Specificity	
	%	95% Confidence Limits	%	95% Confidence limits
Q wave				
V ₄ R	78.6	(42.9 to 95.3)	40.0	(20.7 to 63.6)
V ₃ R	71.4	(41.9 to 91.6)	36.0	(17.2 to 59.3)
V ₁	50.0	(23.0 to 77.0)	22.7	(7.8 to 45.4)
V ₂	28.6	(8.4 to 58.1)	22.8	(7.8 to 45.4)
V ₃	35.7	(12.8 to 64.9)	27.3	(10.7 to 50.2)
↑ ST				
V ₄ R	100	(76.8 to 100)	68.2	(45.1 to 86.1)
V ₃ R	78.6	(49.2 to 95.3)	68.2	(45.1 to 86.1)
V ₁	42.9	(17.7 to 71.1)	45.5	(24.9 to 67.8)
V ₂	35.7	(12.8 to 64.9)	27.3	(10.7 to 50.2)
V ₃	50.0	(23.0 to 77.0)	31.9	(13.9 to 54.9)

↑ ST = presence of ST segment elevation.

Statistical differences among groups (Table 1). When comparing the patients in subgroups AI and AII with acute right ventricular infarction with those in group B, the incidence of a Q wave and ST segment elevation leads in V₂ and V₃ was higher in group B than in group AI, but the incidence of ST segment elevation in leads V₄R and V₃R was higher in group AI than in group B. The incidence of ST segment elevation in lead V₄R being greater than that in leads V₁ to V₃ was higher in group AI than in group B. No statistical differences were found between group AII and group B in any of the studied variables.

Sensitivity and specificity of electrocardiographic alterations (Tables 2 and 3). The sensitivity of a Q wave was maximum in leads V₄R and V₃R, decreasing in the other precordial leads studied. However, the specificity of the Q wave was very low, its maximal value being 40% in lead V₄R (Fig. 4).

ST segment elevation (Fig. 5). The sensitivity of this criterion was higher than that of the Q wave, reaching 100% (95% confidence limits = 76.8 to 100%) in lead V₄R, and decreased in leads V₃R, V₁, V₂ and V₃. However, its specificity was low (68% in lead V₄R) and its efficiency in establishing a correct diagnosis only reached 80.6% in lead V₄R. The negative predictive value of lead V₄R reached 100%.

ST segment elevation in lead V₄R greater than that in leads V₁ to V₃ (Fig. 6). The specificity and predictive value of this criterion were 100%, but its sensitivity was lower than that of the simple criterion of ST segment elevation lead V₄R. Its efficiency in establishing a correct diagnosis reached 91.7%.

Discussion

Electrocardiographic findings in right ventricular infarction. Data from previous studies indicate that an abnormal Q wave and particularly ST segment elevation in one or more right precordial leads (V₃R to V₆R) are both highly sensitive (<70%) and specific (<80%) in identifying patients with right ventricular infarction, as defined by technetium pyrophosphate scintigraphy (16,18,22), two-dimensional echocardiography (23) and postmortem studies (7,12,21). Erhardt et al. (7) were the first to study the significance of ST segment elevation in right precordial leads in acute inferior myocardial infarction. In their group of 18 patients with postmortem study, no patient with purely left inferior ventricular infarction showed ST segment elevation in lead CR₄R, in contrast to all the patients with additional involvement of the posterior right ventricular wall. However, the authors stressed that this finding is only valid

Table 3. Sensitivity, Specificity, Predictive Value and Diagnostic Efficiency of Electrocardiographic Alterations

Criteria	Sensitivity	Specificity	Predictive Value (test +)	Predictive Value (test -)	Efficiency
↑ ST in lead V ₄ R	100% (76.8 to 100%)	68.2% (45.1 to 86.1%)	66.7% (43.0 to 85.4%)	100% (78.2 to 100%)	80.6% (64.0 to 91.8%)
↑ ST in lead V ₄ R higher than in leads V ₁ to V ₃	78.6% (49.2 to 95.3%)	100% (77.2 to 99.8%)	100% (61.5 to 99.8%)	88% (67.4 to 97.3%)	91.7% (73.9 to 96.9%)

The 95% confidence limits are indicated in parentheses. ↑ ST = presence of ST segment elevation.

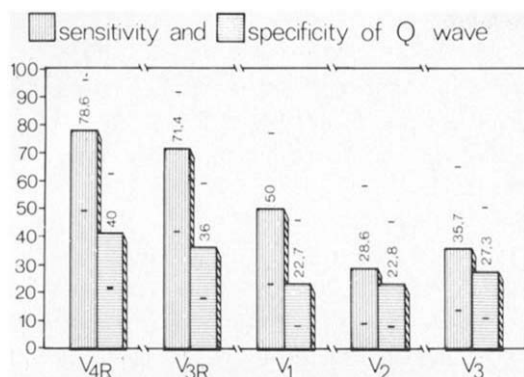


Figure 4. Sensitivity and specificity of the Q wave in the diagnosis of acute right ventricular infarction considering different electrocardiographic leads.

in the absence of anteroseptal infarction, which may interfere with the electrocardiographic interpretation concerning right ventricular infarction, and that small right ventricular infarcts may not show any alteration in lead CR₄R.

Of similar studies were conducted by others (12,21,22), the majority excluded patients with left ventricular infarction of the anterior wall, thereby introducing a bias that decreased sensitivity in the proposed electrocardiographic criteria for the diagnosis of right ventricular infarction. Furthermore, right ventricular necrosis may produce electrocardiographic abnormalities (Q wave and ST segment elevation) in leads V₁ to V₃ (13,14,28,30) that are classically considered the hallmark of anterior or anteroseptal infarction of the left ventricle.

In this study, the aforementioned alterations in leads V₄R, V₃R and V₁ to V₃ were found in patients with and without right ventricular involvement, with anterior left ventricular necrosis as well as with posterior left ventricular necrosis extending to the right ventricle. Therefore, we can conclude that anteroseptal left ventricular infarction and inferior left ventricular infarction with right ventricular involvement may

Figure 5. Sensitivity and specificity of ST segment elevation (↑ST) in the diagnosis of acute right ventricular infarction considering different electrocardiographic leads.

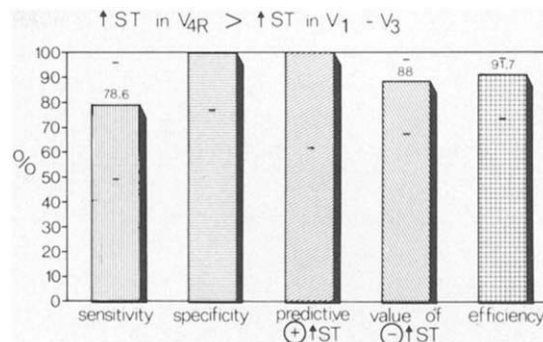
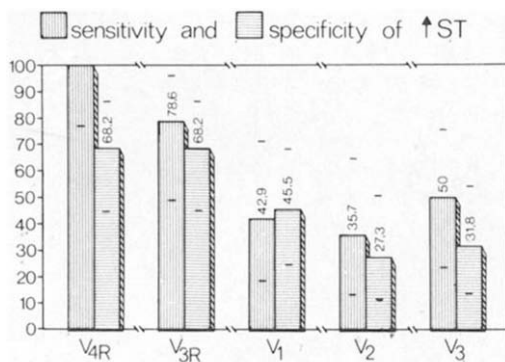


Figure 6. Sensitivity, specificity, predictive value (positive and negative) and efficiency in the diagnosis of acute right ventricular infarction of the criterion of ST segment elevation (↑ST) in lead V₄R being greater than that in leads V₁ to V₃.

show the same electrocardiographic alterations, a fact that decreases the specificity of the classic electrocardiographic criteria for the diagnosis of right ventricular infarction as well as of left ventricular infarction.

ST segment elevation in leads V₃R and V₄R. By excluding patients with necrosis of the anterior right ventricular wall (type V) in whom we did not find any electrocardiographic criterion for its diagnosis, only the upward displacement of the ST segment in leads V₃R and V₄R (mainly in lead V₄R) permitted the differentiation of patients with and without right ventricular infarction. Although the sensitivity of this criterion is high (100% in lead V₄R and nearly 80% in lead V₃R), its specificity as an isolated diagnostic criterion is low, reaching only 68% because the same electrocardiographic alteration was found in some patients with anteroseptal left ventricular necrosis.

ST segment elevation in lead V₄R being greater than that in leads V₁ to V₃. One electrocardiographic criterion of great practical interest that has not been previously described allows the identification of a high percent of patients with right ventricular infarction with a high specificity. This criterion is a greater magnitude of ST segment elevation in lead V₄R than in leads V₁ to V₃ (Table 3, Fig. 5). Its limitations (less sensitivity than the criterion of ST segment elevation in lead V₄R alone) are a consequence of the existence of right ventricular infarction associated with necrosis of both the anterior and posterior left ventricular wall as well as the ventricular septum, in which the most striking ST segment displacement may be found in leads V₁ to V₃. Additionally, in lead V₄R the magnitude of all electrocardiographic deflections is decreased. This may be due, among several other factors, to its location being more distant from the heart than leads V₁ to V₃. Hence, a lesser degree of ST segment elevation in lead V₄R does not necessarily reflect a lesser degree of injury. Despite these limitations, this criterion is more effective than the simple analysis of ST segment elevation alone and should be used in routine clinical practice (Table 3).

Origin of the electrocardiographic alterations in right ventricular infarction. Although there is strong evidence to support the electrocardiographic diagnosis of right ventricular infarction, it is difficult to establish whether these alterations are a direct consequence of right ventricular necrosis or are secondary to necrosis of the interventricular septum. Right ventricular infarction is usually the extension of an inferior left ventricular infarction with septal involvement (5,36), and both conditions may be manifested by changes in the precordial leads over the right ventricle (31).

To establish the origin of the electrocardiographic alterations, it would be necessary to select a group of patients with pure right ventricular infarction and compare the electrocardiographic alterations found with those observed in patients with necrosis of the posterior septum without right ventricular involvement. In dogs with selective necrosis of the posterior wall of the right ventricle (with or without necrosis of the posterior septum), QS complexes and ST segment elevation were recorded in leads V_3R to V_6R (10). Because the right ventricular posterior wall is oriented toward the right lateral segments of the thorax, leads V_3R to V_6R (and sometimes lead V_1) capture the potential variations (ST segment elevation) of this wall. QS complexes are attributed to the transmission of endocavitary potentials of the right ventricle, which are QS or QR in the middle and high portions (10). In contrast, necrosis of the left ventricular posterior wall is better detected by leads V_2 and V_3 , opposite the necrotic wall. The increase in magnitude of the R wave is due to the absence of posterior forces, and ST segment depression is a mirror image of posterior epicardial injury (Fig. 2).

In support of the right ventricular origin of electrocardiographic alterations, there are biventricular infarctions without involvement of the ventricular septum (one in this study) in which the ST segment is elevated in lead V_4R . Erhardt et al. (7) did not observe ST segment elevation in patients with posterior septal infarction without right ventricular involvement. However, other investigators (31-33,36) demonstrated that necrosis of the posterior ventricular septum may cause the appearance of Q waves and ST segment elevation in leads V_3R and V_4R in the absence of right ventricular necrosis. One patient in our study showed these alterations, but the magnitude of ST segment elevation in lead V_4R was less than that in leads V_1 to V_3 . Finally, ST segment elevation in lead V_4R in patients with right ventricular infarction may not be caused by right ventricular necrosis itself, but rather by visualization of the posterior septum through the necrotic right ventricular myocardium (35).

Conclusions. 1) There is no single electrocardiographic alteration to identify infarction of the anterior right ventricular wall (type V). 2) Infarction of the posterior and lateral wall of the right ventricle (types I to IV) is usually accompanied by a Q wave and ST segment elevation in the right

precordial leads. Sensitivity of diagnosis is maximum for the criterion of ST segment elevation in lead V_4R (100%) and decreases from leads V_3R to V_3 . 3) The specificity of a Q wave and ST segment elevation is relatively small because both alterations may be the consequence of several infarct locations. Specificity is maximum (68%) for the criterion of ST segment elevation in leads V_4R and V_3R . 4) The criterion of greater ST segment elevation in lead V_4R than in leads V_1 to V_3 is less sensitive than ST elevation in lead V_4R alone, but offers a higher specificity, predictive value and efficiency in the diagnosis of acute right ventricular infarction because left anterior infarction usually shows greater ST segment elevation in leads V_1 to V_3 than in lead V_4R .

References

1. Myers G, Klein HA, Hiratzka T. Correlation of electrocardiographic and pathologic findings in infarction of the interventricular septum and right ventricle. *Am Heart J* 1949;3:720-70.
2. Levy L, Hyman AL. Difficulties in electrocardiographic diagnosis of myocardial infarction. *Am Heart J* 1950;39:243-54.
3. Zaus EA, Dearn WM. Massive infarction of the right ventricle and right atrium: report of a case. *Circulation* 1952;6:593-8.
4. Abildskov JA, Boyle RS. Further studies of the electrocardiographic effects of experimental myocardial lesions. *Am Heart J* 1965;69:49-60.
5. Erhardt LR. Clinical and pathological observations in different types of acute myocardial infarction: a study of 84 patients deceased after treatment in a coronary care unit. *Acta Med Scand* 1974;560(suppl 1):1-78.
6. Erhardt LR. Right ventricular involvement in acute myocardial infarction. *Eur J Cardiol* 1976;4:411-8.
7. Erhardt LR, Sjögren A, Wahlberg I. Single right sided precordial lead in the diagnosis of right ventricular involvement in inferior myocardial infarction. *Am Heart J* 1976;93:571-6.
8. Daubert JC, Deplace C, Bourdonnec C, Pony JC, Gouffault J. L'infarctus du ventricule droit. I. Diagnostic hémodynamique. Corrélations anatomiques. *Arch Mal Coeur* 1977;70:243-55.
9. Perrault MA, Leclercq JF, Masquet Ch, Nitenberg G, Slama R, Bouvrain Y. Infarctus massif biventriculaire avec rupture d'un pilier mitral et d'un pilier tricuspide. Etude anatomique. *Arch Mal Coeur* 1977;70:1091-5.
10. Medrano GA, De Micheli A. Right posterior necrosis. An experimental study. *J Electrocardiol* 1979;12:197-204.
11. Daubert JC, Langella B, Deplace C, et al. Fréquence et pronostic de l'atteinte ventriculaire droite à la phase aiguë de l'infarctus du myocarde. *Arch Mal Coeur* 1980;73:785-94.
12. Dolgoplosk NA, Libov IA, Miliaeva LV. Diagnosis of right ventricular infarct. *Kardiologia* 1980;20:104-6.
13. Middelhoff C, Buthrer W, Becker AE. Pure right ventricular infarction. *Eur Heart J* 1980;1:369-74.
14. Lloyd EA, Gersh BJ, Kennelly BM. Hemodynamic spectrum of "dominant" right ventricular infarction in 19 patients. *Am J Cardiol* 1981;48:1016-22.
15. Juma Z, Castellanos A, Myerburg RJ. Prognostic significance of the electrocardiogram in patients with coronary heart disease. In: Wellens HJJ, Kulbertus HE, eds. *What's New in Electrocardiography?* The Netherlands: Martinus Nijhoff, 1981:1-22.
16. Candell-Riera J, Figueras J, Valle V, et al. Right ventricular infarction: relationships between ST segment elevation in V_4R and hemodynamic,

- scintigraphic and echocardiographic findings in patients with acute inferior myocardial infarction. *Am Heart J* 1981;101:281-7.
17. Sareli P, Tordjman T, Klein HO, et al. The very early recognition of right ventricular infarction (abstr). *Circulation* 1981;64(suppl IV):IV-141.
18. Braat SH, Brugada P, de Zwaan C, Coenegracht J, Wellens HJJ. Value of electrocardiogram in diagnosing right ventricular involvement in patients with acute inferior wall myocardial infarction. *Eur Heart J* 1983;49:368-72.
19. Chou TC, Van der Bel-Kahn J, Allen J, Brockmeier L, Fowler NO. Electrocardiographic diagnosis of right ventricular infarction. *Am J Med* 1981;70:1175-80.
20. Fiol M, Jaume G, Orellana J, Ibañez J, Guardiola F, Garcia S. Infarto agudo de miocardio de ventriculo derecho. Correlación electrocardiográfica hemodinámica. *Med Clin* 1981;77:195-200.
21. Morgera T, Alberti E, Silvestri F, Pandullo C, Della Mea MT, Camerini F. Right precordial ST and QRS changes in the diagnosis of right ventricular infarction. *Am Heart J* 1984;108:13-8.
22. Croft CH, Nicod P, Corbett JR, et al. Detection of acute right ventricular infarction by right precordial electrocardiography. *Am J Cardiol* 1982;50:421-7.
23. Lombera F, Lopez-Sendon J, Garcia-Fernandez MA, et al. Alteraciones de la contractilidad del ventriculo derecho en el infarto agudo de miocardio inferior. Correlación con V₄R (abstr). *Rev Esp Cardiol* 1982;35(suppl I):34.
24. Willerson JT, Nicod P, Lewis S, Willerson JT, Rude RE. Detection of right ventricular infarction by right precordial electrocardiograms (abstr). *Am J Cardiol* 1982;49:1030.
25. Braat SH, Brugada P, De Zwaan C, Den Dulk K, Wellens HJJ. Right and left ventricular ejection fraction in acute inferior wall infarction with or without ST segment elevation in lead V₄R. *J Am Coll Cardiol* 1984;4:940-4.
26. Funk M, Tyndall AVB. The diagnostic reliability of right precordial electrocardiogram leads in right ventricular infarction (abstr). *Circulation* 1984;70(suppl II):II-386.
27. Forman M, Goodin J, Phelan B, Kopelman H, Virmani R. Electrocardiographic changes associated with isolated right ventricular infarction. *J Am Coll Cardiol* 1984;4:640-3.
28. Geft IL, Shah PK, Rodriguez L, et al. ST elevations in leads V₁ to V₅ may be caused by right coronary artery occlusion and acute right ventricular infarction. *Am J Cardiol* 1984;53:991-6.
29. Bellamy GR, Rasmussen HH, Nasser FN, Cooper RA, Wiseman JC. Evaluation of non-invasive diagnosis of right ventricular infarction (abstr). *Circulation* 1984;70(suppl II):II-310.
30. Roubelakis G, Grosogoeat Y, Lenégre J. Les infarctus isolés du ventricule droit. Etude anatomo-clinique. *Arch Mal Coeur* 1965;58:1420-38.
31. Roubelakis G, Grosogoeat Y, Lenégre J. Les infarctus biventriculaires: étude anatomoélectrique. *Arch Mal Coeur* 1966;59:391-403.
32. Chaignon M, Guize L, Barrillon A, Lenégre J. Derivations electrocardiographiques V₄R-V₃R et conduction auriculo-ventriculaire dans les infarctus myocardiques posterieurs. *Arch Mal Coeur* 1974;67:809-19.
33. Barrillon A, Chaignon M, Guize L, Gerbaux A. Premonitory sign of heart block in acute posterior myocardial infarction. *Br Heart J* 1975;37:2-8.
34. Slama R, Masquet C, Sébastien P, et al. Les perforations septales de l'infarctus myocardique. Progres et limites de leur traitement actuel. *Arch Mal Coeur* 1975;68:449-58.
35. Erhardt LR, Sjögren A. Electrocardiographic changes in right ventricular infarction. A case report. *Acta Med Scand* 1978;204:331-3.
36. San José JM, Domenech LJ, Prieto JA, Riesgo D, Gutierrez J. Alteraciones de la conducción A-V en el infarto inferior y derivaciones precordiales derechas (V₃R-V₄R). *Rev Esp Cardiol* 1978;31:179-84.
37. Hill IGW, Johnston FD, Wilson FN. Form of the electrocardiogram in experimental myocardial infarction. Later effects produced by ligation of the right coronary artery. *Am Heart J* 1938;16:309-20.
38. Osher HL, Wolff L. The diagnosis of infarction of the interventricular septum. *Am Heart J* 1953;45:429-40.
39. Lie JT, Pairolero PC, Holley KE, Titus JL. Macroscopic enzyme-mapping verification of large homogeneous experimental myocardial infarcts of predictable size and localization in dogs. *J Thorac Cardiovasc Surg* 1975;69:599-605.
40. Isner JM, Roberts WC. Right ventricular infarction complicating left ventricular infarction secondary to coronary artery disease. *Am J Cardiol* 1978;42:885-94.
41. Lopez-Sendon J, Coma-Cannella I, Gamallo C. Sensitivity and specificity of hemodynamic criteria in the diagnosis of acute right ventricular infarction. *Circulation* 1981;64:515-25.
42. Galen RS. Predictive value of laboratory tests. *Am J Cardiol* 1975;36:536-8.