The Early Recognition of Right Ventricular Infarction: Diagnostic Accuracy of the Electrocardiographic V₄R Lead

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SUMMARY The sensitivity and specificity of ST-segment elevation in the right precordial lead V₄R as an early indicator of right ventricular infarction were examined in a consecutive series of 110 patients admitted for acute inferior myocardial infarction. The sensitivity was 82.7%, the specificity 76.9% and the positive predictive value 70% in 58 patients with right ventricular infarction documented by autopsy or a combination of radionuclide ventriculography and one or more of the following tests: echocardiography, technetium-99m pyrophosphate scintigraphy and hemodynamic monitoring. The negative predictive value was 87.7%. Because of its simplicity and its high sensitivity and specificity, recording of V₄R should be an intrinsic part of the early evaluation and electrocardiographic examination of acute inferior wall infarction.

THE POTENTIALLY serious and unique hemodynamic consequences of right ventricular infarction were first pointed out by Cohn et al. in 1974. Since then, numerous investigators have examined its recognition and treatment. Its incidence, once thought to be low, has been reported to be as high as 25–45% in patients with inferior wall myocardial infarction. The clinical recognition of right ventricular infarction now rests largely on a typical, but not uniformly present, clinical picture, backed by laboratory evidence of right ventricular dilatation and dysfunction by catheterization, radionuclide ventriculography and echocardiography. Myocardial infarct scintigraphy with technetium-99m (stannous) pyrophosphate (⁹⁹mTc-PYP) is used to detect necrosis in the position typical of right ventricular myocardium.

Early recognition of right ventricular infarction is important because the time of onset of its hemodynamic consequences is unpredictable and can arise as early as within the first hour after the acute event. Prompt fluid therapy may abort the vicious cycle set in motion by right ventricular infarction, which if treated in the conventional way or neglected tends to lead to the true cardiogenic shock usually associated with extensive left ventricular myocardial infarction.

Therefore, the diagnostic criteria cited above tend to be disappointing since they cannot be performed or evaluated in time to avoid the complications of right ventricular infarction. Routine catheterization is evidently not feasible nor desirable. Any diagnostic tool that would help detect or raise suspicion of right ventricular infarction at an early stage would be helpful.

Erhardt suggested in 1974 that right ventricular infarction could be diagnosed electrocardiographically by elevation of the ST segment in the right precordial lead CR₄R. Because of the obvious importance of a simple and rapid noninvasive test for right ventricular infarction, we decided to evaluate the diagnostic accuracy of the more widely available electrocardiographic lead V₄R as an early diagnostic criterion for right ventricular infarction in a prospective series of patients admitted with inferior myocardial infarction.

Protocol

The precordial lead V₄R was recorded in the right fourth intercostal space at the midclavicular line. A standard 12-lead ECG was obtained at admission for acute inferior myocardial infarction and at the time of additional chest pain. The infarction occurred within 10 hours before admission to the study in all cases, and usually within 5 hours.

Acute inferior myocardial infarction was diagnosed by the presence of chest pain, typical ST elevation and development of new Q waves in leads II, III and aV₅, with or without additional similar changes in leads V₅ and V₆ (lateral extension) and increased serum enzymes (glutamic oxalacetic transaminase and total creatine kinase).

All patients with an elevated ST segment in lead V₁ were excluded because ST elevation in V₄R would not be specific enough for right ventricular infarction in the presence of an anteriorly oriented ST vector. Thus, patients with patterns of anteroseptal or anterior wall infarction in addition to the acute inferior infarction were not included. Patients with pericardial disease or left bundle branch block were excluded for the same reason. Patients with chronic lung disease were excluded because of the right ventricular dysfunction often present in this disease. Restrictive cardiomyopathy, pericardial tamponade and rupture of the interventricular septum were not observed during this study.

Based on the criteria of acute inferior myocardial infarction listed above, 110 consecutive patients were admitted to the study from September 1979 to July 1981, and the presence of right ventricular infarction...
was searched for in lead V₄R. The ST-segment shift was considered to be significant if it was elevated 0.5 mm or more above the isoelectric line.²⁹ Examples are shown in figures 1 and 2. All 110 patients underwent careful clinical evaluation. When necessitated by the presence of congestive heart failure or hypotension, hemodynamics were monitored with a Swan-Ganz catheter.³⁰

Techniques

As soon as the clinical state permitted, patients were examined in the Department of Nuclear Medicine. This took place usually on the third day after admission. The patients underwent gated radionuclide equilibrium ventriculography in the 45° left anterior oblique position and scintigraphy with ⁹⁹mTc-PYP to support or exclude the diagnosis of right ventricular infarction.

Equilibrium radionuclide ventriculography was performed according to previously standardized techniques²⁹⁻³⁸ with a scintillation camera equipped with a medium-resolution, high-sensitivity collimator (Elscent CE-1 and CCL-3). The data were acquired in the synchronized multigated acquisition mode, using the R wave of the ECG, and fed into a minicomputer (Elscent Dykomette). Ten minutes after i.v. injection of lyophilized stannous pyrophosphate, 30 mCi of technetium-⁹⁹m pertechnetate were injected to label the red blood cells in vivo. After blood pool equilibrium, up to 3000 kilocounts were acquired with the patient in the 45° left anterior oblique position, adjusted for optimal separation of left and right ventricles. Right ventricular dysfunction due to infarction was considered to be present if the right ventricle was grossly dilated; if severe hypokinesia, akinesia or dyskinesia was readily demonstrated in the free wall of the right ventricle, particularly in its apical portion; and if the right ventricular ejection fraction was below 40%. This rigid criterion was used to eliminate all questionable borderline cases; normal right ventricular ejection fraction in our laboratory and elsewhere is well above this value.⁴,⁷,¹⁹,₂⁸,₃₁,₃₂ Ejection fraction was calculated after carefully eliminating the right atrium in systole from the region of interest, so as not to falsely reduce the ejection fraction.¹⁹ All determinations were repeated twice if the result was in doubt and were evaluated by at least two observers. An example of right ventricular infarction is shown in figure 3.

Dilatation of the right ventricle was judged to be present when the right ventricular apex broadened and lost its typical crescent shape (fig. 3). Our criteria for right ventricular dilatation were consistent with previously published criteria.⁴⁻⁷,¹⁴ Segmental wall motion was assessed by two techniques: isocontour lines of end-diastolic and end-systolic frames (fig. 3) and inspection of continuous-loop, real-time display of ventricular volumes on the oscilloscope. In all patients who recovered, the radionuclide study was repeated approximately 2 months after the acute infarction to document the typical improvement in right ventricular function.³³,³⁴

The studies were preserved and independently re-evaluated 6 months later by two of the investigators. The two observers were in complete agreement on normality or abnormality of right ventricular size and wall motion. This degree of agreement was undoubtedly due to the strict criteria originally used to diagnose right ventricular infarction. In a separate series of hemodynamically stable patients, intra- and interobserver variability for right ventricular size, ejection fraction and wall motion were small (less than 5%).

Myocardial infarct scintigraphy was performed between the second and fourth days of hospitalization, 2 hours after injection of 20 mCi of ⁹⁹mTc-PYP. Scans

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**Figure 1.** Serial electrocardiographic changes in a patient with inferior wall and right ventricular infarction. The ST segment is elevated by approximately 2 mm in V₄R on admission and is depressed in V₁ by more than 1 mm. The ST is only minimally elevated in V₄R on the second day and has become isoelectric on the third day.

**Figure 2.** Five examples of the varying appearance of the ST segment in V₄R. The ST segment is only minimally elevated in A (0.5–1 mm). This was considered a positive test in this and other cases, especially when the QRS complex was small in amplitude. Note the presence of right bundle branch block in V₁ in B, the pattern of true posterior wall infarction in C, and the depressed ST segment in V₁ in all three top examples. Only V₄R is shown in D and E.
were obtained in the anteroposterior, left lateral and 45° left anterior oblique views. The scans were interpreted by two observers and graded in the following manner: 0 = no myocardial activity; 1+ = minimal activity; 2+ = mild uptake in the myocardium, less than in bone; 3+ = activity equal in myocardium and in the sternum; 4+ = activity greater in myocardium than in sternum. Right ventricular infarction was considered to be present if uptake of at least 2+ was found in the area adjacent to the sternum, in addition to the inferior wall of the left ventricle. M-mode echocardiography was also performed to confirm the diagnosis of right ventricular dilatation, according to standard procedures and using a left parasternal approach, with a Smith Kline Instruments Ekoline 2022 echocardiograph and a 2.25-MHz transducer. Ventricular diastolic and systolic diameters were considered acceptable only if delineation of septal and right and left endocardial surfaces was technically adequate, just below the level of the mitral valve. Right ventricular dilatation was considered to be present if the right ventricular diameter exceeded 25 mm.

Hemodynamic studies were performed with a Swan-Ganz catheter introduced into the pulmonary circulation. Pressures were measured with a Mennen-Greatbach physiologic pressure transducer (model 922-122-010) on a Mennen-Greatbach Cardiosentinel pressure monitor and recorded on a direct-writing recorder. The midchest level was chosen as the zero point for pressure measurements. The upper limit of normal right ventricular diastolic pressure was considered to be 8 mm Hg, rather than lower values accepted by some, to adhere to strict criteria of right ventricular infarction. We did not exclude patients in whom the capillary wedge pressure was elevated as a result of left ventricular failure, since failure may coexist in both right and left ventricles.

Criteria of Right Ventricular Infarction

It is difficult to ascertain, from a survey of the literature, the sensitivity and specificity of any one of the criteria of right ventricular infarct mentioned above. We therefore decided to adopt rigid criteria for determining the presence of right ventricular infarction. These criteria were (1) a combination of right ventricular dilatation and dysfunction associated with at least one other criterion (i.e., echocardiographic evidence of right ventricular dilatation, uptake of pyrophosphate in the appropriate location, or elevated right ventricular filling pressure) and (2) postmortem finding of right ventricular infarction. Pyrophosphate scintigraphy was used as additional corroborative evidence of right ventricular infarction when it was positive, but was not used to refute it when it was negative. This approach is consonant with the relatively low sensitivity of this test in right ventricular infarction.

Definitions and Formulas

The following formulas were used for evaluation of the results:

\[
\text{Sensitivity} = \frac{TP}{TP + FN} \times 100
\]

\[
\text{Specificity} = \frac{TN}{TN + FP} \times 100
\]

\[
\text{Predictive value (positive)} = \frac{TP}{TP + FP} \times 100
\]

\[
\text{Predictive value (negative)} = \frac{TN}{TN + FN} \times 100
\]

where TP = true positives, TN = true negatives, FP = false positives, and FN = false negatives.

Posterior probability of disease =

\[
\frac{\text{Prevalence} \times P(O \mid D)}{\text{Prevalence} \times P(O \mid D) + (1 - P) \times P(O \mid \tilde{D})},
\]

where prevalence = the prevalence of right ventricular infarction in a large population of patients with inferior wall infarction, or pretest risk of right ventricular infarction; P(O | D) = the probability of the test result, given the presence of disease in an individual patient; and P(O | \tilde{D}) = the probability of the test result, given the absence of disease in a patient.
value (PV), taking prevalence into consideration, were used:

\[
P(V \text{ positive}) = \frac{\text{sensitivity} \times \text{prevalence}}{\text{sensitivity} \times \text{prevalence} + \text{false-positive rate} \times (1 - \text{prevalence})} \times 100
\]

where false-positive rate = \( \frac{\text{FP}/(\text{TN} + \text{FP})}{100} \).

\[
P(V \text{ negative}) = \frac{\text{specificity} \times (1 - \text{prevalence})}{\text{specificity} \times (1 - \text{prevalence}) + \text{false-negative rate} \times \text{prevalence}} \times 100
\]

where false-negative rate = 100 - sensitivity.

Results

Right ventricular infarction was found to be present by the combined clinical and laboratory criteria in 58 of the 110 patients (52.7%) and absent in 52 (47.3%). The mean age was 63 ± 11 years (± SD) in both groups. There were 41 men and 17 women in the group with right ventricular infarction and 41 men and 11 women in the group without.

Right ventricular infarction was diagnosed on the basis of the combined criteria listed in Table 1. Three criteria or more were positive in 33 of 54 patients (61%) in whom these criteria were searched for. Of these 33 patients with three or more criteria, a positive pyrophosphate uptake was one of the criteria in 21 (64%). The diagnosis was established at autopsy in four patients who died before radionuclide studies could be performed. Thus, the diagnosis of right ventricular infarction was firmly established, either by autopsy or by a combination of three or more criteria in 37 of 58 patients (64%) and by two criteria only in 21 patients. (One positive criterion alone was not sufficient for inclusion in the study.)

Clinical Course

In the group of 58 patients with right ventricular infarction, the clinical course was relatively uneventful in 24 patients (41%) and was complicated by hypotension, oliguria, sinus bradycardia or arrest or atrioventricular block, such as has been described in this disease.1, 6, 9-12, 22, 24-26 in 34 patients (59%). In contrast, the course in the 52 patients without right ventricular infarction was uncomplicated in 35 (67%) and was complicated in only 17 (33%).

Radionuclide Ventriculography

Radionuclide ventriculography was performed in 54 of 58 patients with right ventricular infarction and showed right ventricular dilatation, segmental wall akinesia, hypokinesia or dyskinesia and decreased ejection fraction in all. Figure 3 shows a dilated dyskinetic right ventricle. The mean ejection fraction in these 54 patients was 22 ± 8.0% in the acute stage; ejection fraction was less than 30% in 46 of 54 patients (85%) (fig. 4). When the study was again performed approximately 2 months after the acute infarction in 37 patients who were followed by our clinic, global and segmental improvement was found in 35 of 37 patients (95%), and mean ejection fraction improved from 21 ± 8% to 43 ± 9% (p < 0.01) (fig. 5).

Radionuclide ventriculography was also performed in 49 of 52 patients without right ventricular infarction and showed a normal right ventricular volume with well preserved segmental motion and ejection fraction above 40% in 36 and decreased ejection fraction in 13. The mean left ventricular ejection fraction was 53 ± 16% in the group with right ventricular infarction and 47 ± 14% in the group without it (NS).

Hemodynamic Data

Monitoring was performed in 19 of 58 patients with right ventricular infarction and in four of 52 without it. Right ventricular diastolic filling pressure was elevated (above 8 mm Hg) in 14 of 19 patients with right ventricular infarction and was below 8 mm Hg in five. It was elevated in three of four patients without right ventricular infarction in whom monitoring was re-

![Figure 4. Distribution of individual right ventricular ejection fraction (EF) at infarction. EF is below 25% in 37 of 54 patients (69%) and below 30% in 46 of 54 patients (85%).](image-url)
The right ventricular dimension was normal in the group without right ventricular infarction.

**Myocardial Infarct Scintigraphy**

Infarct localization was attempted in 40 patients with right ventricular infarction and in 16 patients without it. Uptake of $^{99m}$Tc-PYP occurred in the right ventricle in addition to that of the left ventricle in 26 of 40 patients with right ventricular infarction. Uptake was present in the right ventricle in only one of the group without right ventricular infarction, was limited to the left ventricle in 13 and was completely negative in two.

**Autopsy Findings**

Fifteen autopsies were performed. Right ventricular infarction was found in 11. Infarction was limited to the posterior wall of the right ventricle in 10 patients, and extended to the free lateral wall to any significant extent in only one. The septum was infarcted to a significant extent in eight. The right coronary artery was occluded or severely stenotic in all 11 cases, and the left coronary system was significantly involved (one or more stenoses of $\geq 50\%$) in eight.

The presence of right ventricular infarction was strongly suspected before death in one of these 11 patients by radionuclide ventriculography alone and was established by radionuclide combined with at least one more criterion in six patients. The clinical course of four patients was so stormy as to preclude any nuclide studies, so that the diagnosis was strongly suspected before death but had to be considered as established only by autopsy, according to the rigid inclusion criteria of the study.

**ST Segment in V4R**

The ST segment was elevated in 48 of 58 patients (82.7%) with proved right ventricular infarction and in 12 of 52 patients (23%) without it. Thus, the sensitivity of ST elevation of 0.5 mm or more in V4R as an electrocardiographic indicator of right ventricular infarction for this group was 82.7% and its specificity was 76.9%. The predictive value of an elevated ST segment in V4R was 80% and the predictive value of a normal ST segment was also 80%.

Because interpretation of the results of any diagnostic test requires consideration of the prevalence of the disease in the population that is tested, the predictive values listed above need modification. It is reasonable to ascribe a value of 40% for the prevalence of right ventricular infarction in patients with inferior wall infarction. If so, the positive predictive value of an elevated ST segment in V4R is reduced to 70.5%, but the negative predictive value of a normal ST in V4R is enhanced to 87.7%.

The ST segment was elevated by 0.5–0.9 mm in 14 patients and by 1 mm or more in 34 (70.8%). The mean ST-segment elevation for the 48 patients was $1.3 \pm 0.1$ mm. When we analyzed separately the data of the 15 patients who were autopsied, ST elevation was present in V4R in 10 of 11 patients with right ventricu-
lar infarction and in two of four without it. Thus, the sensitivity was 90.9% and positive predictive value 83.3% for this subset. Because of the small number of patients examined, specificity could not be determined.

The time course of the ST elevation was as follows. The ST segment became normal within 1 day in 18 patients and remained elevated for more than 1 day in 30 patients. The ST segment normalized within the first few hours in several patients (fig. 6).

When we analyzed the 10 cases of right ventricular infarction without ST elevation in V₄R (false negatives), a reasonable explanation could easily be given in six. In five patients, ST elevation was minimal in leads II, III and aV₅, so that any elevation of the ST segment would also be minimal in V₄R; in one patient, marked ST elevation in V₅-V₆ accompanied the pattern of inferior myocardial infarction. This leftward ST-segment deviation of lateral infarction could neutralize the rightward-oriented ST vector of right ventricular infarction. In the other patients, any ST elevation that might have been present at the time of the initial insult may have quickly disappeared (fig. 6).

Discussion

Several authors have recognized the occurrence of right ventricular dysfunction in the context of acute myocardial infarction but the existence of right ventricular infarction as a clinical entity, either isolated or accompanying left ventricular infarction, was essentially ignored until its hemodynamic consequences were recognized in 1974 by Cohn et al. Since then, efforts at establishing its presence, boosted recently by the refinement of noninvasive diagnostic techniques, have led to the recognition that right ventricular infarction is a frequent companion of infarction of the inferoposterior wall of the left ventricle. A unified and universally accepted set of criteria for diagnosing right ventricular infarction is still lacking because of the highly variable clinical and hemodynamic picture this entity causes. However, its presence along with inferior infarction of the left ventricle can be diagnosed with a reasonable degree of certainty if it can be established that the right ventricle is dilated and exhibits segmental and global dysfunction and if infarct-avid radionuclide accumulates in the region of the right ventricle. The diagnosis is further strengthened if right ventricular function can be shown to return to normal during recovery from the infarction. We also recognize that right ventricular function may be transiently impaired in severe anterior wall infarction of the left ventricle, and we were therefore careful to exclude patients with anterior infarction when establishing our radionuclide criteria of right ventricular infarction. The selection of only patients with inferior infarction is thus a further guarantee of the correctness of the diagnosis of right ventricular infarction in this series.

By using a combination of two or more criteria to indicate either dysfunction of the right ventricle or necrosis in the location of the right ventricular myocardium, we thus established with a reasonable degree of certainty that inferior wall infarction was accompanied by right ventricular infarction in 52.7% of the 110 cases of this series. This figure is somewhat higher than the usual figures that are cited but is not far removed from the figure of 45% cited by Erhardt or that given by Lopez-Sendon et al. In that series, 19 of 32 patients (59.3%) with inferior myocardial infarction had associated right ventricular infarction. The full-blown clinical picture of right ventricular infarction is now easily recognized. However, the hemodynamic consequences of right ventricular infarction may appear unexpectedly after what appears at first to be an uncomplicated inferior wall infarction and it is therefore evidently desirable to have an early simple and sensitive diagnostic clue to the presence or possibility of right ventricular infarction before the complications ensue. Erhardt demonstrated the presence of right ventricular infarction at autopsy in patients with ST elevation in the lead CRₓR and suggested that CRₓR be used as a diagnostic tool. However, it has become clear that the patients who die represent a selected subset of patients with severe right ventricular infarction, so that it is difficult to determine the diagnostic accuracy of CRₓR in a less selected population of patients who have what appears at first to be an uncomplicated inferior myocardial infarction.

Figure 6. Different time course of normalization of ST elevation in V₄R illustrated in two different patients, in whom right ventricular dysfunction persisted for at least a week (ejection fraction 10–15% in both). (Top) ST elevation of 2 mm is present in the two paced beats, the fusion beat (third beat) and in the spontaneous beats in the admission tracing, and disappeared only 2 days later. (Bottom) ST elevation was no longer present in the tracing done 1½ hours after admission in the second patient.
We therefore prospectively evaluated, using a control group, the sensitivity and specificity of lead V₄R as a clue to right ventricular infarction in a cohort of patients with acute inferior wall infarction. Candell-Riera et al.⁴⁶ showed a significant association between ST elevation in V₄R and some of the criteria usually associated with right ventricular infarction. However, certain methodologic problems exist in that study: (1) the inclusion of cases of anterior myocardial infarction, which increases the number of nonspecific ST-segment elevation in V₄R resulting from anterior ST-vector deviation; (2) the absence of nuclide ventriculography, the index that is most frequently affected by right ventricular infarction; (3) the small number of patients and of autopsies performed (only two); and (4) the inability to determine the specificity of ST elevation in V₄R, since the number of patients without right ventricular infarction with ST elevation in V₄R (false positives) is not clearly documented. Analysis of the data of Candell-Riera using a combination of two criteria, as in our study, provides a relatively low sensitivity (59%) for echocardiography and one other criterion or for hemodynamic evidence and one other criterion.

We circumvented these problems by first establishing the diagnosis of right ventricular infarction using a combination of diagnostic tools in a larger number of patients (including more autopsies) and then determining the sensitivity, specificity and predictive value of ST elevation in V₄R, after rejection of cases in which ST elevation could be nonspecific.

We conclude that ST elevation in V₄R in the presence of inferior wall myocardial infarction and in the absence of left bundle branch block or other causes of anteriorly oriented ST vectors is a sensitive clue to the association of right ventricular infarction (82.7% in the overall group, 90.9% in the autopsy subgroup) with a specificity of 76.9%, a positive predictive value of 70.5% and a negative predictive value of 87.7%, if we are right in assuming that the prevalence of right ventricular infarction in patients with inferior infarction approaches 40%. These results probably underestimate the true sensitivity of V₄R because we have noticed that ST elevation in V₄R is often a very transient finding, which disappears sometimes within 2 hours of the onset of chest pain (fig. 6). Our experience suggests that V₄R recorded in the very early phase of infarction, in the mobile coronary care unit or in the emergency room, will detect patients in whom right ventricular infarction results in only fleeting and evanescent ST elevation in V₄R. Several factors will evidently influence the degree of ST elevation in V₄R. (1) It will expectedly be less prominent in V₄R if it is not prominent in II, III and aVF. (2) ST elevation in V₄R is a rightward as well as anteriorly oriented vector. Thus, leftward ST deviation such as that seen in V₅₋₆ because of extension of the infarction to the lateral wall could cancel out the ST elevation in V₄R; and ST elevation in leads V₅ and V₆ seems to decrease the sensitivity of V₄R for right ventricular infarction. (3) We recognize that ST elevation indicates transmural ischemia, rather than infarction. Thus, it might be more properly stated that ST elevation in V₄R represents the phase of transmural ischemia in the right ventricle. This might explain the appearance of 12 "false-positive" cases among 52 patients without right ventricular infarction. Some of these cases could be actual infarcts that were too small to produce ventricular dilatation or dysfunction, but some other cases could represent transmural ischemia not necessarily followed by necrosis, as is not infrequently also observed in the left ventricle.

V₄R is not the only diagnostic criterion that will decide whether or not right ventricular infarction is present, but is a very useful early, simple warning signal to its very likely existence and to the unique complications it creates, even before these complications occur. ST elevation in V₄R points to the need to further confirm or rule out this possibility as quickly as possible by additional tests so as to anticipate the complications and promptly treat them. In view of its reasonably high sensitivity, specificity and predictive value, it may also be sufficient by itself as a confirmatory electrocardiographic sign of right ventricular infarction when the typical clinical picture is already present.

V₄R should be recorded as early as possible in the presence of inferior wall infarction, because ST elevation in V₄R carries a reasonably high sensitivity, specificity and predictive value for right ventricular infarction (or at least ischemia) and because of its simplicity and rapid yield. It should be an intrinsic part of the early electrocardiographic evaluation of inferior wall infarction.

Addendum

Since acceptance of this article, Croft et al. have published an article entitled, "Detection of Acute Right Ventricular Infarction by Right Precordial Electrocardiography" (Am J Cardiol 50: 421, 1982). Their article essentially confirms our own findings.

Acknowledgment

We gratefully acknowledge the contribution and patience of our nursing staff and the invaluable assistance of the unsung heroes of the "Nuclear Shuttle."

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H O Klein, T Tordjman, R Ninio, P Sareli, V Oren, R Lang, J Gefen, C Pauzner, E Di Segni, D David and E Kaplinsky

Circulation. 1983;67:558-565
doi: 10.1161/01.CIR.67.3.558

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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