The Impact of Right Ventricular Infarction on the Prevalence of Ventricular Arrhythmias during Acute Inferior Myocardial Infarction*

Eldad Bechavia, M.D.; Boris Strasberg, M.D.; Jairo Kusniec, M.D.; Nili Zafiri, M.D.; Alex Sagie, M.D.; Avio Mager, M.D.; and Samuel Sclarovsky, M.D.

To determine the impact of RV infarction on the prevalence and complexity of ventricular arrhythmias during inferior AMI, 57 patients with no prior MI were studied by 24-hour Holter monitoring on the first and tenth days of AMI. Based on radionuclear studies, patients were allocated into two groups: (1) group A, 21 patients (37 percent) with normal RVEF (≥ 40 percent); and (2) group B, 36 patients (63 percent) with depressed RVEF (< 40 percent). There were no significant differences between the groups regarding age and LVEF. Values of RVEF were 47 ± 6 percent and 31 ± 6 percent, respectively (p < 0.05). The RVEF had no influence on the prevalence and complexity of early and late arrhythmias. Stratification of patients in group B into two subgroups based on the extent of RV dysfunction did not reveal any differences in the occurrence of all forms of ectopy (when both groups were matched to group A). Therefore, patients with inferior AMI, with or without RV infarction, have a similar prevalence of arrhythmias. Ventricular ectopic beats may be related to the severity and spread of LV involvement, rather than to RV dysfunction.

(RV EF = right ventricular ejection fraction; LVEF = left ventricular ejection fraction; CPK = creatine phosphokinase; RAO = right anterior oblique; VT = ventricular tachycardia; VF = ventricular fibrillation; PVC = premature ventricular contraction.)

Several studies have examined the short-term prognosis of patients with inferior wall AMI complicated by concomitant RV infarction. Mortality, which has been reported to occur in up to 27 percent of the patients with predominant RV infarction, 1 has generally been ascribed to the larger infarcts and to the hemodynamic hazards associated with RV dysfunction. 2, 3 Pfisterer et al. 4 showed an increased risk of sudden death in patients with residual RV dysfunction after inferior wall AMI, independent of LV function. The reported occurrence of VF during temporary pacing has also been attributed to the presence of RV infarction. 5 As the prevalence of ventricular arrhythmias is closely related to the severity of cardiac dysfunction, it may be suggested that RV infarction could be an unappreciated source of ventricular arrhythmias. We therefore undertook a prospective Holter monitoring study in order to assess the prevalence and complexity of early and late ventricular arrhythmias during inferior wall AMI, with and in the absence of RV involvement.

**Materials and Methods**

**Selection of Patients**

Seventy consecutive patients with a first inferior wall AMI who were admitted to the coronary care unit within 4 h from initial symptoms were considered for inclusion in this study. Criteria for exclusion were a previous history or ECG evidence of prior infarction, valvar heart disease, significant obesity, or more than mild chronic pulmonary disease. Pulmonary embolism was not suspected in any of the patients. An AMI was defined by typical chest pain lasting for more than 30 minutes, ECG changes compatible with transmural (Q wave) AMI in the corresponding inferior leads, and the laboratory detection of elevated serum concentrations of cardiac muscle enzyme (at least twice the upper normal limit of CPK). There were 52 men and 18 women, aged 34 to 80 years (mean, 60 years). All patients had 24-hour Holter monitoring during the first 24 hours of admission. Gated equilibrium radionuclide cineangiography (first pass; 30° RAO view) was performed in 66 patients within 48 hours of infarction.

Four patients who died during the first 48 hours of infarction were excluded from the initial population. Nine patients had to be excluded because of temporary pacemaker implantation (three patients) and Swan-Ganz catheterization (two patients) during the Holter monitoring or due to technically unsatisfactory Holter recording and radionuclear analysis (four patients). From the remaining 57 patients who formed the definite study population, 54 had an additional late (tenth day of AMI) Holter monitor recording. Patients were followed for a mean of 12 days. During this period, patients were constantly monitored for serious ventricular rhythm disturbances (VT; VF). One patient who died on the fifth day of AMI and two others who required urgent aortocoronary bypass surgery could not be analyzed by late Holter monitoring. All patients were treated by vasodilators and intravenous heparin, except for those where anticoagulants were contraindicated. During the first hours of infarction, ten patients who were subsequently

---

*From the Israel and Ione Massada Center for Heart Diseases, Beilinson Medical Center, Petah Tikva, and the Tel Aviv University School of Medicine, Tel Aviv, Israel. Manuscript received January 26, 1989; revision accepted April 9, 1990. Reprint requests: Dr. Strasberg, CCU, Beilinson Hospital, Petah Tikva, Israel 49100
classified as having RV infarction and four other patients without RV infarction received intravenous streptokinase. This difference did not reach any statistical significance.

**Radionuclear Ventriculography**

First-pass ventriculographic studies (RAO, 30° view) were performed at rest within 48 hours from initial symptoms. A single-crystal digital camera (Elscint Apex 415) and integrated computer system with a high-efficiency low-resolution collimator was used. Fifteen milliliter of technetium-99m pertechnetate was injected as a bolus in a medial antecubital fossa vein. Acquisition was performed on a 64 × 64-pixel matrix with a frame rate varying from 20 to 32 frames for 30 seconds. Data analysis was accomplished by nearly completely automated processing. The EF of RV and LV were obtained, as well as the segmental functional images utilizing end-diastolic and end-systolic perimeters. Values obtained were compared with the lower limits of normal, as determined in our radionuclear laboratory (RVEF, 46±5 percent; LVEF, –60±6 percent). Thus, significant RV or LV dysfunction was considered to occur when the corresponding calculated EF fell below the SD of the normal values, i.e., less than 40 percent and 54 percent, respectively.

Utilizing the radionuclear ventriculographic data, patients were grouped as follows: Group A consisted of patients with normal RV function (21 patients [37 percent]; mean RVEF, 47±6 percent). Group B consisted of those patients with depressed RV function (36 patients [63 percent]; mean RVEF, 32±6 percent). In order to better assess the relation between RV dysfunction and ventricular ectopic activity, statistical analyses were carried out separately for patients with RVEF ≥30 percent (group B1) and <30 percent (group B2).

**Monitoring Procedure**

Fifty-seven patients had their 24-hour Holter recording during the first 24 hours after admission. An additional Holter recording was carried out in 54 patients ten days after the onset of AMI. No patient was receiving antiarrhythmic agents or β-adrenergic blockers at the time of the Holter studies. The 24-hour Holter monitor tapes were analyzed by an experienced technician with physician revision of data, using a dynamic electrocardioscanner (Del Mar Avionics Innovator 500) capable of digital templating of ventricular ectopic events. At the completion of the analysis, the computerized arrhythmia scanner tabulates the frequency of each arrhythmia and provides a digital printout of the total number of single ectopic beats for each hour of recording.

**Grading of Ventricular Arrhythmias**

Arrhythmias were graded according to a modified Lown classification. All patients were classified into Lown’s class (PVCs <30/h) or class 2 (PVCs >30/h). Complex ventricular arrhythmias, defined as Lown class 3 (multifocal PVCs), 4A (couplets), 4B (VT), and 5 (Ron-T phenomenon), were considered present if they were recorded on at least two separate 1 h occasions. Patients were also individually allocated to the corresponding categories of complex arrhythmias; for example, if a given patient’s Holter recording showed multiformal PVCs, as well as couplets, he was subgrouped into both corresponding subgroups of complete arrhythmias. In addition, within each Lown class, ectopic activity (number of ectopic episodes) was analyzed separately.

**Statistical Methods**

Clinical data (age, EF) are presented as the mean ± SD. Comparison of ventricular arrhythmias was performed using the χ² test for low-grade arrhythmias (Lown class 1 and 2) and the proportion test for high-grade arrhythmias and for the rest of the variables studied. An unpaired t-test was used to quantify and compare the numbers of ectopic episodes in each Lown category. Values for p less than 0.05 were considered to be significant.

**RESULTS**

The results of the present study are summarized in Tables 1 to 4. There were no significant statistical differences between the two main groups of patients regarding age and LV function. The mean values of LVEF were found within the normal limits. As demonstrated in Tables 1 and 2, early and late ventricular ectopic beats were not related to the presence of RV dysfunction. When we separated the group of patients with RV dysfunction into those with RVEF ≥30 percent (group B1) or <30 percent (group B2), no significant difference in ventricular ectopic beats emerged between these subgroups (Tables 3 and 4). Patients with a low RVEF did not reveal a significantly higher incidence of VF or accelerated idioventricular rhythms compared to patients who exhibited normal RV function. Advanced atrioventricular block was observed in six patients of group A (29 percent) and in three patients of group B (8 percent). This difference was statistically significant (p<0.001).

**DISCUSSION**

The potential arrhythmogenic effect of the infarcted
RV during inferior wall AMI has not been fully elucidated. Its possible arrhythmogenic contributing role has been validated previously in experimental studies. In a canine model, where the right coronary artery supplied only the RV, late induction of sustained VT was evident in all dogs subjected to right coronary occlusion.\(^7\) From this observation, assumptions were made that frequent and complex PVCs may represent an electrical instability of the RV, and arrhythmias could occur more frequently in patients having concomitant RV infarction.

Our study was designed to address the impact of RV dysfunction on the prevalence and complexity of ventricular arrhythmias in patients with first inferior wall AMI. The study confirmed a higher prevalence of RV dysfunction (63 percent)\(^4\) and principally demonstrated that the presence of early RV dysfunction did not influence the overall prevalence and severity of early and late ventricular arrhythmias. Furthermore, stratification of patients with RV infarction into two groups based on the extent of RV dysfunction did not reveal any significant differences in the prevalence of all forms of ventricular ectopy, when both groups were compared to each other or to patients with normal RVEF (Tables 3 and 4). Therefore, RV dysfunction during the early phase of infarction does not necessarily imply an increased prevalence of arrhythmias in the presence of well-preserved LV performance. Residual RV dysfunction after a first inferior wall AMI has been shown to increase the risk for complex ventricular activity and for sudden death.\(^4\) This is perhaps not surprising, in view of the relatively larger infarcts in patients with persistent RV involvement. In our study, early RV dysfunction had no influence on the prevalence and complexity of late ventricular arrhythmias. This lack of influence may be attributed to the gradual improvement of RV function in the first few days following AMI.\(^6\) We did not perform a second radionuclide study, so we cannot compare the impact of late residual RV dysfunction on ventricular arrhythmias.

According to our findings, it is far from established that patients with RV infarction are more likely to have ventricular arrhythmias in the setting of inferior wall AMI than patients without RV involvement. Ventricular ectopic beats, especially during the acute ischemic phase of infarction, may be related more closely to other determinants, such as the severity of myocardial flow reduction, the absence of collateral circulation, and the amount of ischemic LV myocardium, rather than to the presence of RV dysfunction.

ACKNOWLEDGMENT: We thank Ms. Silvia Shein and Ms. Judith Sadi for assistance in performing the Holter and statistical analyses. We also thank Gill Sher for preparation of this manuscript.

### REFERENCES


---

Table 3 — Prevalence of Early Ventricular Arrhythmias with Respect to RV Dysfunction

<table>
<thead>
<tr>
<th>Group</th>
<th>Age, yr</th>
<th>LVEF, percent</th>
<th>RVEF, percent</th>
<th>No. in Lown Class (percent)</th>
<th>Mean No. per hr</th>
<th>No. with VF (percent)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (N = 21)*</td>
<td>60 ± 10</td>
<td>63 ± 11</td>
<td>47 ± 6</td>
<td>18 (86) 3 (14) 4 (19) 9 (43) 9 (43) 3 (14)</td>
<td>22 ±31 33 ±46 6 ±8</td>
<td>0</td>
</tr>
<tr>
<td>B1 (N = 22‡)</td>
<td>64 ± 10</td>
<td>62 ± 11</td>
<td>35 ± 3§</td>
<td>17 (77) 5 (23) 1 (5) 14 (64) 14 (64) 1 (5)</td>
<td>24 ±36 18 ±18 5 ± 4</td>
<td>2 (9)</td>
</tr>
<tr>
<td>B2 (N = 14¶)</td>
<td>58 ± 11</td>
<td>62 ± 9</td>
<td>24 ± 4¶</td>
<td>10 (71) 4 (29) 3 (21) 6 (43) 6 (43) 2 (14)</td>
<td>30 ±31 20 ±20 5 ± 4</td>
<td>2 (14)</td>
</tr>
</tbody>
</table>

*RVEF ≥ 40 percent; ‡140 percent > RVEF ≥30 percent; §RVEF <30 percent; ‡p<.005 vs group A; ¶p<.05 vs group B1.

Table 4 — Influence of Early RV Dysfunction during Inferior Wall AMI on Prevalence of Late Ventricular Arrhythmias (Stratification of Patients with RV Dysfunction)

<table>
<thead>
<tr>
<th>Group</th>
<th>No. in Lown Class (percent)</th>
<th>Mean PVCs per hr</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (N = 20)</td>
<td>19 (95) 1 ± 5 1 ± 5 3 (15) 0 1 (5) 8 ± 24</td>
<td></td>
</tr>
<tr>
<td>B1 (N = 20)</td>
<td>19 (95) 1 ± 5 0 1 (5) 0 1 (5) 4 ± 7</td>
<td></td>
</tr>
<tr>
<td>B2 (N = 14)</td>
<td>14 (100) 0 0 1 (7) 0 0 3 ± 5</td>
<td></td>
</tr>
</tbody>
</table>