The incidence of atrial arrhythmias during inferior wall myocardial infarction with and without right ventricular involvement

The atrial arrhythmia profile during inferior wall acute myocardial infarction (AMI) has not been systematically examined with respect to right ventricular (RV) involvement. To this end, 62 consecutive patients with first inferior wall AMI and no other conditions known to increase susceptibility for rhythm disturbances were studied by 24-hour Holter monitoring during the first and tenth day of infarction. Based on radionuclear ventriculography performed on day 2 of infarction, patients were allocated to two groups: group A—36 patients (56%) with right ventricular ejection fraction (RVEF) <40% (mean ± SD, 31 ± 6%) and group B—26 patients (42%) with normal (>40%) RVEF (mean ± SD, 47 ± 5%). There were no significant differences between the two groups with respect to age, sex, or left ventricular (LV) function. In the group as a whole, ectopic activity in the different categories of atrial arrhythmias was significantly higher during the first day than on the tenth day of infarction. Comparing the two groups, 33 patients (92%) in group A had a mean hourly frequency of one or more atrial premature contractions (APCs) during the first day of infarction compared with 16 patients (69%) in group B (p < 0.001). Atrial and supraventricular tachycardia were recorded more frequently in group A patients (16 of 36 [44%] versus 8 of 26 [31%]) as well as atrial fibrillation (AF) (7 of 36 [19%] versus 1 of 26 [4%]). Quantitative analysis showed a similar trend for a higher rate of ectopic events in group A patients. Ectopic activity was neither influenced by LVEF nor by age or sex. Using stepwise regression analysis, RVEF was independently related to the prevalence of AF and APCs. The data presented indicate that patients with inferior wall MI accompanied by RV dysfunction are more prone to develop atrial rhythm disturbances than patients with preserved RV function. Atrial infarction or ischemia, atrial distension, and a raised right atrial pressure may account for this trend. (Am J Cardiol 1992;124:367.)

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The spectrum of atrial arrhythmia ranges from a single atrial premature contraction (APC) to atrial fibrillation (AF). These were commonly observed in patients with acute myocardial infarction (AMI), and have been reported to occur frequently in patients with atrial infarction. The hemodynamic hazards associated with right ventricular (RV) infarction are assuming progressively greater importance. In this respect, an excess prevalence of atrial arrhythmias, especially during the evolutionary vulnerable phase, may have a considerable hemodynamic impact. Although atrial arrhythmias apparently do not carry the same prognostic implications as ventricular arrhythmias, still the accompanying loss of atrioventricular (AV) functional integrity and its potential adverse effect on RV diastolic and systolic function may contribute to the development of cardiogenic shock. Despite widespread clinical concern for RV infarction, the incidence of atrial arrhythmias during inferior wall MI has not yet been documented with respect to RV involvement. We therefore undertook a prospective Holter monitoring (HM) study to assess the prevalence of early and late atrial arrhythmias during inferior wall MI.

METHODS

Study patients. Seventy-five consecutive patients who were admitted to the coronary care unit within 4 hours of symptom onset of an inferior wall MI were considered for inclusion in this prospective study. Patients were ineligible
were used. A bolus of 15 mCi of technetium-99m per techni-graphic studies (30-degree right anterior oblique view) others with no evidence of RV involvement.

with intravenous heparin except for those in whom antico-

eliminated from a second study. All patients were treated

One patient who died on the fifth day of MI and two

had an additional HM study on the tenth day after infarc-

62 patients who formed the definite study population, 59

radionuclear analysis (four patients). From the remaining

patients) or because of technically unsatisfactory recording or

right heart catheterization during the HM period (five pa-

cluded because of temporary pacemaker implantation or

the first 24 hours of infarction. Nine others had to be ex-

sion).

of the upper limit of normal). There were 55 men and 20

cardiac enzymes (creatine kinase had to be more than twice

minutes; by the appearance of pathologic Q waves in the

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Elscint, Inc., Hackensack, N.J.) and integrated computer

were interpreted by an experienced physician.

and questionable episodes of arrhythmias. All recordings

scanned visually and printouts were made from all definite

addition to the computerized analysis, the scanners were

24-hour HM tapes were initially scanned by an experienced

extremely low-resolution collimator were used. A bolus of 15 mCi of technetium 99m pertechnetate was injected in a medial antecubital fossa vein. Ac-

quisition was performed on a 64 x 64 pixel matrix with a

frame rate varying from 20 to 32 frames for 30 seconds.

Data analysis was accomplished by nearly completely au-
tomated processing. Right ventricular ejection fraction (RVEF) and left ventricular ejection fraction (LVEF) were obtained, as well as the segmental functional images utilizing end-diastolic and end-systolic perimeters. Significant RV or LV dysfunction was considered to occur once the corresponding EF fell below the standard deviation of the normal values as determined in our radionuclear labora-
tory (RVEF = 46 ± 5%; LVEF = 60 ± 6%)—less than 40% and 51%, respectively.

Based on the radionuclear data, patients were grouped as follows: group A—36 patients (58%) with depressed RVEF (mean ± SD, 31 ± 6%) and group B—26 patients (42%) with normal RVEF (mean ± SD, 47 ± 5%).

Holter monitoring procedure. Sixty-two patients had 24-hour HM during the first 24 hours following admission. An additional recording was made in 59 patients 10 days after infarction. No patient was receiving antiarrhythmic, inotropic, or β-adrenergic blocking agents at the time of the studies. None of them had clinical signs of pericarditis. The 24-hour HM tapes were initially scanned by an experienced technician, using a dynamic electrocardioscanner (Innovator 500, Del Mar Avionics, Irvine, Calif.) capable of digital templating of atrial and ventricular ectopic events. At the completion of the analysis, the computerized arrhythmic scanner tabulates the frequency of each arrhythmia and provides a digital printout of the total number of single ectopic beats and arrhythmias for each hour of recording. In addition to the computerized analysis, the scanners were scanned visually and printouts were made from all definite and questionable episodes of arrhythmias. All recordings were interpreted by an experienced physician.

Grading of atrial arrhythmias. Atrial arrhythmias were graded from isolated APCs to complex atrial arrhythmias. For convenience, complex atrial arrhythmias were sepa-

rated into two categories (Tables I to III). The first cate-
gory consisted of atrial tachycardia and supraventricular tachycardia, defined as three or more consecutive APCs with an atrial rate greater than 100/min. Atrial fibrillation (AF) was considered as the second category of complex

for inclusion if they had a history or electrocardiographic
evidence of previous MI, aortocoronary bypass surgery or
coronary angioplasty, chronic or paroxysmal atrial ar-
rhythmias, valvular or congenital heart disease, pericardi-
tis, cardiomyopathy, preexisting heart failure, significant
obesity, chronic pulmonary disease or pulmonary hyper-
tension, or if they were receiving digitalis or antiarrhyth-
tic therapy. Thyrotoxicosis or pulmonary embolism were
suspected in none of the patients. An acute inferior wall MI
was defined by typical chest pain lasting for more than 30
minutes; by the appearance of pathologic Q waves in the

In Table I, the values are shown for the number of patients with one or more arrhythmic episodes per 24 hours.

Table I. Prevalence and event rates of early (Holter I) versus late (Holter II) atrial ectopic activity in 59 patients with inferior wall MI

<table>
<thead>
<tr>
<th></th>
<th>APCs</th>
<th>AT + SVT</th>
<th>AF</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M/hr</td>
<td>M/24 hr</td>
<td>M/24 hr</td>
</tr>
<tr>
<td>Holter I</td>
<td>9 ± 14</td>
<td>4 ± 7</td>
<td>2 ± 1</td>
</tr>
<tr>
<td>Holter II</td>
<td>1 ± 2</td>
<td>26 (44)</td>
<td>1</td>
</tr>
<tr>
<td>p Value</td>
<td>&lt;0.00</td>
<td>&lt;0.001</td>
<td>NS</td>
</tr>
</tbody>
</table>

AF, Atrial fibrillation; APCs, atrial premature contractions; AT, atrial tachycardia; M, mean number of ectopic events; MI, myocardial infarction; NS, not significant; SVT, supraventricular tachycardia.

*Number of patients with a mean hourly frequency of one or more atrial premature contractions.

†Number of patients with one or more arrhythmic episodes per 24 hours.
Table II. Prevalence and event rates of atrial ectopic activity during the early phase of inferior wall MI in patients with depressed versus normal RV function

<table>
<thead>
<tr>
<th>Group</th>
<th>Age (yr)</th>
<th>LVEF (%)</th>
<th>RVEF (%)</th>
<th>APCs M/hr</th>
<th>No. of patients (%)*</th>
<th>AT + SVT M/24 hr</th>
<th>No. of patients (%)*</th>
<th>AF M/24 hr</th>
<th>No. of patients (%)†</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (n = 36)</td>
<td>61 ± 11</td>
<td>62 ± 10</td>
<td>31 ± 6</td>
<td>13 ± 17</td>
<td>33 (92)</td>
<td>5 ± 8</td>
<td>16 (44)</td>
<td>3 ± 1</td>
<td>7 (19)</td>
</tr>
<tr>
<td>B (n = 26)</td>
<td>60 ± 9</td>
<td>62 ± 10</td>
<td>47 ± 5</td>
<td>6 ± 5</td>
<td>18 (89)</td>
<td>1</td>
<td>8 (31)</td>
<td>1</td>
<td>1 (4)</td>
</tr>
<tr>
<td>p Value</td>
<td>NS</td>
<td>NS</td>
<td>&lt;0.001</td>
<td>0.05</td>
<td>0.01</td>
<td>NS</td>
<td>0.02</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

LVEF, Left ventricular ejection fraction; RVEF, right ventricular ejection fraction; other abbreviations as in Table 1.

*Number of patients with a mean hourly frequency of one or more atrial premature contractions.
†Number of patients with one or more arrhythmic episodes per 24 hours.

Table III. Prevalence and event rates of atrial ectopic activity during the late phase (tenth day) of inferior wall MI in patients with depressed versus normal RV function

<table>
<thead>
<tr>
<th>Group</th>
<th>Age (yr)</th>
<th>LVEF (%)</th>
<th>RVEF (%)</th>
<th>APCs M/hr</th>
<th>No. of patients (%)*</th>
<th>AT + SVT M/24 hr</th>
<th>No. of patients (%)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (n = 34)</td>
<td>61 ± 11</td>
<td>62 ± 10</td>
<td>30 ± 6</td>
<td>3 ± 3</td>
<td>17 (90)</td>
<td>1</td>
<td>1 (10)</td>
</tr>
<tr>
<td>B (n = 25)</td>
<td>60 ± 9</td>
<td>63 ± 10</td>
<td>47 ± 5</td>
<td>3 ± 3</td>
<td>9 (36)</td>
<td>1</td>
<td>2 (8)</td>
</tr>
<tr>
<td>p Value</td>
<td>NS</td>
<td>NS</td>
<td>&lt;0.001</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

Abbriviations and footnote symbols as in Tables I and II.

Atrial arrhythmias. Within each category the number of ectopic episodes was analyzed separately. Atrial arrhythmias were diagnosed using standard electrocardiographic criteria.⑨

Statistical analyses. Clinical data are presented as the mean ± SD. Clinical variables were compared between patient groups using the t test. Inter- and intragroup comparisons of the proportions of patients having a given arrhythmia and cumulative event rates of ectopic episodes in each category during the first and tenth day of infarction were based on a t test for unpaired and paired data as well as on McNemar's test, the chi square test (with Yates' correction), and a proportion test as appropriate. For small sample comparison, Mann-Whitney and Wilcoxon nonparametric tests were used. Stepwise multiple regression analysis and Pearson correlation were conducted to identify clinical variables (age, sex, RVEF, and LVEF) related to atrial rhythm disturbances. R-square and t values were reported to delineate the relative importance of each factor selected by the multiple regression analysis. Probability values less than 0.05 were considered statistically significant.

RESULTS

For the entire group of patients, the incidence of atrial ectopic activity was significantly higher during the first day of MI than on the tenth day (Table I). Comparing the two groups, there were no significant differences with respect to age, sex distribution, or LVEF (mean ± SD, 62 ± 10% for both groups). Of the 36 patients in group A, 33 (92%) had one or more APCs per hour, compared with 18 patients (69%) in group B (Table II). Atrial and supraventricular tachycardia were more often present in group A patients (16 of 36 [44%] versus 8 of 26 [31%]). However, this trend was not as significant as the one observed with respect to AF (7 of 36 [19%] versus 1 of 26 [4%]). Quantitative analysis revealed a mean hourly frequency of APCs of 13 ± 17 in group A patients compared with 6 ± 5 in group B patients (p = 0.05). In patients with complex atrial arrhythmias, arrhythmic episodes were recorded more frequently in group A patients (p = 0.02). Episodes of AF were single in two patients (one from each group) and multiple (two to four episodes) in the remaining group A patients. The prevalence of late rhythm disturbances (tenth day after MI) was comparable among group A and B subjects (Table III).

Stepwise multiple regression analysis revealed that the prevalence of atrial rhythm disturbances was unrelated to age, sex, or LVEF. After adjusting for the other selected variables, RVEF was independently related to the prevalence of APCs and AF (R square = 7% and 7.7%; significance of t = 0.02 and 0.02, respectively). A weaker nonsignificant association was observed between RV function and the prevalence of atrial and supraventricular tachycardia. The correlation between RV function and rhythm
disturbances was further explored using the Pearson correlation. Likewise, RVEF was significantly related to the prevalence of APCs ($p = 0.019$) and to the occurrence of AF ($p = 0.014$).

**DISCUSSION**

Atrial rhythm disturbances, occurring in about 20% to 45% of MI patients, usually disappear within a few days unless significant ventricular dysfunction persists or pericarditis occurs. In this study, the prevalence of atrial arrhythmias was investigated in a carefully preselected group of patients with first inferior wall MI. Stringent inclusion criteria ensured selection of patients with no other conditions known to increase susceptibility for rhythm disturbances or that could impose a further hemodynamic burden on the RV. The major observations were: (1) The incidence of atrial ectopic activity in patients with inferior wall MI is significantly higher during the first than on the tenth day of infarction. (2) Patients with RV dysfunction have an increased incidence of early atrial arrhythmias when compared with patients with an intact RV. (3) RVEF is independently related to the prevalence of APCs and AF. In accordance with previously reported data, our LVEF data for patients with inferior wall MI was comparable with the normal control values. The prevalence of RV dysfunction was also in close agreement with that in previous reports.

This study provides no information about the naturally occurring trigger responsible for the higher propensity toward atrial rhythm disturbances among patients with RV dysfunction. However, several precipitating factors alone or in combination may be important in accounting for this trend. These include atrial infarction, an increased right atrial pressure, atrial distension, and autonomic factors. Their etiologic role as an arrhythmogenic trigger and their contribution to the observed incidence of atrial arrhythmias have not yet been determined.

It has been suggested that AF early in the course of inferior wall MI is primarily related to left atrial ischemia or infarction, induced by a proximal occlusion of the circumflex coronary artery. However, in the setting of inferior wall MI complicated by RV infarction, a circumflex artery occlusion is unlikely to be a major ischemic cause of an increased prevalence of atrial ectopic activity, since the right coronary artery is almost always the culprit vessel. In addition, as the sinus node artery originates from the right coronary artery in 55% and from the AV nodal artery in almost 90% of cases, one could not ignore their potential role in the genesis of atrial arrhythmias during RV infarction. Bouts of tachycardia can be precipitated by sinus node ischemia and dysfunction, making AF, atrial ectopic activity, and junctional rhythms more likely, similar to sick sinus syndrome.

**Role of atrial infarction in genesis of atrial arrhythmias.** In most published studies of atrial infarction, particular attention has been given to the occurrence of atrial arrhythmias. Of note is the study by Soderstrom, in which 26 out of 47 patients with atrial infarction exhibited either paroxysmal or sustained AF. Consequently, one difficulty in truly attributing atrial rhythm disturbances to RV infarction stems largely from its unreported association with right atrial infarction. In postmortem studies, the highest reported incidence of atrial infarction among autopsy-verified cases of MI is 42%,. The hypothesis that right atrial infarction might be a major determinant of arrhythmogenesis in patients with RV infarction is gaining indirect support from the consistently higher reported rate of right atrial as opposed to left atrial infarction. Cushing et al. reported that out of 31 autopsy-proven cases of atrial infarction, 27 were right-sided. In an experimental model of RV infarction, necropsy findings of right atrial necrosis, varying from 40% to 95% of the atrial mass, were apparent in all examined hearts.

**Role of atrial pressure and enlargement in genesis of atrial arrhythmias.** Maintaining the contribution of atrial contraction to diastolic filling is particularly beneficial in patients with RV infarction, in whom diastolic filling is a crucial determinant of systolic function. However, the infarcted RV imposes increased preload and afterload on the right atrium, resulting in enhanced right atrial pressure and subsequent enlargement. Under these circumstances, it is likely that susceptibility to atrial rhythm disturbances is substantially aggravated, as demonstrated by Legrand et al., who noted a large RV infarct in nearly all patients who experienced an episode of AF. In addition, the study by Sugiura et al. provided an insight into the importance of right atrial pressure and its possible adverse arrhythmogenic effect. Discriminant analysis in this study revealed that the rise in right atrial pressure identified a group of patients who were much more likely to have AF. Another pertinent observation has emerged from the study of Sanfilippo et al. Once sinus rhythm is not maintained, atrial enlargement can develop as a consequence of atrial arrhythmias, contributing to a self-perpetuating anatomic-arrhythmogenic interrelated cycle. This vicious cycle would be expected to affect mainly the right atrium in cases of RV infarction.

**Clinical implications.** The main implication of this study is that patients with inferior wall MI would be expected to have a higher incidence of atrial ectopic
activity once RV dysfunction coexists. Whether the hemodynamic state and prognosis are adversely affected by the magnitude of difference in arrhythmic events is a question to be addressed in further studies.

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REFERENCES