PAROXYSMAL ATRIAL FIBRILLATION AND FLUTTER ASSOCIATED WITH ACUTE MYOCARDIAL INFARCTION: HEMODYNAMIC EVALUATION IN RELATION TO THE DEVELOPMENT OF ARRHYTHMIAS AND PROGNOSIS

YOSHINORI KOBAYASHI, M.D., TAKAO KATOH, M.D., TERUO TAKANO, M.D. AND HIROKAZU HAYAKAWA, M.D.

The hemodynamic background associated with the occurrence of paroxysmal atrial fibrillation and flutter (PAF), in patient with acute myocardial infarction (AMI) were evaluated. Sixty-seven of 381 consecutive AMI patients (17.6%) were noted to have PAF in the acute phase of infarction. These 67 patients with PAF (group 1) were compared with 60 randomly selected patients without PAF (group 2). The hospital mortality rate was 25.4% in group 1, and 11.7% in group 2 (p<0.01). The hemodynamic variables measured before the onset of PAF in group 1, showed significantly more unfavorable values than those in group 2, which were measured at the time of admission. The 67 patients in group 1 were divided into 50 patients who survived (group S) and 17 patients who died in the hospital (group D). The hemodynamic status in group D demonstrated significantly larger deterioration before the onset of PAF than in group S. Hemodynamic variables were compared before and during PAF in groups D and S, cardiac index (CI) decreased significantly, and stroke index (SI) decreased by 46% in group D, with no decrease in CI and less decrease in SI (28. p<0.05) in group S. In conclusion, not only the occurrence of PAF, but the prognosis of patients with PAF is dependent on the severity of hemodynamic disturbance imposed by AMI. Atrial contribution to ventricular filling has great importance in the maintenance of the cardiac output in this patient population. (Jpn Circ J 1992; 56: 1-11)

It has been previously documented that, in acute myocardial infarction (AMI), paroxysmal atrial fibrillation (PAF) occurs in 5 to 17%,4,7,12 and atrial flutter in 1 to 5%,2,4,8,10 of patients, respectively. These studies also noted increased mortality in the patients with atrial arrhythmias. Prior studies3,5-7,9,11 have emphasized that the most important underlying mechanism producing atrial arrhythmias is the pump failure associated with severe myocardial infarction. However, hemodynamic data is limited. The first aim of present study was to confirm these mechanistic theories by the comparison of hemodynamic variables between the patients with and without PAF.

It has been suggested that PAF in association with AMI carves a poor prognosis. The second aim of this study was to characterize the clinical features of PAF in survivors and
**TABLE I INCIDENCE OF PAF ACCORDING TO KILLIP CLASSIFICATION**

<table>
<thead>
<tr>
<th></th>
<th>No. with PAF</th>
<th>No. without PAF</th>
<th>Total No.</th>
<th>Incidence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Killip I</td>
<td>36</td>
<td>236</td>
<td>272</td>
<td>13.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>19</td>
<td>45</td>
<td>64</td>
<td>29.7</td>
</tr>
<tr>
<td>III</td>
<td>4</td>
<td>13</td>
<td>17</td>
<td>23.5</td>
</tr>
<tr>
<td>IV</td>
<td>8</td>
<td>20</td>
<td>28</td>
<td>28.6</td>
</tr>
<tr>
<td>Total</td>
<td>67</td>
<td>314</td>
<td>381</td>
<td>17.6</td>
</tr>
</tbody>
</table>

※1 = p < 0.01, ※2 = p < 0.05 (chi square analysis)

non-survivors of AMI.

It has been reported that the mechanism producing arrhythmia is responsible for the high mortality rate in patients with PAF, and not the arrhythmia itself. Stated differently, pump failure imposed by extensive myocardial infarction is the common mechanism underlying both the appearance of PAF, and the poor prognosis of patients.

The third aim was therefore to show the differences in hemodynamic variables and other clinical parameters of severity of infarction between surviving and non-surviving patients with PAF.

Recent hemodynamic evaluations using various methods have demonstrated that the atrial contribution to ventricular filling is of greater significance in patients with heart disease such as ischemic heart disease, especially with congestive heart failure. It can be anticipated that the development of PAF has greater unfavorable effects on the hemodynamics in the critical patient than the non-critical patients. The last aim of this study was to compare the negative hemodynamic effects contributed by the arrhythmia itself, between survivors and non-survivors.

**MATERIALS AND METHOD**

During the years 1984-'87, 389 patients were hospitalized for AMI in the coronary care unit (CCU) of Nippon Medical School. Eight patients already had preexisting chronic atrial fibrillation at the time of admission, and were excluded from this study. Of the other 381 patients, 308 were men and 73 were women.

AMI was diagnosed if patients satisfied at least 2 out of following 3 criteria: 1) typical clinical symptoms. 2) ST-T wave changes with or without new Q wave in ECG. 3) diagnostic rise in the serum enzyme level of creatine kinase (CK) and CK-MB.

All patients were monitored by ECG continuously during their stay in the CCU. Atrial fibrillaion was defined electrocardiographically by fine irregular atrial waves (f waves) associated with absolutely irregular ventricular response; atrial flutter by regular atrial activity (F waves) with a F-F interval of approximately 200 msec associated with various patterns of atrioventricular conduction.

In 344 (90%) of patients, Swan-Ganz catheter was inserted at the time of admission; 37 (10%) did not undergo hemodynamic evaluation as they died immediately after the admission, or could not undergo catheterization due to technical difficulties. Atrial pressure was also monitored from a radial artery during the first 2-5 days in the CCU.

Hemodynamic variables including cardiac output (CO), pulmonary arterial pressure (PAP), pulmonary capillary wedge pressure (PCWP), central venous pressure (CVP), systolic and diastolic arterial pressure (BPs, Bpd), and heart rate (HR) were measured at the time of admission and at 8 AM every morning during their stay in the CCU.

Cardiac index (CI) and stroke volume index (SI) were calculated by the following equations:

Cardiac index = cardiac output / body surface area

Stroke index = cardiac index / heart rate

**Statistical analysis**

Results are reported as mean ± standard deviation, the chi square test and Student's t test were used for statistical analysis. P values lower than 0.05 were considered significant.

**RESULTS**

The incidence and clinical features of PAF in all patients

In 67 patients (17.6%) [group 1] out of 381 AMI patients, one or more episodes of PAF were observed in the acute phase of myocardial infarction: 51 patients had atrial fibrilla-
TABLE II CLINICAL FINDINGS — PATIENTS WITH PAF VS WITHOUT PAF —

<table>
<thead>
<tr>
<th>Clinical Findings</th>
<th>PAF (+)</th>
<th>PAF (-)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group 1 (N.)</td>
<td>Group 2 (N.)</td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>60M, 7F</td>
<td>49M, 11F</td>
<td>N.S.</td>
</tr>
<tr>
<td>Age</td>
<td>67.2±11.4</td>
<td>60.3±11.9</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>Infarction site</td>
<td>A; 37, I; 23</td>
<td>A; 33, I; 22</td>
<td>N.S.</td>
</tr>
<tr>
<td></td>
<td>NTM; 7*1</td>
<td>NTM; 5</td>
<td></td>
</tr>
<tr>
<td>Peak CK (I.U.)</td>
<td>3872±2652 (41)</td>
<td>3315±2466 (44)</td>
<td>N.S.</td>
</tr>
<tr>
<td>Peak CK-MB (I.U.)</td>
<td>383±224 (41)</td>
<td>340±250 (44)</td>
<td>N.S.</td>
</tr>
<tr>
<td>EF*2 (RI ventriculography) (%)</td>
<td>39.1±15.9 (26)</td>
<td>46.8±9.2 (25)</td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td>No. of vessels*3</td>
<td>1.6±0.8 (15)</td>
<td>1.4±0.9 (25)</td>
<td>N.S.</td>
</tr>
<tr>
<td>Scoring of asynery*4</td>
<td>6.1±1.8 (12)</td>
<td>4.1±2.2 (23)</td>
<td>p&lt;0.01</td>
</tr>
</tbody>
</table>

(Other complications)

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (N.)</th>
<th>Group 2 (N.)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiogenic shock</td>
<td>8 (12%)</td>
<td>3 (5%)</td>
<td>N.S.</td>
</tr>
<tr>
<td>Cardiac rupture</td>
<td>5 (7%)</td>
<td>1 (2%)</td>
<td>N.S.</td>
</tr>
<tr>
<td>PMD*5</td>
<td>4 (6%)</td>
<td>1 (2%)</td>
<td>N.S.</td>
</tr>
<tr>
<td>Pericarditis</td>
<td>8 (12%)</td>
<td>3 (5%)</td>
<td>N.S.</td>
</tr>
<tr>
<td>LV aneurysm</td>
<td>4 (6%)</td>
<td>0</td>
<td>N.S.</td>
</tr>
<tr>
<td>VT*6</td>
<td>30 (45%)</td>
<td>14 (23%)</td>
<td>p&lt;0.05</td>
</tr>
</tbody>
</table>

| Mortality rate | 17/67 (25.4%) | 7/60 (11.7%) | p<0.05 |

*1 A = anterior, I = inferior, NTM = nontransmural
*2 = LV ejection fraction, *3 = No. of vessels of significant stenosis in CAG, *4 = AHA scoring of asynery of LV in LVG,
*5 = papillary muscle dysfunction, *6 = ventricular tachycardia.

The incidence of PAF according to Killip classification

There were significant differences in PAF incidence between patients of Killip class I and class II, and class I and class IV (p<0.01, p<0.05 respectively, Table I).

The comparison of clinical findings and hemodynamic variables between the patients with and without PAF

Sixty patients [group 2] were selected from the serial 314 patients in whom PAF had not developed in acute phase of myocardial infarction by picking up every 5th patient in the series for comparison of various clinical findings and hemodynamic variables with those of the patients with PAF (group 1).

Table II shows the clinical findings in patients of group 1 (PAF(+)) as compared to patients of group 2 (PAF (-)). The mean age in group 1 was 67.2±11.4 years, and was significantly higher than that in group 2 (60.3±11.9 years). The frequency of different sites of infarction was similar between both groups. The peak values of CK and CK-MB appeared to be higher in group 1 than in group 2, but there was no statistically significant difference between the 2 groups.

Left ventricular (LV) ejection fraction, assessed by radionuclide ventriculography (LVG), was lower (p<0.05), and scoring of
asynergy of wall motion in LVG was higher (p<0.01) in group 1 than in group 2.

The incidence of other associated complications including cardiogenic shock, cardiac rupture, papillary muscle dysfunction and LV aneurysm, tended to be higher in group 1 than group 2. With regard to the other complicated arrhythmias, only ventricular tachycardia was significantly more frequent in group 1 than in group 2 (p<0.05). The mortality rate was significantly higher in group 1 than in group 2 (p<0.05). The hemodynamic variables are compared between the patients of group 1 and group 2 in Fig. 1. These variables were measured during sinus rhythm, within 24h before the onset of PAF in group 1, and at the time of admission prior to various therapeutic interventions in group 2. There were significant differences in PAP (mean), PCWP, CVP, and BP (mean) between two groups (p<0.05, p<0.001, p<0.05 and p<0.01, respectively).

The comparison of clinical features of PAF between survivors and non-survivors

The 67 patients of group 1 were divided into 50 patients [group S] who survived at the end of the hospital course and 17 patients [group D] who died in hospital.

The time of the onset of the first episode of PAF was significantly later in group D (non-survivors) than group S (survivors) (5.1±6.9 vs 3.0±1.8 postinfarction day, p<0.05) (Fig. 2). In addition, there were 3 patients in group D but no patient in group S, in whom PAF had occurred after the second postinfarction week.

The ventricular response immediately after the onset of atrial fibrillation was significantly lower in group D than group S (112±25 vs 134±25 beats per min, p<0.05).
Ten of the 17 patients (59%) in group D had recurrent episodes of PAF, in contrast to 15 of the 50 patients (30%) in group S (p<0.05).

There was no significant difference in the duration of PAF between two groups (group D: 11.4±20.8 vs group S: 13.5±22.8h).

**The comparison of clinical findings and hemodynamic variables between survivors and non-survivors**

Table III shows the various clinical findings of group D (non-survivors) as compared to group S (survivors). We found that the patients in group D were significantly older than those in group S. The values of peak CK and CK-MB tended to be higher in group D than in group S, although there was no statistically significant difference. Cardiogenic shock occurred in 8 patients and cardiac rupture in 4 patients in group D. In these 12 patients, these complications were the main causes of death. Of the other 5 non-survivors, 3 patients died of reinfarction, and 2 patients died suddenly during the post-CCU period.

In Fig. 3, the hemodynamic variables measured within 24h before the onset of PAF are compared between group S and group D. There were significant differences in CVP,

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**TABLE III CLINICAL FINDINGS IN PATIENTS WITH PAF — SURVIVORS VS NON-SURVIVORS —**

<table>
<thead>
<tr>
<th>Clinical Findings</th>
<th>non-survivors (group D) N=17</th>
<th>survivors (group S) N=50</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>12M, 5F</td>
<td>48M, 2F</td>
<td>N.S.</td>
</tr>
<tr>
<td>Age</td>
<td>72.2±9.1</td>
<td>65.5±11.7</td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td>Infarction site</td>
<td>A; 10, I; 7</td>
<td>A; 27, I; 16</td>
<td>N.S.</td>
</tr>
<tr>
<td>Infarction site</td>
<td>NTM; 0*1</td>
<td>NTM; 7</td>
<td></td>
</tr>
<tr>
<td>Peak CK (I.U.)</td>
<td>5123±3302</td>
<td>3550±2276</td>
<td>N.S.</td>
</tr>
<tr>
<td>Peak CK-MB (I.U.)</td>
<td>441±202</td>
<td>374±244</td>
<td>N.S.</td>
</tr>
</tbody>
</table>

(Other complications)
- Cardiogenic shock 8 (47%) 0 (0%) p<0.005
- Cardiac rupture 4 (24%) 1 (2%) p<0.005
- PMD*2 1 (6%) 3 (6%) N.S.
- LV aneurysm 1 (6%) 3 (6%) N.S.
- Pericarditis 2 (12%) 6 (12%) N.S.
- VT*3 12 (71%) 18 (36%) p<0.05

*1 = A = anterior, I = inferior, NTM = nontransmural
*2 = papillary muscle dysfunction, *3 = ventricular tachycardia.

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BP (mean), and CI (p<0.01) between two groups. Thus, the hemodynamic status prior to the onset of PAF was poorer in non-survivors than in survivors.

In 9 patients of group S and 6 patients of group D, the hemodynamic variables were also measured within half an hour after the onset of fibrillation prior to therapeutic interventions. The changes in hemodynamic variables associated with the development of this arrhythmia were compared between two groups. PCWP could not be measured in 2 patients of group S and 1 patient in group D, because of dislocation of catheter tip.

In group S there was no significant difference in all variables except stroke index (SI) before and after the onset of the arrhythmia (Fig. 4). SI decreased by 28% after the development of this arrhythmia in group S. In contrast, in group D, CI decreased significantly, from 2.52±0.96 l/min/m² before the arrhythmia to 2.03±0.97 l/min/m² during that (Fig. 5), and SI decreased by 46% in group D. The ratio of the decrease in SI by this arrhythmia was significantly greater in group D than group S (p<0.05).

DISCUSSION

The incidence of atrial fibrillation of 16.5% in the present series of 381 patients admitted with AMI corresponds to the upper range of previous reports (5–17%). The high incidence of this arrhythmia in our study was presumably due to the recent advent of continuous ECG monitoring systems and the protracted stay of critical cases in our CCU, which was often as long as 2 weeks.

Atrial flutter is a rare atrial arrhythmia.
associated with acute myocardial infarction; we found an incidence of 4.1%, which was within the range reported in previous studies\textsuperscript{2,4,8,10}.

*The underlying mechanisms of the development of PAF*

DeSanctis et al\textsuperscript{4} suggested that the major factors responsible for the occurrence of supraventricular arrhythmias during acute myocardial infarction are: 1) anatomic factors; compromise of the blood supply to atrial structures (the sinus node, the AV node, or the atrial musculature), and pericarditis, 2) autonomic factors: enhanced vagal tone accompanying inferoposterior infarction and sympathomimetic reaction in patients with severe myocardial infarction, 3) hemodynamic factors: “pump failure” with left atrial hypertension accompanied with reduction of coronary perfusion, 4) iatrogenic factors: digitalis, anti-arrhythmic agents and sympathomimetic agents.

Although Liberthon et al\textsuperscript{8} James et al\textsuperscript{13} and Löfmark et al\textsuperscript{10} indicated that the mechanisms of atrial tachyarrhythmias were the pericarditis, the compromise of the blood supply to atrium, and atrial conduction disturbance respectively, most of other previous studies\textsuperscript{3,5–7,9,11} emphasized that the most important underlying mechanism of development of atrial arrhythmias was pump failure associated with severe myocardial infarction.

Hunt et al\textsuperscript{9} demonstrated that radiological abnormalities (pulmonary congestion, or edema) and isolated hypotension were more commonly found in patients with atrial fibrillation. In our study, the incidence of PAF according to Killip classification also indicated that the patients with complicating congestive heart failure, pulmonary edema, and cardiogenic shock were likely to have PAF.

Our clinical data such as peak values of CK and CK-MB, ejection fraction, scoring of LV asynergy and other associated complications suggested that the development of PAF was closely related to extensive myocardial infarction.

Pericarditis appeared to be more frequent in patients with PAF than without PAF, suggesting that the pericarditis might be one of the causes of PAF in the course of acute myocardial infarction, as demonstrated by Liberthon et al\textsuperscript{8}.

The most widely accepted observation is
that atrial tachyarrhythmias are mainly caused by left ventricular failure, but there is only one report\textsuperscript{11} in which the hemodynamic status of a small number of patients with AMI complicated by atrial fibrillation was evaluated. We could confirm the validity of these previous mechanistic theories by comparing hemodynamic data between a significant number of patients with and without PAF.

Sugiura et al\textsuperscript{11} demonstrated that no significant differences were observed in the hemodynamic measurements at admission between the patients with and without atrial fibrillation, but the pulmonary capillary wedge pressure and the right atrial pressure increased significantly before the onset of atrial fibrillation. We observed such deterioration of hemodynamic variables in the patients with “late onset” PAF, as will be mentioned in the following section.

The clinical features of PAF associated with poor prognosis

Klass et al\textsuperscript{3} demonstrated that in 4 “late onset” cases of atrial fibrillation, 3 cases died and that late onset atrial fibrillation is associated with a poor clinical course and prognosis. We also found that all of our 3 patients in whom the first episode of PAF occurred after the second postinfarction week died. In all of them, moderate congestive heart failure was already present at the time of admission and it was further aggravated during their hospital course accompanied by the development of PAF. Thus “late onset” PAF is to be considered one of signs of deteriorating hemodynamic condition in patients with AMI. On the other hand, it is considered that the factors responsible for the occurrence of “early onset” PAF are not only hemodynamic changes but also enhanced vagal tone and/or sympathomimetic reaction during the acute phase of infarction.

While Klass et al\textsuperscript{3} and others\textsuperscript{5} reported that an increased mortality was associated with an uncontrollable ventricular response, Julian et al\textsuperscript{2} and others\textsuperscript{7} did not verify these findings and even indicated that a slower rate may be more harmful. We could also demonstrate that ventricular response was significantly lower in non-survivors than in survivors.

In our study, the recurrence of PAF was found to be associated with an increase in

\textit{Japanese Circulation Journal Vol.36, January 1992}
the mortality rate. In contrast, some previous authors reported that repetitive episodes of atrial fibrillation were not associated with an increased mortality. However, if atrial tachyarrhythmia may be a sign of the protracted hemodynamic disturbance in patients with AMI, these arrhythmias may be recurrent.

**The underlying mechanism of the poor prognosis in the patients with PAF**

Our results reviewing clinical findings including age, peak value of CK and CK-MB, and the frequency of other complications (Table III) for non-survivors as compared to survivors suggested that not only the occurrence of PAF, but also the prognosis in the patients with PAF depends on the severity of underlying myocardial infarction. In other words, the poor clinical status of patients with extensive myocardial infarction is closely related to the occurrence of PAF. In the patients with PAF, more critically ill patients seem to deteriorate, resulting in a greater frequency of lethal complications. In the comparison of hemodynamic variables before the onset of PAF between survivors and non-survivors (Fig. 4), CI and BP (mean) were significantly lower and CVP was higher, and other hemodynamic variables tended to be less favorable in non-survivors than in survivors. These hemodynamic findings support the concept that the prognosis depends on the severity of infarction among the patients with PAF. In our series of patients, most of the non-survivors died of complications such as cardiogenic shock and cardiac rupture during the hospital stay. Since these differences were observed between survivors and non-survivors, we conclude that the mechanism producing arrhythmia, and not arrhythmia itself responsible for the high mortality in patients with AMI and PAF, as previous investigators indicated.

**The hemodynamic aggravation caused by the development of PAF**

It is well-known that the most important mechanism aggravating the hemodynamic status of patients with atrial fibrillation is the reduced atrial contribution to ventricular filling due to the lack of atrial contractions. Previous studies dealing with the contribution of atrial contraction in man have been performed in the following situations: 1) conversion of atrial fibrillation to regular sinus rhythm with D.C. shock; during atrial pacing and ventricular pacing using either single pacing or synchronous atrioventricular pacing. From these reports, the overall increase of stroke index by effective atrial contraction, the ratio of atrial contribution to ventricular filling (atrial contribution ratio), is approximately 20% both in patients with and without heart disease.

Bencimol et al and Killip et al suggested that in patients with heart disease, the contribution of atrial systole appeared to be of greater significance compared with patients without heart disease. In addition, Hammermeister et al pointed out that in their evaluation of the rate and amount of left ventricular filling by means of single plane cineangiography, the atrial contribution ratio was greater in the diseased heart than in the normal subject (38% vs 16%, coronary heart disease: 33%). Furthermore, an investigation of the atrial contribution by means of ECG-gated radionuclide angiocardiography also showed that the atrial contribution ratio in patients with old myocardial infarction (32.9%) and with dilated cardiomyopathy (43.0%) was significantly higher than normal subjects (15.4%), and that there was a significant negative correlation (r = −0.76) between LV ejection fraction and atrial contribution ratio in patients with ejection fraction less than 50%.

These reports indicated that the atrial contribution to ventricular filling is very important in patients with diminished ventricular function. The underlying mechanism of this phenomenon was suggested by Walsh et al to be as follows: although the normal left ventricle utilizes compensatory mechanisms such as enhancement of inotropic state and chamber dilatation to maintain cardiac output, these compensatory reserves are less readily available in the setting of reduced left ventricular performance, and therefore the loss of atrial systole may be less readily tolerated.

Decrease in stroke index associated with the development of atrial fibrillation demonstrated that the atrial contribution ratio was 28% in survivors, 46% in non-survivors, and the overall ratio was 35.2%. This corres-
ponds to the values of atrial contribution ratio in patients with heart disease, as demonstrated previously. On the basis of our results, it is considered that non-survivors had more severe heart failure as confirmed by the hemodynamic variables and clinical findings. In such situations the compensatory mechanisms of left ventricle were less available, resulting in the greater decrease in the stroke volume than survivors.

**Therapeutic implications**

On the basis of our results, we conclude that most important mechanism contributing to the development of PAF in the acute phase of myocardial infarction is pump failure. The severity of hemodynamic disturbance strongly influences the prognosis of patients. Although PAF itself could not be shown to be the leading cause of death in any critically ill patient, it is reasonable to assume that PAF might play an indirect, unfavorable role on the clinical course of these patients. This appears to be so, as the deterioration in hemodynamic status after the development of PAF was more severe in non-survivors than in survivors. These results led us to believe that, although treatment must be directed toward the mechanism producing arrhythmia (mostly pump failure) in patients with PAF, treatment must be simultaneously directed at terminating the arrhythmia. This is because the deterioration in hemodynamics imposed by the development of the arrhythmia cannot be ignored, especially in critical cases in whom the atrial contribution to ventricular filling is of great importance. From our empirical observations of the treatment of critical patients, antiarrhythmic agents possessing negative inotropic effects could not be utilized to terminate this arrhythmia. Furthermore there were great difficulties in maintaining sinus rhythm, even if the arrhythmia could be terminated by electrical cardioversion. We also observed that digitalis was the most suitable drug to manage this arrhythmia in terms of its capability to control the ventricular response and its positive inotropic effect, but it did not produce either immediate effects or conversion to sinus rhythm in patients with refractory pump failure. More advanced therapeutic methods are required.  

**Acknowledgment**

The authors express their gratitude to William J. Mandel, M.D. of Cedars-Sinai Medical Center, Los Angeles, CA, USA, for reading the manuscript.

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