Early atrial fibrillation during evolving myocardial infarction: a consequence of impaired left atrial perfusion


ABSTRACT Seven of 214 patients (3%) with acute myocardial infarction (120 inferior and 94 anterior) developed atrial fibrillation within 3 hr of the onset of chest pain. All seven patients had an inferior infarction and in all seven the left circumflex artery was occluded proximal to the origin of its left atrial circumflex branch. In five patients this occlusion was acute and was the cause of inferior infarction and in the remaining two patients the occlusion was old and the inferior infarction was due to an acute occlusion of the right coronary artery that also supplied extensive collaterals to the previously occluded left circumflex artery. All seven patients also had impaired perfusion to the atrioventricular nodal artery, as evidenced by total occlusion proximal to its origin or by stenosis proximal to its origin associated with second- or third-degree atrioventricular block. In contrast, early atrial fibrillation did not occur in any of the 18 patients with inferior myocardial infarction due to acute occlusion of the distal left circumflex artery or in any of the five patients with inferior infarction due to acute occlusion of the proximal left circumflex artery if perfusion to the atrioventricular nodal artery was not impaired. Early atrial fibrillation did not occur in any of the 90 patients with inferior infarction due to acute occlusion of the right coronary artery, including 12 patients with occlusion proximal to the sinus nodal artery, but without coexistent occlusion of the left circumflex artery. Our data indicate that early atrial fibrillation in acute myocardial infarction occurs when there is coexistent occlusion of the left circumflex artery proximal to the origin of its left atrial circumflex branch and impaired perfusion of the atrioventricular nodal artery. Since these two arteries both contribute to left atrial perfusion, our data suggest that acute left atrial ischemia is the pathophysiologic mechanism of early atrial fibrillation. Circulation 75, No. 1, 146-150, 1987.

Atrial Fibrillation complicates acute myocardial infarction in about 20% of patients. It usually occurs later than 24 hr after the onset of infarction as a consequence of either pericarditis or heart failure. In contrast, atrial fibrillation during the early hours of myocardial infarction is rare and its pathogenesis is poorly understood. The purpose of this study was to investigate the pathogenesis of atrial fibrillation in the early hours of an acute myocardial infarction in a population of patients who were admitted within 3 hr of the onset of acute myocardial infarction and in whom there was subsequent angiographic documentation of coronary anatomy.

Methods

Patient population. The patient population was drawn from 244 consecutive patients with acute myocardial infarction who were enrolled in our ongoing studies of intravenous or intracoronal streptokinase in acute myocardial infarction. Patients were included if they satisfied the following criteria: (1) chest pain of less than 3 hr duration at the time of evaluation by our team, (2) ST segment elevation in two or more leads, (3) persistence of both the chest pain and the electrocardiographic changes after sublingual nitroglycerin, (4) absence of contraindications to thrombolytic and anticoagulant therapy, and (5) informed consent from the patient after approval by the patient's physician.

Twenty-two patients who received intravenous streptokinase but did not undergo coronary angiography were excluded from this study. None of these 22 patients developed atrial fibrillation. An additional eight patients who developed atrial fibrillation were also excluded for the following reasons: four had documented chronic atrial fibrillation, two developed atrial fi-
brillation immediately after direct current countershock for ventricular fibrillation \[9, 30\] and two developed transient atrial fibrillation during right atrial pacing \[11, 12\].

The study population consisted of the remaining 214 patients, 167 men and 47 women, who were 59 ± 11 years old (range 31 to 80). Seventy-five patients received intracoronary and 139 received intravenous streptokinase.

Definition of early atrial fibrillation. Atrial fibrillation was defined as early if it commenced within 3 hr of the onset of chest pain. Of note, no patient in this study developed atrial fibrillation between 3 and 24 hr after the onset of chest pain.

Coronary anatomy. Coronary angiography was performed within 2 hr of admission in the 75 patients who received intracoronary streptokinase and 2 to 7 days after admission in the 139 patients who received intravenous streptokinase.

Artery of infarction. The artery of infarction and the site of occlusion were obvious in 170 patients: the 75 patients who received intracoronary streptokinase and the 95 patients who received intravenous streptokinase and either had an anterior infarction with severe stenosis or occlusion of the left anterior descending artery (64 patients) or an inferior infarction with stenosis or occlusion of the right or the left circumflex coronary artery, but not both (31 patients). In the remaining 44 patients with inferior infarction who received intravenous streptokinase and had stenoses in both the right coronary and the left circumflex artery, the artery of infarction and the site of occlusion were identified by (1) angiographic criteria of an ulcerated atheromatous plaque, i.e., indistinct luminal margins and subintimal ulceration of the coronary artery after thrombolysis \[13, 14\] and (2) the regional pattern of left ventricular dysfunction on contrast and radionuclide ventriculograms and by the regional distribution of decreased perfusion assessed by pretestreptokinase \[20\] TI scintigraphy.

Assessment of atrial blood supply. Coronary angiograms were examined to identify the following potential sources of atrial blood supply:

1. The sinus nodal artery that arises from the proximal right coronary artery in about 60% of patients and from the proximal left circumflex artery in 40% of patients and supplies most of the right atrium, including the sinoatrial node, and also contributes to the blood supply of the left atrium \[15, 16\].

2. The left atrial circumflex artery that arises from the proximal left circumflex artery and supplies most of the left atrium \[15, 17\].

3. The atrioventricular nodal artery that originates from the right coronary artery in 90% of patients and from the left circumflex artery in 10% of patients and supplies the atrioventricular node and also the left atrium directly and/or by Anastomoses with other left atrial branches \[15, 16\].

Results

Early atrial fibrillation. Only seven of the 214 patients (3%) developed atrial fibrillation within 3 hr of the onset of chest pain. Two patients were already in atrial fibrillation when first seen by the paramedical officers and five patients were in sinus rhythm with second- or third-degree atrioventricular block on admission and subsequently developed early atrial fibrillation.

Early atrial fibrillation and the location of infarction. All seven patients who developed early atrial fibrillation had acute inferior myocardial infarction. None of the 94 patients with acute anterior infarction developed early atrial fibrillation. Therefore, this analysis is limited to the 120 patients with acute inferior myocardial infarction. The clinical data on the seven patients who developed early atrial fibrillation are summarized in table 1.

Early atrial fibrillation and the coronary anatomy

Early atrial fibrillation and acute occlusion of the left circumflex coronary artery (table 2). In 28 of the 120 patients the inferior infarction was due to acute occlusion of the left circumflex artery. In 10 of these 28 patients the occlusion was proximal to the origin of the left atrial circumflex branch and five of these 10 patients developed early atrial fibrillation. All five of these patients also had impaired perfusion of the atrioventricular nodal artery, as evidenced by either total occlusion proximal to its origin or severe stenosis associated with second- or third-degree atrioventricular block. None of the five patients with acute occlusion of the left circumflex artery proximal to the left atrial circumflex branch but without impaired perfusion of the atrioventricular nodal artery and none of the 18 patients with acute occlusion distal to the origin of the left atrial circumflex artery.

### TABLE 1

Clinical and angiographic data on patients with early atrial fibrillation

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yr)/sex</th>
<th>Infarct artery</th>
<th>Compromised perfusion to</th>
<th>Precipitating factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>42/M</td>
<td>LCX</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
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</tr>
<tr>
<td>3</td>
<td>62/M</td>
<td>LCX</td>
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<td>Yes</td>
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<tr>
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<td>Yes</td>
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<td>LCX</td>
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</tr>
<tr>
<td>6</td>
<td>70/M</td>
<td>RCA*</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>7</td>
<td>71/M</td>
<td>RCA*</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

AF = atrial fibrillation; AVB = atrioventricular block; AVN = atrioventricular nodal; BP = blood pressure; LACX = left atrial circumflex branch; NE = norepinephrine; NTG = nitroglycerin; RCA = right coronary artery; SNA = sinus nodal artery; SR = sinus rhythm.

*Old complete occlusion of the left circumflex artery proximal to the origin of the left atrial circumflex branch.
branch developed early atrial fibrillation, even though five of these 18 patients had impaired perfusion of the atrioventricular nodal artery.

**Early atrial fibrillation and acute occlusion of the right coronary artery (table 3).** Only two of the 92 patients in whom the inferior infarction was due to acute occlusion of the right coronary artery developed early atrial fibrillation and these were the only two patients with acute right coronary artery occlusion in whom there was a coexistent old occlusion of the left circumflex artery proximal to the origin of its left atrial circumflex branch. Of interest, postreperfusion angiography in these two patients demonstrated filling of the occluded left circumflex artery from the right coronary artery. In both these patients, the atrioventricular nodal artery arose from the right coronary artery such that there was impaired perfusion to both the left atrial circumflex branch and the atrioventricular nodal artery.

None of the 90 patients with inferior infarction due to acute occlusion of the right coronary artery but without compromised perfusion to the left atrial circumflex branch developed early atrial fibrillation, even though 84 of them had impaired perfusion to the atrioventricular nodal artery (three with second-degree and 11 with third-degree atrioventricular block). Of note, 12 of these 84 patients had acute occlusion proximal to the origins of both the sinus nodal artery and the atrioventricular node artery. In the remaining six patients neither the left atrial circumflex branch nor the atrioventricular nodal artery were compromised.

**Potential precipitating factors of early atrial fibrillation.** Three patients who were initially in sinus rhythm with atrioventricular block developed atrial fibrillation during the course of intravenous administration of dopamine and/or norepinephrine given for hypotension (table 1). Early atrial fibrillation did not occur during intravenous administration of catecholamines in any of the 13 patients with anterior infarction or in any of the 21 patients with inferior infarction but without coexis-

<table>
<thead>
<tr>
<th>Compromised perfusion to</th>
<th>No. of patients who developed early atrial fibrillation</th>
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</thead>
<tbody>
<tr>
<td>LACx</td>
<td>AVN artery</td>
</tr>
<tr>
<td>Yes</td>
<td>Yes</td>
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<td>No</td>
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**TABLE 2**
Relationship of early atrial fibrillation to compromised perfusion of the left atrial circumflex (LACx) and atrioventricular nodal (AVN) arteries in patients with acute occlusion of the left circumflex coronary artery

**TABLE 3**
Relationship of early atrial fibrillation to compromised perfusion of the left atrial circumflex (LACx) and the atrioventricular nodal (AVN) arteries in patients with acute occlusion of the right coronary artery

<table>
<thead>
<tr>
<th>Compromised perfusion to</th>
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<tr>
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fibrillation persisted for 16 hr after the onset of chest pain.

Discussion

Our study confirms previous reports\textsuperscript{6-8} that atrial fibrillation is a rare event during the early hours of an evolving acute myocardial infarction. The rarity of early atrial fibrillation is consistent with our finding that its development requires both occlusion of the proximal left circumflex artery and total or near-total occlusion proximal to the origin of the atrioventricular nodal artery. Acute occlusion of the left circumflex artery occurs in only about 15% of patients with myocardial infarction and occlusion proximal to the left atrial circumflex branch obviously occurs even less frequently. Since the atrioventricular nodal artery originates from the right coronary artery in 90% of patients and from the circumflex coronary artery in only 10% of patients, early onset atrial fibrillation will most often require coexistent total or near-total occlusion of both the right and the proximal circumflex coronary arteries. This was the case in six of the seven patients in our study; the seventh patient had proximal occlusion of a dominant left circumflex artery that gave origin to the atrioventricular nodal artery.

Our conclusions appear to contradict those of James,\textsuperscript{19} who reported that atrial fibrillation in acute myocardial infarction was caused by coexistent occlusion of the sinus nodal artery and the atrioventricular nodal artery. However, review of the anatomic findings in the nine patients who developed atrial fibrillation in James' study reveals that eight of them had occlusion of the left circumflex artery either directly or as a consequence of left main coronary artery occlusion. The ninth patient had hemochromatosis, which may be an alternative explanation for the development of atrial fibrillation.\textsuperscript{20}

Our observation that early atrial fibrillation was accompanied by impaired perfusion to the atrioventricular nodal artery is consistent with James' finding\textsuperscript{19, 21} of an association between atrioventricular conduction delay and occurrence of atrial arrhythmias in acute myocardial infarction. Whether this reflects an electrophysiologic role for atrioventricular conduction delay in the genesis of early atrial fibrillation or whether atrioventricular block is simply a marker of compromised left atrial perfusion caused by obstruction of the atrioventricular nodal artery is not known. However, the finding that atrioventricular block alone or in combination with compromised perfusion to the sinus nodal artery did not result in early atrial fibrillation, even during infusion of catecholamines, unless there was also compromised perfusion of the left atrial circumflex branch suggests that atrioventricular block per se does not have a primary role in the development of early atrial fibrillation.

Of note, since the obstruction that impaired perfusion to the atrioventricular nodal artery was also always proximal to the origin of the posterior descending artery, and since according to Kugel\textsuperscript{18} this artery may contribute to the left atrial blood supply, it is not possible to rule out that compromised perfusion of the posterior descending artery also contributed to the development of early atrial fibrillation.

Our anatomic findings suggest that acute left atrial ischemia is the primary cause of early atrial fibrillation. This conclusion is supported by our observations that (1) early atrial fibrillation reverted to sinus rhythm in close temporal relation to the resolution of myocardial ischemia, and (2) administration of catecholamines, known to accentuate myocardial ischemia,\textsuperscript{22} preceded early atrial fibrillation in three patients with compromised left atrial perfusion. However, a direct effect of catecholamine administration on atrial conduction\textsuperscript{21} in the genesis of early atrial fibrillation cannot be excluded.

Potential limitations of study. The lack of atrial perfusion studies precludes a precise definition of the extent and distribution of impaired atrial perfusion and the lack of electrophysiologic studies precludes the determination of the electrophysiologic mechanism and the site of origin of atrial fibrillation. Therefore, we cannot rule out the possibility that some patients who developed early atrial fibrillation due to impaired left atrial perfusion may also have had some impairment of right atrial perfusion, but our data suggest that coexistent right atrial involvement would not have been a prerequisite for the development of early atrial fibrillation.

Clinical implication. The spontaneous onset of early atrial fibrillation in patients with acute myocardial infarction is rare and usually short-lived in patients in whom reperfusion of the artery of infarction is successful. It is probably due to left atrial ischemia secondary to occlusion of the circumflex artery proximal to the origin of its left atrial circumflex branch and coexistent impairment of atrioventricular nodal artery perfusion.

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References

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