Atrial fibrillation—a marker for abnormal left ventricular function in coronary heart disease

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SUMMARY Retrospective study of 1176 patients with known coronary heart disease by cardiac catheterisation disclosed 10 patients (0·8%) with atrial fibrillation. Comparison with 25 randomly selected patients with coronary heart disease with sinus rhythm showed that atrial fibrillation correlated significantly with impaired haemodynamic function, mitral regurgitation, and abnormalities of left ventricular contraction. Atrial fibrillation is, therefore, a useful marker of extensive myocardial dysfunction.

Although the prognosis of atrial fibrillation in acute myocardial infarction has received widespread attention,[1-3] the significance of this arrhythmia in chronic coronary heart disease has not been evaluated. In this study, we reviewed 1176 consecutive patients undergoing cardiac catheterisation and angiography for coronary heart disease. The clinical and haemodynamic data of 10 patients with chronic atrial fibrillation were compared with a randomly selected control population of 25 patients with coronary heart disease in sinus rhythm in order to examine the association of this arrhythmia with clinical course and haemodynamic function.

Subjects and methods

In the three year period between 1973 and 1976, 1176 patients who underwent cardiac catheterisation were found to have coronary heart disease. These patients were catheterised for known or suspected coronary heart disease or for the evaluation of chest pain. In addition to a complete history and examination, all had a 12 lead electrocardiogram, posterior-anterior and lateral chest x-rays, and right and left heart catheterisation, including left ventricular angiography and coronary arteriography. Patients with primary valvular heart disease, such as congenital or rheumatic valvular disease, and primary myocardial disease were excluded. The diagnosis of atrial fibrillation was made on the basis of accepted criteria.[4] Chronic atrial fibril-

lation was defined as persistent atrial fibrillation documented for at least six months before admission. Cardiomegaly was determined by the chest x-ray film, and was defined as a cardiothoracic ratio of greater than 50%. Haemodynamic studies were performed by methods and criteria previously published.[5] Single plane ventriculography was performed in the 40° right anterior oblique projection. Significant coronary heart disease was defined as luminal narrowing of more than 50% on selective arteriography. The presence of mitral regurgitation was assessed during beats which were not ventricular premature contractions.

Patients with atrial fibrillation and significant coronary heart disease were compared with a control group of 25 patients with coronary artery disease in sinus rhythm. These were selected randomly from our records but were matched for age and sex. The clinical, haemodynamic, and angiographic features of these two groups were compared using χ^2 or Fisher's exact test for qualitative data and Student's t test for quantitative data. Significance is defined as a p value of less than 0·05.

Results

Retrospective review of 1176 patients with documented significant coronary artery disease by angiography showed 10 (0·8%) patients with chronic atrial fibrillation. Their ages ranged from 43 to 67 years. The ages of patients with sinus rhythm ranged from 46 to 73 years. There were no significant age or sex differences between the two groups. Table 1

Table 1   *Characterisation of coronary heart disease associated with atrial fibrillation*

Clinical status	Patients with atrial fibrillation (N=10)	Control subjects (N=25)	p*
Congestive heart failure	7 (70%)	2 (8%)	<0.001
Third heart sound	5 (50%)	0 (0%)	<0.001
Normal electrocardiogram	1 (10%)	14 (56%)	<0.005
Anterior infarction on electrocardiogram	6 (60%)	1 (4%)	<0.002
Cardiomegaly on chest x-ray film	10 (100%)	3 (12%)	<0.001
Diabetes mellitus	4 (40%)	7 (28%)	NS
History hypertension	5 (50%)	9 (36%)	NS
Systolic murmur	4 (40%)	6 (24%)	NS
Smoking history	4 (40%)	10 (40%)	NS

*Fisher's exact test.

Table 2   *Haemodynamic and angiographic characterisation of coronary heart disease associated with atrial fibrillation*

Catheterisation data	Patients with atrial fibrillation (N=10)	Control subjects (N=25)	p value
Single vessel disease	3 (30%)	8 (32%)	NS
Double vessel disease	2 (20%)	9 (36%)	NS
Triple vessel disease	5 (50%)	8 (32%)	NS
Contractile pattern normal	0 (0%)	10 (40%)	<0.005
Asynergy			
Inferior	3 (30%)	6 (24%)	NS
Anteroapical	4 (40%)	7 (28%)	NS
General hypokinesia	3 (30%)	2 (8%)	NS
Mitral regurgitation	5 (50%)	3 (12%)	<0.05
LVEDP (mean) (mmHg)	19±9.9	16±7.0	<0.01
LVEDV index (mean) (ml/m)	95±34	59±28	<0.005
Ejection fraction	0.30±0.17	0.66±0.18	<0.001

LVEDP, left ventricular end-diastolic pressure (mmHg); LVEDV, left ventricular end-diastolic volume/body surface area (ml/m).

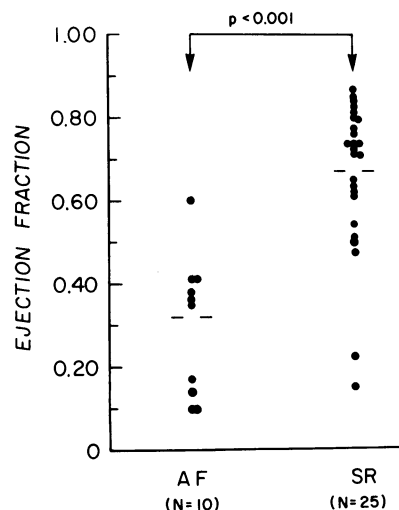details the clinical characteristics of the two groups. There were highly significant correlations between atrial fibrillation and congestive heart failure (p<0.001), the presence of a third heart sound (p<0.001), and cardiomegaly (p<0.001). Anterior myocardial infarction was present in 6 of the 10 patients with atrial fibrillation compared with two of the 25 patients with sinus rhythm (p<0.002), while the electrocardiogram was normal in only one of the former compared with 14 of the latter (p<0.005). There was no significant difference between the two groups in the duration of symptoms, or in the presence of hypertension, diabetes mellitus, or a systolic murmur. The haemodynamic and angiographic findings are summarised in Table 2. No significant differences were found in the severity or anatomical distribution of coronary arterial obstruction. Analysis of left ventricular contractile patterns disclosed that this was normal in 10 (40%) of the control group with sinus rhythm but in none of those with atrial fibrillation (p<0.005). Furthermore, the latter had a significantly decreased ejection fraction compared with control subjects (0.30 vs 0.66, p<0.001) (Fig.). There was an increase in both the mean left ventricular end-diastolic pressure (19±9.9 mmHg vs 16±7.0 mmHg, p<0.01) and the mean left ventricular end-diastolic volume index (95±34 ml/m² vs 59±28 ml/m², p<0.005) in the patients with atrial fibrillation, in whom mild mitral regurgitation with opacification of the left atrium during one or two beats was statistically more frequent (three vs five patients).

Discussion

Although atrial fibrillation in the course of acute myocardial infarction has been extensively studied,[1-9] our observations on the clinical and angiographic significance in chronic coronary heart disease have not been previously described. Our data demonstrate that atrial fibrillation in patients with chronic coronary heart disease is associated with significant impairment of left ventricular function,

Fig.   *Comparison of left ventricular ejection fraction in patients with atrial fibrillation and control group with normal sinus rhythm.*

mitral regurgitation, and left ventricular contraction abnormalities.

In the acute setting, considerable debate still exists concerning the prognostic significance of this arrhythmia. Several authors[6-8] have found an increased mortality in patients with atrial fibrillation during acute myocardial infarction. Cristal et al.[3] have emphasised that a greater overall mortality occurred when this rhythm was associated with acute anterior infarction. Others, however, have not shown an enhanced mortality.[2 9-11] A variety of mechanisms leading to the genesis of atrial fibrillation in acute infarction has been proposed which includes left ventricular failure, atrial infarction, pericarditis, and endogenous catecholamine release.[1 6] It is of interest that while the incidence of atrial fibrillation in acute

myocardial infarction is approximately 10%,[7-9 12] our study population of 1176 patients with coronary artery disease discloses an incidence of 0·08%. Thus, while this rhythm is relatively frequent in the acute infarction period, it probably results from transient causes since persistent atrial fibrillation in chronic coronary artery disease is uncommon.

Whatever the mechanism of initiation of atrial fibrillation in patients with coronary artery disease, the absence of concerted atrial systole reduces effective cardiac output by 5 to 15%.[13] It is well known that in patients with impaired left ventricular function, the advent of atrial fibrillation results in prompt clinical deterioration.[13] Our findings that atrial fibrillation is associated with myocardial infarction and seriously impaired left ventricular function suggest that the latter may cause the arrhythmia by increasing left atrial pressure and, thus, by a vicious circle mechanism, lead to further deterioration of myocardial performance. The findings of heart failure and left ventricular haemodynamic deterioration are not related to differences in coronary anatomy since the extent and the distribution of angiographic lesions were similar in the group with atrial fibrillation and the control group. Another function of effective atrial contraction is the role in mitral and tricuspid valve closure in preventing ventriculoatrial regurgitation.[13 14] This may be an additional explanation of the finding of mitral regurgitation in 50% of the group with atrial fibrillation compared with 12% of the patients with sinus rhythm (p<0·05).

While an irregular heart rate may result in a variable position of the mitral leaflets in presystole with resulting valvular regurgitation, we do not believe that atrial fibrillation is the sole cause of this. Mitral regurgitation is frequently associated with left ventricular dilatation, contractile abnormalities, and papillary muscle dysfunction, all of which were found in our patients with atrial fibrillation.[14-16] Therefore, atrial fibrillation and mitral regurgitation may coexist in the setting of advanced coronary heart disease.

In summary, atrial fibrillation in chronic coronary heart disease is unusual. The presence of this arrhythmia is, however, correlated with impairment of haemodynamic function, mitral regurgitation, and abnormalities of left ventricular contraction. It is, thus, a useful marker of extensive myocardial dysfunction.

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