Haemodynamic events in right and left ventricle during angina induced by atrial pacing

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Atrial pacing to the point of angina or up to a maximum rate of 160/min was carried out in 37 patients during coronary angiography. In 9 patients with normal coronary arteries both the right and left ventricular end-diastolic pressures fell or remained steady with increasing heart rate. The same response was observed in 14 patients with angiographically proven coronary artery disease. In the remaining 14 patients with proven coronary artery disease the end-diastolic pressure in the left ventricle rose at the point when the patient experienced angina. In 10 of this latter group there was a simultaneous rise in the right ventricular end-diastolic pressure. In 4 patients the rise in end-diastolic pressure was seen in the left ventricle only.

The haemodynamic events in the right ventricle during angina appeared in most cases to mimic the events occurring in the left ventricle. There was no correlation between the angiographic findings and the changes in end-diastolic pressure in the two ventricles during angina.

Atrial pacing has been shown to be a simple and useful means of assessing patients with ischaemic heart disease (Sowton *et al.*, 1967; Balcon, Maloy, and Sowton, 1968). The left ventricular end-diastolic pressure in subjects with normal coronary arteries, with or without chest pain, usually falls as the heart rate is increased (Parker, Khaja, and Case, 1971; Arbogast and Bourassa, 1973). In patients with proven coronary artery disease ventricular function is depressed during the induced myocardial ischaemia with a concomitant rise in left ventricular end-diastolic pressure. The left ventricular end-diastolic pressure returns to its control value after pacing is stopped and after the disappearance of angina (Friesinger, Conti, and Pitt, 1967; Helfant *et al.*, 1970; Parker *et al.*, 1971). This rise in left ventricular end-diastolic pressure is not seen in all patients (Bahler and Macleod, 1971) and not all patients will develop angina on pacing.

Overall, exercise appears to be a superior stress, usually demonstrating functional abnormalities such as a raised end-diastolic pressure even in the absence of pain (Parker *et al.*, 1969). For the individual patient atrial pacing still remains a useful test, particularly in assessing change in left ventricular performance before and after operation (Di Luzio, Roy, and Sowton, 1974).

The effects of ischaemia on the right ventricle and the relation between any ischaemic changes in the right ventricle and the arteriographic status of the coronary arteries is unknown. In the present study a comparison of the events occurring in both the right and the left ventricles during angina induced by atrial pacing has been made.

Subjects and methods

Thirty-seven patients with a clinical diagnosis of ischaemic heart disease and severe 'angina' were referred for coronary arteriography. Routine assessment, including history, physical examination, electrocardiogram, full blood count, chest x-ray, and exercise test was carried out in all patients with the exception of 7 patients in whom the exercise test was omitted because of rest pain or recent infarction. The exercise test was carried out using a bicycle ergometer, with the patient being encouraged to exercise to the point of angina or exhaustion. Electrocardiographic changes were recorded during exercise and the maximum work performed by the patient was expressed as kilopond metres (1 kpm = 9.8 Nm). Before coronary angiography, atrial pacing using a No. 5 electrode catheter passed to the right atrium via a cubital vein cutdown was carried out. Pressure changes in the right ventricle were recorded by means of a Cournand catheter also passed into the right ventricle via the cubital vein. Pressure changes in the left ventricle were recorded via a pigtail or Gensini catheter passed to the left ventricle retrogradely via a femoral artery percutaneous puncture.

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The atrial pacing rate was increased in increments of 10 beats/min, each period of pacing being maintained for 2 minutes until i) the patient experienced anginal pain, ii) a rate of 160 beats/min was reached, iii) ischaemic changes occurred in the ST segment of the electrocardiogram, iv) capture became intermittent. After a suitable rest period left ventriculography and right and left coronary angiography were carried out.

The coronary angiographic findings were graded, such that a 50 per cent stenosis was designated grade 1, greater than 70 per cent stenosis grade 2, and total vessel occlusion grade 3. The end-diastolic pressures in the right and left ventricles at rest, at each pacing rate, and at the end point of pacing were compared with the exercise tolerance and the angiographic findings in each patient.

Results

The coronary arteriograms were normal in 9 patients (Table 1). In these patients the left ventricular end-diastolic pressure fell initially, with a slight rise at higher pacing rates (Fig. 1). There was little change in the right ventricular end-diastolic pressure as the pacing rate increased. In the 14 patients with proven coronary artery disease (Table 2) in whom the final end-diastolic pressure was lower than the initial pressure, the pattern of response was similar to that seen in patients with normal coronary arteries.

It is not immediately obvious why 14 of the

TABLE 1 Nine patients with normal angiograms in whom the right and left ventricular end-diastolic pressures remained steady or fell on pacing

Case No.	RV Before	RV After	LV Before	LV After	End point of pacing	Exercise (kpm)	Coronary angiogram
1	8 <i>1.1</i>	8 <i>1.1</i>	10 <i>1.3</i>	12 <i>1.6</i>	130/min	2400	Normal
2	10 <i>1.3</i>	5 <i>0.7</i>	16 <i>2.1</i>	10 <i>1.3</i>	130/min 2:1 block	13000	Normal
3	7 <i>0.9</i>	1 <i>0.1</i>	14 <i>1.9</i>	15 <i>2.0</i>	160/min	600	Normal
4	6 <i>0.8</i>	4 <i>0.5</i>	10 <i>1.3</i>	4 <i>0.5</i>	110/min pain no ECG change	2700	Normal
5	7 <i>0.9</i>	5 <i>0.7</i>	12 <i>1.6</i>	9 <i>1.2</i>	160/min	2400	Normal
6	3 <i>0.4</i>	3 <i>0.4</i>	7 <i>0.9</i>	8 <i>1.1</i>	160/min	2400	Normal
7	4 <i>0.5</i>	2 <i>0.3</i>	9 <i>1.2</i>	0	140/min vague pain	4500	Normal
8	7 <i>0.9</i>	4 <i>0.5</i>	15 <i>2.0</i>	10 <i>1.3</i>	150/min pain	2200	Normal
9	4 <i>0.5</i>	5 <i>0.7</i>	9 <i>1.2</i>	9 <i>1.2</i>	160/min	6800	Normal

Figures in italics are pressures in kPa.
1 kpm = 9.8 Nm.

FIG. 1 Average left and right ventricular end-diastolic pressure at different atrial pacing rates in 9 patients with normal coronary arteriograms.

patients with apparently similar arteriographic findings did not respond with a steep rise in end-diastolic pressure. However, in 6 of these patients (Table 2) atrial pacing to a rate of 160/min was carried out without pain, and in general this group had a higher mean exercise level (3592 kpm) than the group (Table 3) who developed angina and steep rises in left ventricular end-diastolic pressure. This suggests that these patients were functionally better off than those listed in Table 3, and that atrial pacing to 160/min was not sufficiently stressful to produce an ischaemic response. Atrial pacing was stopped within half a minute of the patient stating that pain was present. The subjective awareness of pain differs from patient to patient and it may be

TABLE 2 Fourteen patients with proven coronary artery disease with no change in end-diastolic pressure on pacing

Case No.	RV Before	After	LV Before	After	End point of pacing	Exercise (kpm)	Coronary angiograms	Grading of lesion
10	5 0.7	5 0.7	7 0.9	7 0.9	140/min pain	2520	Cx 2	LAD 0 R 3
11	7 0.9	7 0.9	12 1.6	12 1.6	120/min 2:1 block	—	Cx 2	LAD 2 R 1
12	3 0.4	4 0.5	13 1.7	15 2.0	160/min vague pain	2500	Cx 2	LAD 0 R 0
13	5 0.7	4 0.5	10 1.3	10 1.3	160/min	5600	Cx 0	LAD 2 R 3
14	5 0.7	5 0.7	10 1.3	9 1.2	160/min pain	1400	Cx 0	LAD 3 R 1
15	2 0.3	2 0.3	8 1.1	8 1.1	150/min pain	4600	Cx 2	LAD 2 R 1
16	8 1.1	9 1.2	5 0.7	5 0.7	160/min	7300	Cx 0	LAD 1 R 1
17	7 0.9	7 0.9	11 1.5	12 1.6	120/min pain	2400	Cx 3	LAD 2 R 3
18	8 1.1	8 1.1	30 4.0	30 4.0	160/min	—	Cx 2	LAD 2 R 3
19	5 0.7	4 0.5	18 2.4	17 2.3	160/min	5400	Cx 1	LAD 1 R 3
20	9 1.2	6 0.8	19 2.5	12 1.6	140/min	3600	Cx 2	LAD 0 R 3
21	9 1.2	9 1.2	21 2.8	21 2.8	110/min pain	600	Cx 2	LAD 2 R 0
22	9 1.2	10 1.3	12 2.9	12 2.9	160/min	—	Cx 0	LAD 2 R 0
23	3 0.4	4 0.5	11 1.5	10 1.3	160/min	—	Cx 0	LAD 0 R 3

1 = 50%; 2 = 70%; 3 = 100% stenosis.

Cx = Circumflex coronary artery.

LAD = Left anterior descending coronary artery.

R = Right coronary artery.

Figures in italics are pressures in kPa.

that atrial pacing for a further minute in the remaining 8 patients of Table 2 would have produced similar rises in end-diastolic pressure to those experienced by patients listed in Table 3.

In the remaining 14 patients (Table 3) the left ventricular end-diastolic pressure rose at a much lower pacing rate than in normals. The final rise in end-diastolic pressure at the onset of angina was much higher than the rise seen in the normals at higher pacing rates. In 10 of these 14 patients there was a simultaneous rise in the right ventricular end-diastolic pressure (Fig. 2), though the peak pressure was not as high as that in the left ventricle. In 4 of the patients the right ventricular end-diastolic pressure did not rise (Fig. 3).

There was no correlation observed between the angiographic appearances in those patients with coronary artery disease and the events occurring in either the left or right ventricle.

Discussion

In patients with coronary artery disease the left ventricular end-diastolic pressure follows the normal response to an increasing heart rate up to the point of angina. At the onset of angina the left ventricular end-diastolic pressure usually rises, though its failure to rise does not exclude coronary artery disease. It may well be that very sensitive patients complain of angina earlier and atrial pacing is terminated before a decline in ventricular function is observed. It is possible that continued pacing at this point would elicit a rise in end-diastolic pressure.

In spite of the apparent rarity of infarction of the right ventricle (Wartman and Hellerstein, 1948), the response of the right ventricle to ischaemia was, in the majority of patients, similar to that occurring in the left ventricle, i.e. the right ventricular end-diastolic pressure rose significantly above the resting value. The reason for the failure of the right ven-

TABLE 3

Case No.	RV Before	RV After	LV Before	LV After	End point of pacing	Exercise (kpm)	Coronary angiogram Grading of lesion
<i>Ten patients in whom there was a rise in both right and left ventricular end-diastolic pressure on pacing</i>							
24	6 0.8	8 1.1	8 1.1	30 4.0	100/min pain	500	Cx 2 LAD 2 R 2
25	8 1.1	11 1.5	10 1.3	15 2.0	130/min pain	400	Cx 2 LAD 2 R 1
26	5 0.7	10 1.3	5 0.7	23 3.1	130/min pain	3000	Cx 2 LAD 3 R 2
27	5 0.7	20 2.7	7 0.9	30 4.0	160/min pain	—	Cx 2 LAD 2 R 2
28	4 0.5	13 1.7	10 1.3	32 4.3	160/min pain	2200	Cx 2 LAD 3 R 3
29	1 0.1	15 2.0	12 1.6	30 4.0	110/min pain	—	Cx 2 LAD 2 R 2
30	4 0.5	8 1.1	11 1.5	25 3.3	110/min pain	420	Cx 2 LAD 2 R 2
31	3 0.4	10 1.3	20 2.7	25 3.3	150/min pain	3400	Cx 2 LAD 2 R 3
32	5 0.7	8 1.1	10 1.3	13 1.7	120/min pain	1200	Cx 2 LAD 2 R 2
33	7 0.9	10 1.3	13 1.7	33 4.4	140/min pain	—	Cx 2 LAD 2 R 2
<i>Four patients with a rise in left ventricular end-diastolic pressure only</i>							
34	10 1.3	9 1.2	15 2.0	25 3.3	140/min pain	1300	Cx 1 LAD 2 R 2
35	7 0.9	7 0.9	5 0.7	17 2.3	120/min pain	600	Cx 2 LAD 0 R 3
36	3 0.4	3 0.4	21 2.8	26 3.5	150/min pain	—	Cx 0 LAD 2 R 2
37	8 1.1	8 1.1	8 1.1	28 3.7	160/min pain	—	Cx 0 LAD 2 R 0

Abbreviations as for Table 2.

Figures in italics are pressures in kPa.

FIG. 2 Average end-diastolic pressures at different atrial pacing rates in 10 patients in whom there was a rise in both left and right ventricular end-diastolic pressure on pacing.

FIG. 3 As in Fig. 2, in 4 patients in whom there was a rise in left ventricular end-diastolic pressure only.

tricular end-diastolic pressure to rise in 4 of the patients at the point of angina despite obvious rises in the left ventricular end-diastolic pressure is obscure.

The end-diastolic pressure rise in the right ventricle occurred simultaneously with that in the left, suggesting that the rise in right ventricular pressure is a primary event and not secondary to pressure changes transmitted via the pulmonary vasculature. This direct effect may be due to ischaemic changes affecting the septum or the right ventricle itself.

There did not appear to be any correlation between events occurring in either ventricle in response to ischaemia, and the pattern of disease seen on the coronary angiogram.

It is concluded from this study that the pattern of response to ischaemia seen in the left ventricle, a rise in end-diastolic pressure, is also seen in the right ventricle in the majority of patients.

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