Evaluation of rapid atrial pacing in diagnosis of coronary artery disease

Evaluation of atrial pacing test

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Seventeen patients presenting with anginal-type pain were studied by bicycle exercise testing, rapid atrial pacing, and coronary angiography. Ten patients with angina and abnormal pacing tests at rates less than 180/minute were found to have significant coronary artery disease as demonstrated by coronary angiography. Seven patients with pacing-induced chest pain only at rates of 180 and above had normal coronary angiograms. This suggests that patients requiring rates of 180 or more to produce a positive atrial pacing test, following our protocol, do not usually have significant coronary artery disease though confirmation requires a larger study.

Atrial pacing has proved to be a useful, reproducible test (Balcon *et al.*, 1969) in assessing myocardial function (Linhart, 1972), in order to enable myocardial lactate metabolism to be studied, providing objective evidence of myocardial ischaemia (Neill, 1968; Parker *et al.*, 1969; Forrester *et al.*, 1971), and in the diagnosis of ischaemic heart disease (Livesley and Oram, 1973), but does not correlate with coronary angiography as well as the angina-limited work load during progressive exercise testing (Bahler and MacLeod, 1971). To make the test more sensitive, Cokkinos *et al.* (1973) advocated the use of atropine, to enable a higher pacing rate to be used. Using atropine, we had seen a few patients in whom pacing at very fast rates produced evidence of ischaemia, but the exercise test and coronary angiogram were normal. To assess these findings further, we studied 17 patients, referred with a possible diagnosis of angina pectoris, by exercise and pacing tests, and then coronary angiography.

Patients and methods

Seventeen patients were studied by exercise testing and atrial pacing to evaluate their presenting

symptom of chest pain after informed consent was given for the pacing study, and coronary angiography was subsequently performed, to assess their suitability for coronary artery surgery. Two other patients with normal exercise and pacing tests up to 210/min were not considered to have sufficiently severe symptoms to warrant further investigation. No patients were receiving medication apart from, in some cases, β -blockers, which were stopped one week before investigation. The assessment of whether the pain was typical or atypical of angina pectoris was made prospectively by one of us (D. C. Fluck) on the basis of the patient's history, physical examination, chest x-ray film, and resting electrocardiogram.

Exercise test

This was performed on one occasion on an Elema-Schönander bicycle ergometer. After being familiarized with the equipment, the patients were rested and then performed continuous multistage ergometry, starting at 25 Joules per second and increasing every minute by 25 Joules per second, until limited by fatigue. angina, ischaemic electrocardiographic abnormalities, or arrhythmias. Patients were monitored by bipolar transthoracic electrocardiograph leads, and a full standard electrocardiogram was recorded immediately after exercise. An ischaemic exercise electrocardiogram was defined as

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FIG. 1 ST changes on stopping pacing associated with pacing induced chest pain (Case 4).

one with horizontal (< 1 mV/s) or downsloping ST depression more than 0.1 mV, occurring 80 ms beyond the R wave, for five consecutive beats (Roitman, Jones, and Sheffield, 1970).

Atrial pacing test

Catheterization was performed in the post-absorptive state after premedication with diazepam 5 mg orally 2½ hours before pacing, in all patients, as diazepam can increase myocardial blood flow (Ikram, Rubin, and Jewkes, 1973). A Zucker No. 6 or 7 bipolar pacing catheter was introduced into the mid-point of the main coronary sinus, and a No. 18 polyethylene catheter into the brachial artery. The position of the coronary sinus catheter was confirmed at the beginning and end of the study by the injection of contrast medium. Atropine 0.6 mg or 1.2 mg i.v. was administered 5 minutes before pacing. Pacing was begun using a Devices pacemaker at a rate of 20 beats/minute above the highest exercise heart rate obtained, or 160/minute, whichever was the less (Cases 10, 13, and 15 could only be paced at lower rates), and increased by 10 beats/minute every minute for 5 minutes, until limited by pain or the development of conduction block, when the rate was kept constant for the rest of the 5-minute period.

A bipolar transthoracic electrocardiographic lead was monitored throughout. We used pacing-

induced chest pain, suggestive of angina pectoris, and similar to the patients' original pain, as our end-point (Kelemen *et al.*, 1973). ST depression, persisting after pacing was stopped, except for the first post-pacing beat (Parker *et al.*, 1969) (Fig. 1 and 2), was measured.

Coronary cineangiography

This was subsequently performed, together with left ventriculography, by the Judkins technique. The degree of coronary artery narrowing was assessed independently (Dr. R. Pridie), each vessel being graded as: 0—normal; 1—plaque without significant narrowing ($< 50\%$); 2—50 to 70 per cent narrowing in area; 3— > 70 per cent narrowing in area; 4—vessel occluded.

Results (Tables 1 and 2)

Group A

Seven patients required pacing up to 180 beats/minute or more to produce chest pain, associated with > 1 mm ST depression in 6. All 7 patients had normal exercise electrocardiograms, but no significant coronary arterial lesions on angiography.

Group B

All 10 patients had significant coronary arterial

FIG. 2 ST changes on stopping pacing in a patient in whom pain did not occur.

TABLE 1 Results of pacing and angiography: Group A

Case No.	Sex	Age (y)	History	Exercise test	Pacing		Angiography					
					Rate	Post-pacing ST-depression (mm)	Pain	LM	LAD	Cx	R	EF
1	M	39	Atypical	—	210	1.5	+	0	0	0	0	82.6
2	M	59	Atypical	—	180	1.5	+	0	0	0	0	71.1
3	M	23	Atypical	—	200	2	+	0	0	0	0	70.4
4	F	46	Atypical	—	180	2.5	+	0	0	0	0	87.0
5	M	36	Angina	—	190	1.5	+	0	0	0	0	67.1
6	M	53	Angina	—	180	1.5	+	0	1	1	0	67.6
7	M	46	Atypical	—	180	0	+	0	1	0	0	69

Abbreviations: LM=left main coronary artery; LAD=left anterior descending coronary artery; Cx=circumflex coronary artery; R=right coronary artery; and EF=ejection fraction.

TABLE 2 Results of pacing and angiography: Group B

Case No.	Sex	Age (y)	History	Exercise test	Pacing		Angiography					
					Rate	Post-pacing ST-depression (mm)	Pain	LM	LAD	Cx	R	EF
8	M	53	Angina	+	130	0.5	+	0	3	3	4	67.4
9	M	54	Angina	—	150	0.5	+	0	3	1	0	81.2
10	M	56	Angina	—	150	1.5	+	0	4	1	2	83.1
11	M	50	Angina	+	120	1	+	1	3	2	1	76.1
12	M	53	Angina	+	170	1	—	0	3	2	3	62.5
13	M	57	Angina	+	145	2	+	3	1	2	4	65.2
14	M	57	Angina	+	170	2	+	0	4	2	4	61.1
15	M	56	Angina	+	140	4	+	0	3	3	4	42.6
16	M	64	Angina	+	140		+	0	3	3	3	67.4
17	M	55	Angina	—	150	3	+	0	1	4	4	70

Abbreviations: LM=left main coronary artery; LAD=left anterior descending coronary artery; Cx=circumflex coronary artery; R=right coronary artery; EF=ejection fraction.

lesions on angiography, 9 of whom had pacing induced angina at rates <180 beats/minute. Seven patients had ischaemic electrocardiographic abnormalities on exercise.

Discussion

When the atrial pacing test was first introduced (Sowton *et al.*, 1967), pacing up to 160/minute was used. Increasing the pacing rate further is often possible after intravenous atropine, even to ≥ 200 /minute, as described by Cokkinos *et al.* (1973), who advocated pacing at these fast rates to increase the sensitivity of the test. However, at these faster, sometimes unphysiological rates, not only is the oxygen consumption of the heart increased, but shortening diastole can decrease coronary blood flow (Knoebel *et al.*, 1970; Corday *et al.*, 1959) both directly by decreasing the time during which coronary blood flow occurs (Laszt and Muller, 1957; Gorlin, 1966), and indirectly by decreasing cardiac output, as a result of the decreased diastolic

filling time of the heart (Corday and Lange, 1966). Furthermore, rates of this level are rarely achieved in maximal exertion. Hence, it is conceivable that ischaemia could be produced in normal hearts if the heart is paced sufficiently fast.

In our 17 patients, the 7 who required pacing up to 180/minute or more to precipitate angina-like pain, had normal coronary angiograms, left ventriculograms, and had normal exercise electrocardiograms. Thus, with our protocol, an angina-limited pacing rate of 180/min appears to differentiate between patients with normal coronary arteries, who can be paced up to this rate, or higher, before developing angina and hence do not require coronary arteriography, while those developing pain at lower pacing rates are likely to have coronary artery disease. In contrast, Cokkinos *et al.* (1973) reported 4 patients with chest pain at rates ≥ 180 /minute, all subsequently shown to have abnormal coronary angiograms. However, all 4 had a positive double Master test. In addition, Walsh, Rickards, and Balcon (1975), reported 14 out of 50 patients,

in which a negative pacing test was associated with coronary angiograms showing >75 per cent narrowing in at least one coronary artery. These findings could be the result of differences in the pacing protocols; the importance of exercise protocols has been thoroughly investigated, in particular, a rapid increase in work load results in angina at a higher triple product (of heart rate, systolic pressure, and ejection time) than if the increase is more gradual, producing angina after three minutes exercise (Redwood *et al.*, 1971), stressing the importance of using identical pacing protocols when studies are compared.

Although it is possible that pacing at rates of 180/minute or more could produce ischaemia in normal hearts, some patients with normal coronary angiograms do have angina at rates of 150 or less (Kemp *et al.*, 1973). Some of our patients with normal coronary angiograms and pacing-induced angina may belong to this group and have an abnormal myocardium (Richardson *et al.*, 1974), but we suggest that myocardial ischaemia may also occur in normal hearts if stressed sufficiently.

Coronary angiography was performed in the Cardiac Department, Harefield Hospital by arrangement with Dr. M. K. Towers.

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