Use of atropine for higher right atrial pacing rates

'Maximal' pacing for diagnosis of coronary artery disease

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In 27 out of 79 patients investigated for coronary insufficiency a negative right atrial pacing test was obtained. These 27 patients had developed a Wenckebach or 2:1 block at rates of 110 to 150/min. Atropine, 1.0 mg, given intravenously to these patients abolished the conduction defect in 21 (77%). These patients were paced at heart rates up to 180 to 240 min. In 8, ischaemic ST changes or anginal pain occurred at these higher rates which were associated with a higher pressure-rate product in 7. The use of atropine may widen the scope of right atrial pacing and show that the negative results seen in some patients with angiographically proven coronary artery disease are due to inadequate maximal pacing rates without its use.

Right atrial pacing is a new technique for the evaluation of coronary artery disease, offering the attractive features of safety and reproducibility, which has been used quite widely since its introduction by Sowton *et al.* (1967).

Patients with the clinical diagnosis of angina pectoris and angiographically proven coronary artery disease do not always develop angina or ischaemic ST changes with right atrial pacing (Parker *et al.*, 1969; Helfant *et al.*, 1970; Conti *et al.*, 1970; Forrester *et al.*, 1971). This may be due to the main drawback of this technique, namely the occurrence of the Wenckebach phenomenon or even 2:1 block whenever atrial rates of 160-170/min are exceeded (Forrester *et al.*, 1971; Steiner *et al.*, 1971).

We tried to obtain higher heart rates than those produced by conventional right atrial pacing in some patients with suspected coronary artery disease who had given a negative result with heart rates up to 160 to 170/min.

Subjects and methods

Investigations by right atrial pacing were made on 79 patients with the clinical suspicion of coronary artery disease, between September 1971 and May 1972.

A bi- or tripolar catheter electrode (USCI) was used. This was inserted either percutaneously through the femoral vein or by an arm cutdown. Battery pacemakers (PEMCO 1427-C) were used, which can attain discharge frequencies up to 220 to 240/min. All patients were

paced until either anginal chest pain or ischaemic ST depression greater than 0.5 mm (Sowton *et al.*, 1967; Lau *et al.*, 1967) appeared, or until an atrioventricular conduction defect occurred. Leads I, II, aVF, V1 or V2, and V6 were usually recorded.

A positive result, as judged by ischaemic ST changes or anginal pain, was observed in 52 of our patients at heart rates of 110 to 220/min. Of these, 4 patients developed a positive test at heart rates above 187/min. In these patients the faster pacing rates were accompanied by a first-degree atrioventricular block only. These patients are reported elsewhere (Cokkinos, 1973).

In 27 patients a negative test was observed at heart rates of 110 to 150/min. Above these rates a Wenckebach or 2:1 block developed. To all 1.0 mg of atropine was given intravenously and pacing was repeated. These patients constitute the object of our study (Table 1).

In all patients the systemic blood pressure was monitored continuously. In 6 of the patients to whom atropine was given, the pulmonary artery, capillary wedge, or left ventricular end-diastolic pressures were measured during pacing. The pressure-rate product (systolic blood pressure $\times$ heart rate/min) (Robinson, 1967) was calculated in every patient at the various heart rate levels. This product has been found to correlate equally well with the tension-time index with the oxygen consumption of the myocardium in exercise studies (Robinson, 1967; Detry, Piette, and Brasseur, 1970) and has been used in atrial pacing studies as well (Higgs *et al.*, 1971; Chiong, West, and Parker, 1972).

Coronary arteriograms were performed immediately after the pacing study or at a later date in 14 patients either by the Sones or Judkins technique. A double Master test was obtained in 11 patients.

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TABLE I Results of 'maximal' atrial pacing in 27 patients after initial negative test

Case No.	Age (yr)	Sex	Master test	Heart rate (Hz)	Pain	ST changes	Pressure-rate product mmHg/min ($\times 10^3$)	Coronary arteriograms*
1	52	M	+	1.15 2.22	— +	— +	24.0 30.8	RCA: 90% obstruction LAD: 70% " L. circ.: 80% "
2	67	M	+	1.13 2.19	— +	— +	20.6 33.4	RCA: 70% obstruction LAD: 80% "
3	45	F	o	1.13 2.19	— —	— —	15.6 20.5	
4	47	M	o	1.13 2.15	— —	— —	18.2 21.0	
5	60	M	o	1.14 2.17	— —	— —	18.2 22.1	
6	52	M	o	1.15 2.20	— +	— +	18.0 22.0	
7	56	F	o	1.15 2.22	— —	— —	15.7 25.3	Normal
8	58	F	—	1.13 2.20	— —	— —	14.3 18.0	Normal
9	52	M	o	1.15 2.19	— +	— +	21.9 24.3	
10	60	M	o	1.13 2.19	— +	— +	18.2 26.6	
11	50	M	—	1.13 2.24	— —	— —	25.4 21.6	Normal
12	65	F	—	1.14 2.19	— —	— —	21.0 28.0	Normal
13	68	M	o	1.15 2.18	— +	— +	24.0 34.2	
14	50	F	o	1.13 2.14	— —	— —	15.6 16.8	Normal
15	56	M	+	1.12 2.12	— —	— —	14.9 14.9	RCA: 90% obstruction LAD: 70% "
16	56	M	+	1.11 2.19	— +	— +	13.2 14.3	RCA: 70% obstruction LAD: 60% "
17	53	M	o	1.12 2.22	— —	— —	14.4 25.9	Normal
18	58	M	+	1.13 2.21	— +	— +	22.1 10.5	RCA: 80% obstruction LAD: 60% " L. circ.: 60% "
19	61	M	o	1.12 2.18	— —	— —	16.8 27.0	
20	36	F	—	1.13 2.22	— —	— —	13.7 23.1	Normal
21	58	M	o	1.15 2.21	— —	— —	22.5 32.1	
22	63	M	o	1.12 2.19	— —	— —	19.2 29.9	
23	65	F	—	1.13 2.22	— —	— —	15.6 26.4	
24	60	M	o	1.11 2.20	— —	— —	18.4 30.0	
25	34	M	o	1.15 2.19	— —	— —	24.7 36.1	
26	50	F	—	1.12 2.12	— —	— —	13.2 14.4	Normal
27	46	M	—	1.12 2.17	— —	— —	12.6 18.2	

The first line for each patient represents the result of the initial test and the second line the result after atropine.
o, not done.

Master test: —negative.

„ „ +positive.

* See text

RCA, right coronary artery.

LAD, left anterior descending artery.

L. circ., left circumflex artery.

TABLE 2 Prolongation of PR interval with increasing right atrial pacing rates

Case No.	Age (yr)	At rest		PR intervals during pacing and at heart rates shown at head of each column						
		Heart rate/min	PR interval	Before atropine						
				100	110	120	130	140	150	160
1	52	88	0.16		0.22				0.23	
2	67	95	0.16				0.12	W		
3	45	95	0.16			0.17	0.17	W		
4	47	80	0.18			0.24	0.32	W		
5	60	75	0.14		0.24			0.26	W	
6	52	75	0.16			0.28	0.36		0.36	W
7	56	80	0.14				0.16		0.22	W
8	58	63	0.18			0.32	0.34	W		
9	52	72	0.18					0.24	W	2:1
10	60	67	0.24		0.32	0.34	0.36	W		
11	50	90	0.16			0.20	W			
12	65	75	0.18		0.20		0.22	0.24	W	
13	68	85	0.16			0.10	0.20	0.22	W	
14	50	80	0.17			0.18	0.19	W		
15	56	75	0.16		W					
16	56	87	0.17		0.20	0.20	W			
17	53	80	0.16		0.19	W				
18	58	90	0.18		0.20		0.20	W		
19	61	80	0.17		0.19	0.21	0.17	W		
20	36	95	0.20		0.20	0.24	W			
21	58	80	0.16		0.17		0.18		0.20	W
22	63	100	0.18		0.19	0.20	W			
23	65	65	0.10	0.18		0.19	0.19	W		
24	60	70	0.17		0.19	W				
25	34	87	0.16		0.20			0.28	0.28	W
26	50	75	0.19	0.28		0.30	W	2:1		
27	46	80	0.17	0.17	0.17	W				

* Appearance of anginal pain and ischaemic ST changes.

† Limit of pacemaker frequency.

W Occurrence of Wenckebach block.

Results

Heart rate (Tables 1 and 2)

Atropine was successful in abolishing or significantly improving the degree of the atrioventricular block in 21 of the 27 patients in whom it was used. Heart rates of 214 to 240/min were achieved in 9 patients (Cases 1, 6, 7, 11, 17, 18, 20, 21, and 23) without any atrioventricular conduction delay, or with first-degree block only (Fig. 1 and 2). In 7 patients (Cases 3, 8, 12, 19, 22, 24, and 25) a Wenckebach phenomenon was seen at heart rates of 180 to 220/min. In 5 patients (Cases 2, 9, 10, 13, and 16) pacing was stopped at heart rates of 180 to 190/min because of angina, without any evidence of atrioventricular delay (Fig. 2).

In the 6 remaining patients (Cases 4, 5, 14, 15, 26, and 27) a Wenckebach period occurred at heart rates of 115 to 170/min despite the use of atropine (Fig. 3).

Occurrence of angina or ST changes (Table 1)

Of the 21 patients in whom higher rates were achieved with the use of atropine, 8 (Cases 1, 2, 6, 9, 10, 13, 16, and 18) developed a positive test at heart rates of 180 to 220/min. In all both ST changes and angina were observed (Fig. 1 and 2). In 2 of these (Cases 1 and 18) a positive test was obtained only at heart rates of 210 to 220/min.

Coronary arteriograms (Table 1)

In 5 patients (Cases 1, 2, 4, 16, and 18) in whom a positive pacing test was obtained, there was obstructive disease of both the right and left coronary arteries. In 8 patients in whom the pacing test was negative at heart rates of 200 to 240 (Cases 7, 8, 11, 12, 14, 17, 20, and 26) the coronary arteries were completely normal. One patient (Case 15) with bilateral coronary artery disease could not be paced

After atropine

100	110	120	130	140	150	160	170	180	190	200	210	220	230	240
	0.17				0.23		0.23					0.23*		
		0.13		0.14			0.14	0.14*						
			0.14	0.17	0.21		0.21		0.22	W				
0.18		0.22	0.30	0.30	W									
			0.18	0.22		0.24	0.26	W						
				0.20				0.20	0.20	0.20*				
			0.16			0.16		0.18		0.18	W	0.16†		
					0.14	0.20	0.14	0.20	0.16*					
					0.16			0.16	1.195*			0.16		0.16†
				0.22		0.24		0.16		0.27	W			
					0.19	0.20		0.18*						
			0.18	0.19	W									
	W													
	0.17		0.17		0.16		0.16		0.16*					
	0.15		0.16		0.17		0.17			0.17		0.17†		
	0.14			0.14		0.16	0.18	0.18	0.18	0.17	0.18*			
	0.20		0.20		0.20		0.22	0.24	W					
	0.14		0.14		0.14		0.14		0.16			0.16†		
	0.15			0.15		0.16		0.16		0.18	0.19†			
		0.18		0.18		0.18		0.18	0.18	W				
	0.14		0.14		0.16		0.16		0.16		0.17	0.18†		
	0.17		0.17			0.17		0.17		0.18	W			
					0.22			0.22	0.22	W				
0.16		0.29	W	2.1										
		0.18		0.18		0.18	W							

above 115 beats/min because of a Wenckebach type block occurring at this rate.

Double Master test (Table 1)

This was positive in 3 patients in whom a positive atrial pacing test was obtained at heart rates of 187 to 220/min (Cases 1, 2, and 18). In 6 patients in whom atrial pacing rates of 180 to 240/min were associated with a negative result, the Master test was negative also (Cases 8, 11, 12, 20, 23, and 26). In 2 patients (Cases 15 and 16) in whom the Master double test was strongly positive, pacing rates of 115 and 166 respectively only could be obtained before a Wenckebach block occurred.

Systemic pressures (Table 3)

In most of the patients the changes at heart rates up to 200/min were minimal. In only 2 (Cases 11 and 18) did the blood pressure fall conspicuously at heart rates of 210 and 240/min, respectively. In the former the atrial pacing test, Master test, and coronary arteriograms were all normal. The latter developed anginal pain at 210 beats/min (Fig. 4).

Pressure - rate product (Table 1)

This increased with the faster heart rates in 25 of the patients. It decreased only in Cases 11 and 18 who developed a pronounced fall in arterial pressure with very fast heart rates.

Capillary wedge, pulmonary artery, or left ventricular end-diastolic pressures (Table 4)

In 3 patients these pressures increased with atrial pacing: in Cases 13 and 18 this occurred when the patients first complained of anginal pain. In Case 11 angina did not occur but the left ventricular end-diastolic pressure rose from 7.5 to 12.5 mmHg at a heart rate of 220/min. In 3 patients not developing angina (Cases 4, 5, and 22) these pressures did not change significantly at heart rates of 150 to 180/min.

No complications occurred as a result of the procedure. The chest pain and ST changes regressed when the cardiac rate was diminished. In the two patients in whom hypotension occurred with a very fast pacing rate, the blood pressure rose immediately after the heart was slowed (Fig. 4).

FIG. 1 Case 9. Left: pacing rate 146/min: a Wenckebach block has occurred. Middle: pacing rate 180/min: a 2:1 block has occurred. Right: after atropine, ischaemic ST changes appear with a heart rate of 187/min.

Discussion

The interesting finding in our study is that standard right atrial pacing was negative at heart rates of 110 to 150/min in 27 of our 79 patients, in whom a Wenckebach or 2:1 atrioventricular block occurred at higher rates. In 21 of these patients (77%) faster heart rates could be achieved with the use of atropine. This drug reverses the decrease of conductivity within the atrioventricular node seen with fast atrial pacing rates (Damato and Lau, 1970).

It is also interesting that in 8 patients in the atropine group, and in 4 more patients from our whole series, a positive test was obtained only after heart rates of 180/min were exceeded. Pacing up to the usual rates of 170/min may not be fast enough to provoke angina pectoris or ischaemic ST changes in all patients with coronary artery disease. Thus a parallel to the difference between maximal exercise tests and the standard double Master test as regards the frequency of positive results may exist in right atrial pacing as well (Mason *et al.*, 1967; Doan *et al.*, 1965). In this context it should be remembered that

Balcon *et al.* (1969) found that each individual patient developed angina at the same tension-time index, regardless of whether this was produced by atrial pacing or exercise.

Bahler and Macleod (1971) found that the atrial rate at which angina occurred with pacing bore little relation to the severity of the underlying coronary artery disease, while Cheng (1971) found a tendency for patients with more advanced disease to have a positive test at lower heart rates. Our coronary arteriographic studies are too few to permit any conclusion on this point. However, since the faster heart rates were associated with a significantly higher pressure-rate product in most of our patients, it is conceivable that in some individuals a significant increase in the oxygen consumption is needed to reveal a coronary circulation that is unable to meet such greatly increased requirements. Case 18 developed angina after the arterial pressure fell with a very fast heart rate, despite a decrease in the pressure-rate product, and it can be postulated

FIG. 2 Case 13. Left: pacing rate 130/min: the PR interval is 0.20 sec. Middle: pacing rate 150/min: a Wenckebach-type block has occurred. Right: after atropine, with a pacing rate of 180/min; ischaemic ST changes and anginal pain occurred.

TABLE 3 Brachial artery pressures (mmHg) with varying atrial pacing rates

Case No.	Heart rate/min														
	100	110	120	130	140	150	160	170	180	190	200	210	220	230	240
1			150/9			160/9		160/100					140/90*		
2	170/100			165/100				150/100		180/100*					
3	120/80			120/80		120/80				110/80					
4				140/90		140/110									
5			130/90		140/90			130/85							
6					120/100	120/100		110/80				110/80*			
7					105/80				115/80					115/80	
8				110/80					100/80			90/70			
9					140/80	150/100				130/80*					
10				140/90			130/80			140/80*					
11			170/90		170/100			180/100					190/100		90/80
12					150/90	150/100				150/100					
13					165/90	160/100	160/110		190/120*						
14		120/80		120/90	120/90										
15		130/75													
16		120/90				120/90				130/90*					
17			120/80			120/90				120/100			115/95	118/95	
18			170/90	170/90		160/90			110/90	110/90			50/40		
19			140/80			140/90			150/100						
20		110/80	110/90			105/90							105/95		
21						150/90			160/90				150/95		
22	160/80		160/90			160/90			160/90	160/100					
23	130/90			120/80		120/80			130/90				120/80		
24		160/80				150/80			150/90		150/90				
25	160/80			165/80					190/100						
26		120/75	120/80												
27	110/80		105/90					110/95							

* Occurrence of anginal pain and ischaemic ST changes.

FIG. 3 Case 26. Progressive atrioventricular conduction defect from left to right: First column: first-degree AV block (PR: 0.30 sec) at a heart rate of 125/min. Second column: Wenckebach block occurring at a frequency of 145/min. Third column: 2:1 block occurring with a frequency of 150/min. Fourth column: despite the use of atropine, a Wenckebach type of block persists at 145 beats/min.

TABLE 4 Pulmonary artery, capillary wedge, or left ventricular end-diastolic pressures with various pacing rates

Case No.	Heart rate/min						
	100	120	130	150	170	180	210-220
4			22/12 16 (PAP)	25/14 18 (PAP)			
5	12 (CWP)	12 (CWP)			12 (CWP)		
11	7.5 (LVEDP)			7.5 (LVEDP)			12.5 (LVEDP)
13	12 (LVEDP)	13 (LVEDP)			20* (LVEDP)		
18	6 (LVEDP)		7 (LVEDP)				22* (LVEDP)
22	32/14 20 (PAP)	33/12 20 (PAP)		32/15 20 (PAP)		30/13 18 (PAP)	

* Appearance of anginal pain.
CWP, Capillary wedge pressure.
LVEDP, Left ventricular end-diastolic pressure.
PAP, Pulmonary artery pressure.

FIG. 4 Case 18. Progressive decrease of blood pressure with increase in heart rate. First column: heart rate 140/min; blood pressure 165/110 mmHg. Second column: heart rate 180/min; blood pressure 110/80 mmHg. Third column: heart rate 214/min; blood pressure 40 mmHg; ischaemic ST changes. Fourth column: heart rate 120/min; blood pressure 170/110 mmHg.

that in some patients a very fast heart rate can decrease the myocardial blood flow beyond a critical point, producing ischaemia. It has already been reported that very fast atrial pacing rates can severely affect the myocardial (Knoebel *et al.*, 1970) and peripheral blood flow (Benchimol and Goldstein, 1969). In fact, in only 2 of our patients did a conspicuous fall in blood pressure occur with heart rates above 200/min. Similarly, a heart rate of 220/min was associated with a mild rise in the left ventricular end-diastolic pressure of a patient with a negative test with normal coronary arteries. An increase in the left ventricular end-diastolic pressure was found in 2 of our patients who developed angina, as already observed (Parker *et al.*, 1969).

We do not have enough coronary arteriograms to claim that a negative atrial pacing test at maximal heart rates is proof of a normal coronary arterial tree. However, the results in the patients who underwent coronary arteriography tend to support this view. Moreover, all patients with a positive test had significant coronary artery disease.

One difficulty in the interpretation of 'ischaemic'

ST changes in right atrial pacing has been the development of first-degree AV block with faster rates, with superimposition of the pacemaker spikes on the ST segment (Lau *et al.*, 1968). The shorter PR intervals seen in most of our patients with the use of atropine should probably help in this determination. However, we also feel that both anginal pain and ST changes should be taken into account in evaluating the results of a pacing test (Sowton *et al.*, 1967).

We think that the use of atropine can widen the scope of right atrial pacing. The achievement of faster pacing rates may offer some insight into the discrepancies seen between this technique and the exercise tests, and help to explain the occasional 'negative' tests seen in patients with clinically well-documented angina pectoris and arteriographically proven coronary artery disease. Moreover, as we have recently found out, it reverses the decrease in atrioventricular conduction seen with β -blocking agents (Sowton *et al.*, 1970). Its use can facilitate the study of the effect of these drugs on the anginal pain appearing with rapid right atrial pacing.

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