Determination of the Optimal Timing for Performing Digital Ventriculography During Atrial Pacing Stress Tests in Coronary Heart Disease

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To detemine the optimal time for recording left ventricular angiograms during atrial pacing stress tests, digital subtraction left ventriculograms were obtained using 12 ml of contrast material in 40 patients at rest and at peak pacing. Nineteen of the 40 patients had a third digital left ventriculogram performed between 5 and 10 seconds and 21 patients had a third digital left ventriculogram performed 30 seconds after pacing was stopped. Coronary angiography showed significant coronary artery disease (CAD) in 29 patients and no evidence of significant CAD in 11 patients. Ejection fraction (EF) increased or did not change at peak pacing in 10 of 11 patients without CAD. In the 29 patients with CAD, mean EF decreased an average of 10 percentage points ($p < 0.001$) and fell 2 or more percentage points in 25 patients (86%) at peak pacing. These changes in EF were accompanied by the development of wall motion abnormalities, which occurred in segments of myocardium that were supplied by coronary arteries with angiographic CAD (more than 50% diameter narrowing). In contrast, the mean EF during the postpacing studies decreased only 2.2 percentage points (difference not significant) over rest values. Moreover, 15 of 29 patients (52%) with CAD had a decrease in EF of 2 or more percentage points. Therefore, the sensitivity of the atrial pacing stress test was diminished when the analysis was performed at 10 or 30 seconds after pacing. It is concluded that EF changes and wall motion abnormalities induced by atrial pacing are of short duration. As a result, the optimal time for performing left ventricular analysis of EF and wall motion during atrial pacing is apparently at the peak heart rate and not 10 to 30 seconds after pacing is stopped.

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Atrial pacing may induce myocardial ischemia, which, in turn, may produce a decrease in left ventricular (LV) ejection fraction (EF) and the development of wall motion abnormalities in patients with coronary artery disease (CAD).[1-4] These pacing intervention studies are useful as an adjunct to coronary angiography because they yield important information about the functional significance of existing CAD.[5] The duration of ischemia induced by pacing is not well defined. The hypothesis for this study is that the optimal time for assessing LVEF and wall motion occurs at the peak heart rate, although preload and afterload at the peak heart rate are considerably different from those at rest. Digital subtraction angiography provides an opportunity to record left ventriculograms during stress interventions because the volumes of contrast material used are so low that they do not significantly affect the baseline hemodynamic state.[6,7] In the present study, low-dose (12 ml) digital left ventriculography was used to assess the optimal timing for performing an analysis of LV volume, EF and wall motion during atrial pacing stress tests in patients with CAD.

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Methods

Digital angiographic technique: Digital angiography was performed using the method of mask mode subtraction. A detailed description of our imaging system has been presented previously.[8,9] Digital angiograms were acquired using fluoroscopic images that were digitized at 30 frames/s into a 512 × 512 × 8-bit matrix. Every picture element in the image matrix was assigned a number corresponding to one of 256 shades of gray. This process of computerized mask mode substraction enhances the visualization of iodinated contrast material approximately 3 to 4 times relative to the level of contrast material required to visualize the left ventricle with

cine film-based systems.[10,11] After mask mode subtraction processing, the images were reconverted to analog format for storage on ¾-inch videotape (Sony U-matic #5800 recorder).

Patients: Forty patients participated in this study. Patients were studied because of chest pain syndromes that were refractory to medical treatment, because they had an exercise stress test suggesting significant CAD, or because the referring physician wanted to know the coronary anatomy for purposes of clinical decisions. One patient without CAD was studied because of recurrent ventricular tachycardia without a history of chest pain. Twenty-nine of the 40 patients had angiographic evidence of significant CAD. This group included 19 men and 10 women, mean age age 53.4 years (range 38 to 65). Eleven of the 40 patients had normal coronary angiographic findings. This group included 3 men and 8 women, mean age 56.6 years (range 36 to 79). There was no significant difference in the mean age between the groups with or without CAD.

Clinical protocol: Patients undergoing left ventriculography during routine cardiac catheterization for clinically indicated reasons were asked to participate in this study. Patients with left main CAD and those with unstable angina were excluded. Also excluded were patients with primary valvular heart disease or cardiomyopathy. Drugs such as long-acting nitrates and β-adrenergic antagonists were withheld for 24 hours before the study. Selective coronary angiograms were recorded in standard and caudal or cranial projections. At least 2 of these projections were orthogonal. Significant CAD was defined as at least 50% diameter narrowing of a coronary artery in at least 1 projection.

After coronary angiography was completed, a No. 6Fr Cordis bipolar temporary pacemaker wire was passed from the right femoral vein under fluoroscopic control and placed against the lateral border of the right atrium. The pacemaker wire was connected to a Medtronic external pulse generator (model 5375). A No. 7Fr Cordis angled pigtail catheter was then passed retrogradely across the aortic valve into the left ventricle. A digital left ventriculogram was performed at rest in the 30° right anterior oblique (RAO) projection by injecting 12 ml of meglumine iothalamate (Vascoray®), which had been previously diluted 1:1 with water. The diluted Vascoray was injected at a rate of 8 ml/s for 3 seconds. To perform the pacing study, the right atrium was stimulated at 2 mA initially at a rate of 20 beats/min above the patient's heart rate at rest. The rate was increased in increments of 10 beats/min every minute until chest pain developed or until a heart rate of 140 beats/min was reached. If atrioventricular Wenckebach block developed, 1 mg of atropine was administered intravenously and the pacing study was continued. When the endpoint heart rate was achieved, another 12-ml digital left ventriculogram was performed. The pacemaker was then turned off. In 19 patients a third 12-ml digital left ventriculogram was obtained between 5 and 10 seconds after the atrial pacing was stopped. In the other 21 patients, the third 12-ml digital left ventriculogram was obtained 30 seconds after atrial pacing was stopped.

Image analysis: The rest, pacing and postpacing digital left ventriculograms recorded on videotape were reviewed and the cardiac cycles in which the greatest concentration of contrast was seen in the left ventricle were selected for analysis. The LV images from the chosen cardiac cycles were redigitized by the computer so that the boundary of the left ventricle could be electronically traced by the operator directly on the video image. End-diastolic and end-systolic volumes at rest, during atrial pacing, and after pacing were calculated by the computer using the area-length method corrected for magnification by a grid. Figure 1 is an example of a digital left ventriculogram in end diastole and end systole obtained at rest, during peak pacing, and 10 seconds after pacing in a

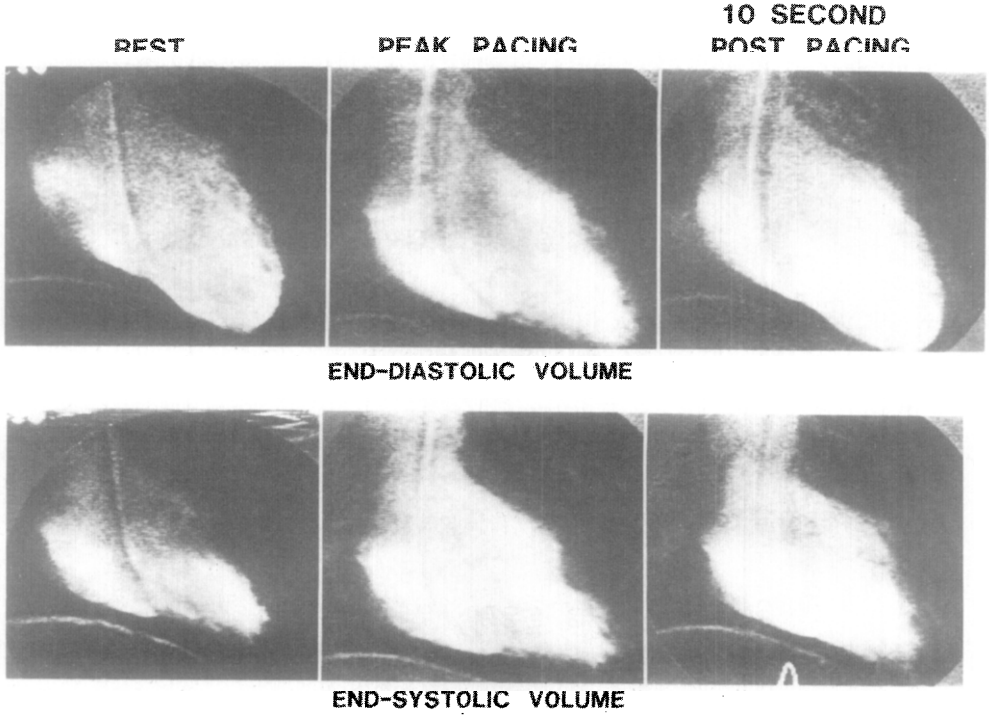

FIGURE 1. Patient 6—isolated frames from digital subtraction left ventriculograms obtained with 12 ml of contrast material. Frames at end diastole (**top row**) and end systole (**bottom row**) are shown for the studies recorded at rest, peak pacing and 10 seconds after pacing was stopped. The baseline study revealed inferior wall hypokinesia with a global ejection fraction (EF) of 40%. At peak atrial pacing to a heart rate of 140 beats/min, the patient had an abnormal electrocardiographic response, but had no chest pain. However, the left ventricle has diffuse akinesia of the anterior, apical and inferior walls, with an EF of 16%. In the 10-second postpacing study, significant hypokinesia of the anterior and inferior walls is still present and the EF is still depressed below rest at 34%, but has improved over that during the peak pacing study.

TABLE I Left Ventriculogram 5 to 10 Seconds After Pacing

Pt	Diameter Stenosis (%)				Heart Rate (beats/min)			Ejection Fraction (%)			Abnormal ECG During or Post Pacing	Chest Pain During Pacing	LVEDP (mm Hg)		
	LAD	Cx	RCA	Diag	Rest	Peak Pacing	5–10 Sec Post	Rest	Peak Pacing	5–10 Sec Post			Baseline	After Resting 12 ml Dig Angio	After 10 Sec Post Pacing Dig Angio
Abnormal Coronary Arteries															
1	100	75	70	...	72	130	73	37	29	42	−	−	8	9	8
2	...	50	50	...	76	142*	107	63	68	78	−	−	18	20	15
3	...	80	...	...	87	155	100	65	64	56	+	−	10	10	12
4	50	50	75	...	50	110	46	63	54	68	+	+	19	21	23
5	80	...	...	...	48	112	66	77	78	81	+	−	16	22	16
6	60	75	100†	80	80	140	85	40	16	34	+	−	24	25	33
7	...	...	100†	...	73	140	78	69	58	66	−	−	15	17	20
8	100†	...	...	...	65	110	68	57	44	58	+	+	16	12	19
9	30	...	...	100†	68	150	67	64	37	61	−	−	16	18	17
10	50	...	90	...	66	90	66	67	65	71	+	+	18	20	24
11	...	...	90	...	55	130	58	74	54	64	+	+	26	22	26
12	...	75	...	...	74	140	77	76	72	71	+	−	12	11	12
13	70	...	80	...	70	140	75	50	44	55	−	−	8	8	10
14	...	100†	100†	...	62	140	74	42	30	30	−	+	25	17	24
Mean ± SD					68 ± 11	131 ± 18	74 ± 16	60 ± 13	51 ± 18	60 ± 15			17 ± 6	16 ± 6	19 ± 7
Normal Coronary Arteries															
15					86	155	83	57	65	60	−	−	6		17
16					79	136	76	84	82	83	+	−	12	16	13
17					68	140	78	66	66	68	−	−	12	16	18
18					56	130	58	71	78	80	+	+	19	24	20
19					86	150	96	59	76	70	−	−	11	8	8
Mean ± SD					75 ± 13	142 ± 10	78 ± 14	67 ± 11	73 ± 7	72 ± 10			12 ± 5	16 ± 7	15 ± 5

* After atropine; † with collaterals.

Cx = circumflex; Diag = diagonal; Dig Angio = digital angiogram; ECG = electrocardiogram; LAD = left anterior descending; LVEDP = left ventricular end-diastolic pressure; RCA = right coronary artery; SD = standard deviation.

TABLE II Left Ventriculogram 30 Seconds After Pacing

	Diameter Stenosis (%)				Heart Rate (beats/min)			Ejection Fraction (%)			Abnormal ECG During or Postpacing	Chest Pain During Pacing	LVEDP (mm Hg)		
	LAD	Cx	RCA	Diag	Rest	Peak Pacing	30 Sec Post	Rest	Peak Pacing	30 Sec Post			Baseline	After Rest 12-mi Dig Angio	30 Sec Postpacing Dig Angio
Abnormal Coronary Arteries															
1	...	...	75	...	79	140	75	39	24	42	−	−	36	36	36
2	100*	...	...	...	90	140	73	45	38	35	+	−	16	16	24
3	50	100*	...	50	60	130*	62	74	66	84	−	−	20	20	20
4	...	70	70	...	58	140	62	73	69	72	+	−	8	8	10
5	80	...	...	...	60	140	52	67	74	60	−	−	8	10	16
6	90	...	...	...	93	140	90	65	66	67	+	−	14	14	18
7	...	75	100†	...	70	140	65	51	42	46	+	−	16	26	24
8	60	...	100†	...	60	140	70	57	54	67	−	+	20		24
9	...	50	100†	...	83	120*	83	70	58	63	+	+	12	17	17
10	60	...	...	...	98	160	108	72	68	70	+	−	7	10	11
11	80	90	100†	...	76	133	74	48	35	48	−	+	12	12	19
12	...	...	100	...	63	130	68	54	43	38	−	+	31	29	29
13	...	95	30	...	72	140	68	71	43	49	−	−	12	15	22
14	...	...	90	...	64	120	68	74	48	62	+	−	19	18	21
15	100	...	100†	...	48	120	53	36	13	36	+	+	20		14
Mean ± SD					72 ± 15	136 ± 11	71 ± 14	60 ± 13	49 ± 18	56 ± 15			17 ± 8	18 ± 8	20 ± 7
Normal Coronary Arteries															
16	...	...	...	...	57	100	56	78	82	78	−	−	8	14	14
17	25	...	...	...	60	136	52	83	86	85	+	−	10		10
18	...	...	...	...	68	120	65	76	76	73	−	+	14	14	20
19	...	...	...	...	75	125	74	76	75	76	−	+	8	8	11
20	...	...	...	...	61	140*	82	73	80	77	+	−	14	18	18
21	...	...	...	...	75	130	77	56	55	64	−	+	14	14	15
Mean ± SD					66 ± 8	125 ± 14	68 ± 12	74 ± 9	76 ± 11	76 ± 17			11 ± 3	14 ± 4	15 ± 4

* After atropine; † with collaterals.

Cx = circumflex; Diag = diagonal; Dig Angio = digital angiogram; ECG = electrocardiogram; LAD = left anterior descending artery; LVEDP = left ventricular end-diastolic pressure; RCA = right coronary artery; SD = standard deviation.

patient with significant 3-vessel CAD (patient 6). Statistical analysis was performed with a Student t test for paired data comparing end-diastolic volumes, end-systolic volumes and EF at rest with those obtained during atrial pacing or after pacing.

Coronary angiograms were reviewed on a Vanguard projector by 3 cardiologists. Angiographic narrowings were measured with calipers and were defined as anatomically significant when the diameter at the stenosis was at least 50% narrowed compared with the angiographically normal-appearing arterial segment proximal to the lesion. Wall motion abnormalities at rest, during atrial pacing and after pacing were outlined by an experienced angiographer and the results were reviewed by the panel. The silhouette of the left ventricle in the RAO projection was separated into anterobasal, anterior, apical, inferior and inferobasal segments.[12] Wall motion was qualitatively defined as normal, hypokinetic, akinetic or dyskinetic, based on the analysis developed by Alderman et al.[13] Regions of normal and abnormal wall motion seen on digital ventriculograms were related to the presence or absence of anatomically significant coronary artery narrowing in the artery supplying those myocardial regions. Arterial lesions were defined as functionally significant if rest or pacing-induced wall motion abnormalities were appreciated.

Results

The patient characteristics and results recorded at rest, during peak pacing, and after pacing are shown in Tables I and II. In these tables, the patients were separated into 2 groups: those in whom a third left ventriculogram was recorded 5 to 10 seconds after atrial pacing was stopped (group 1) (Table I) and those in whom a third left ventriculogram was recorded 30 seconds after pacing was stopped (group 2) (Table II). Statistical analysis was performed between the 2 groups to compare rest and peak pacing values using a Student t test for paired data. Whenever there was no significant difference between group 1 and 2 for the various measurements, the data were combined.

In 10 patients with CAD and 4 without CAD, the pacing study was stopped because chest pain developed. Atrioventricular Wenckebach block developed in 3 patients with CAD and in 1 patient without CAD, and these patients received 1 mg of atropine during the study. Two patients with CAD and 1 patient without CAD were not paced to the target heart rate despite the absence of chest pain or ischemic electrocardiographic changes because of the reluctance of the operator performing the pacing study. For the postpacing studies, there was no difference in the heart rate at 10 seconds or 30 seconds after pacing. In addition, there was no difference in the mean heart rate at rest compared with the postpacing value in patients with CAD or without CAD. No complications occurred in any of the 40 patients during or after the atrial pacing studies and the 3 digital left ventriculograms.

Hemodynamic effects: The mean LV end-diastolic pressure (EDP) at rest in the 29 patients with CAD was 17 ± 7 mm Hg. This was significantly higher than the mean LVEDP in the 11 patients without CAD (12 ± 4 mm Hg, $p < 0.05$). In the group with CAD the LVEDP was 17 ± 7 mm Hg after the initial rest 12-ml digital ventriculogram and 20 ± 7 mm Hg within 2 minutes after the postpacing digital ventriculogram. Analysis of variance revealed no significant difference in the mean LVEDP at these 3 times in the patients with CAD ($F = 1.37$). Similarly, in the 11 patients without CAD, there was no significant difference in the mean LVEDP at rest (12 ± 4 mm Hg), after the baseline 12-ml digital ventriculogram (15 ± 5 mmHg), or within 2 minutes after the postpacing digital ventriculogram (15 ± 4 mm Hg) ($F = 2.03$).

TABLE III Angiographic Results

	Rest	Peak Pacing	Postpacing (10- and 30-sec groups)
Normal Coronary Arteries (11 patients)			
EDV	110 ± 29	72 ± 22‡	110 ± 22
ESV	32 ± 12	18 ± 7‡	29 ± 10
SV	78 ± 25	54 ± 19‡	81 ± 18
EF	71 ± 10	75 ± 9	74 ± 8*
Abnormal Coronary Arteries (29 patients)			
EDV	138 ± 39	99 ± 30‡	145 ± 35*
ESV	57 ± 31	52 ± 31†	64 ± 34†
SV	80 ± 21	48 ± 19‡	82 ± 19
EF	60 ± 13	50 ± 18‡	58 ± 15

Values compared with rest: * $p < 0.05$; † $p < 0.01$; ‡ $p < 0.001$.
EDV = end-diastolic volume; EF = ejection fraction; ESV = end-systolic volume; SV = stroke volume.

Angiographic results: The results of the volumetric measurements from the digital left ventriculograms are summarized in Table III. The individual patient data for end-diastolic volume are shown in Figure 2. In the 11 patients without CAD and the 29 patients with CAD, the end-diastolic volume decreased 34.7% and 27.4%, respectively ($p < 0.001$), during peak pacing. In the patients without CAD, end-diastolic volume returned to baseline levels after pacing. However, in the patients with CAD, the mean end-diastolic volume increased slightly (7.5%) above the baseline value during the postpacing study ($p < 0.05$).

The patient data for end-systolic volume are shown in Figure 3. In the patients without CAD, mean end-systolic volume decreased 41.8% ($p < 0.001$) during peak pacing and returned to baseline levels after pacing. The patients with CAD had a more variable response in end-systolic volume at peak pacing. The mean end-systolic volume decreased only 10%, which was significantly different ($p < 0.001$) from the mean change in patients without CAD. During the postpacing ventriculogram, the mean end-systolic volume increased 14.6% compared with the rest value ($p < 0.01$) in the patients with CAD.

The response of EF to atrial pacing is summarized in Figure 4 for the 11 patients without CAD. The mean EF increased 3.8 percentage points during peak pacing and achieved a p value of 0.06 compared with values obtained at rest. In 10 of the 11 patients (91%) EF increased or did not change (i.e., 2% or less) during peak pacing. Only 1 patient without CAD had at least a 2 percentage point decrease in EF, and this patient's EF at rest was 84%. During the postpacing study, mean EF increased 3.1 percentage points above the value at rest and achieved a p value of 0.05 compared with rest. In 10 patients (91%) EF increased or did not change during

the postpacing study. In 1 of the patients without CAD EF decreased 3 percentage points at 30 seconds after pacing, and this patient had an EF at rest of 76%.

The EF response to atrial pacing in the 29 patients with CAD is shown in Figure 5. At the peak pacing rate, the mean EF decreased 9.9% ($p < 0.001$) compared with the rest value. In 4 patients EF increased or did not change, whereas in 25 (86%) EF decreased at least 2 percentage point at peak pacing. During the postpacing studies the mean EF decreased 2.2 percentage points from the baseline value, which was not a significant change. In only 15 patients (52%) with CAD did EF decrease by 2 percentage points or more during the postpacing study. There was no significant difference in EF response between the group of patients who had studies at 5 to 10 or 30 seconds after pacing in either the mean decrease in EF (0.5 and 3.7 percentage points, respectively) or in the number of patients who showed a decrease in EF of more than 2 percentage points (7 of 14 and 8 of 15, respectively).

Wall motion analysis: Four of the 29 patients with CAD had anterior wall hypokinesia at rest and all 4 had corresponding lesions in the left anterior descending or diagonal coronary arteries. Eight of the 10 patients with CAD who had inferior wall hypokinesia at rest had complete occlusion of the right coronary artery, but 7 of these 8 patients had collateral blood supply to the right coronary artery. Two of the 13 patients with wall motion abnormalities at rest had abnormal contraction at rest in both anterior and inferior walls, with corresponding lesions in the left anterior descending and right coronary arteries. Sixteen patients with CAD had normal wall motion at rest.

During the peak pacing study, in 23 of the 29 patients (79%) with CAD either new wall motion abnormalities developed or existing hypokinesia worsened, whereas in 4 patients segmental wall motion abnormalities did not develop. Two patients did not have wall motion abnormalities during peak pacing, but did show segmental hypokinesia during postpacing study. Alternatively, in 8 patients segmental wall motion abnormalities developed during the peak pacing study, but the abnormalities reverted to normal at the 5- to 10-second or 30-second postpacing study.

Only 1 of the 11 patients without CAD had mild anterior hypokinesia at rest, and it improved at peak

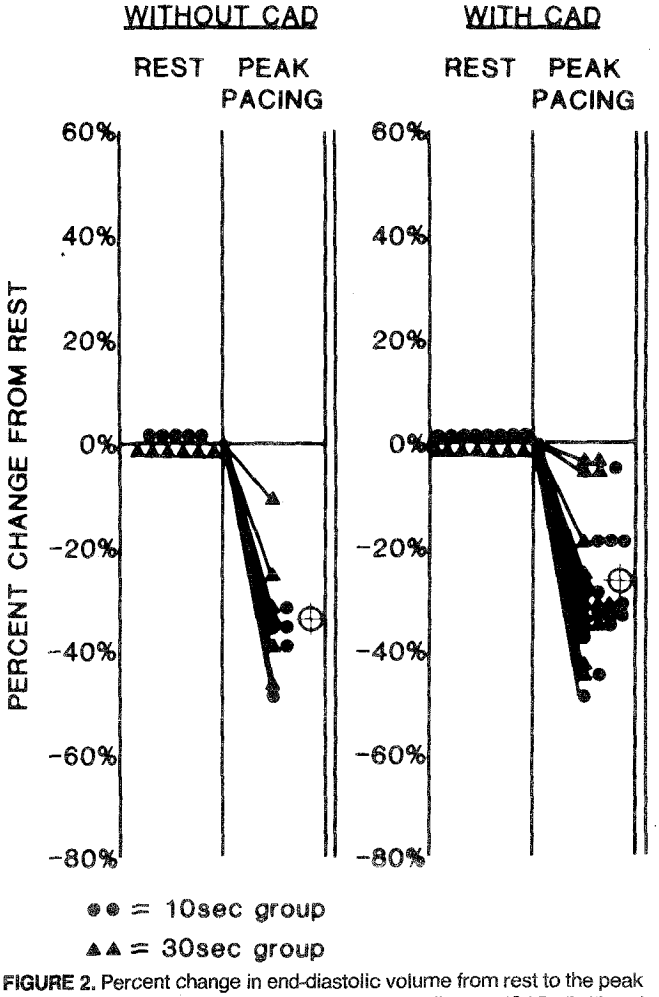

FIGURE 2. Percent change in end-diastolic volume from rest to the peak pacing for the patients without coronary artery disease (CAD) (**left**) and patients with CAD (**right**). The **circles** represent patients who subsequently underwent left ventriculography 10 seconds after pacing and **triangles** represent patients who subsequently underwent left ventriculography 30 seconds after pacing. There is no significant difference in the percent decrease in end-diastolic volumes between the patients with and those without CAD.

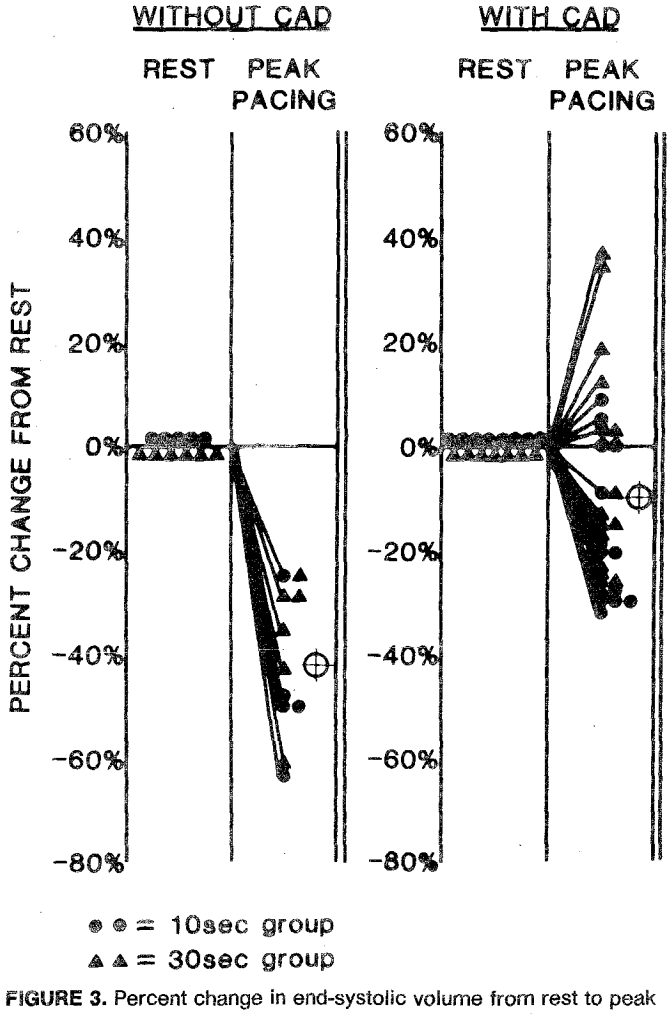

FIGURE 3. Percent change in end-systolic volume from rest to peak pacing for the patients without coronary artery disease (CAD) (**left**) and those with CAD (**right**). There is a significant difference ($p < 0.001$) in the amount that the end-systolic volume decreased in patients without CAD compared those with CAD.

pacing as EF increased. In 2 patients without CAD, mild anterior hypokinesia developed during peak pacing. EF did not change significantly in these 2 patients during pacing.

Discussion

Coronary angiography delineates the anatomy of the coronary arteries and reveals the presence and severity of coronary narrowing. However, coronary angiography alone does not yield information about the functional significance of atherosclerotic lesions.[14] In addition, there is often disagreement among observers about the severity of specific coronary lesions.[15–19] Therefore, it would be clinically useful to have a stress test that reliably demonstrates alterations in segmental ventricular function produced by myocardial ischemia resulting from specific coronary artery lesions. Atrial pacing is a means of inducing myocardial ischemia and has the benefit of being easily performed at the time of cardiac catheterization. It is not dependent on the patient's ability to cooperate with or perform physical exertion.

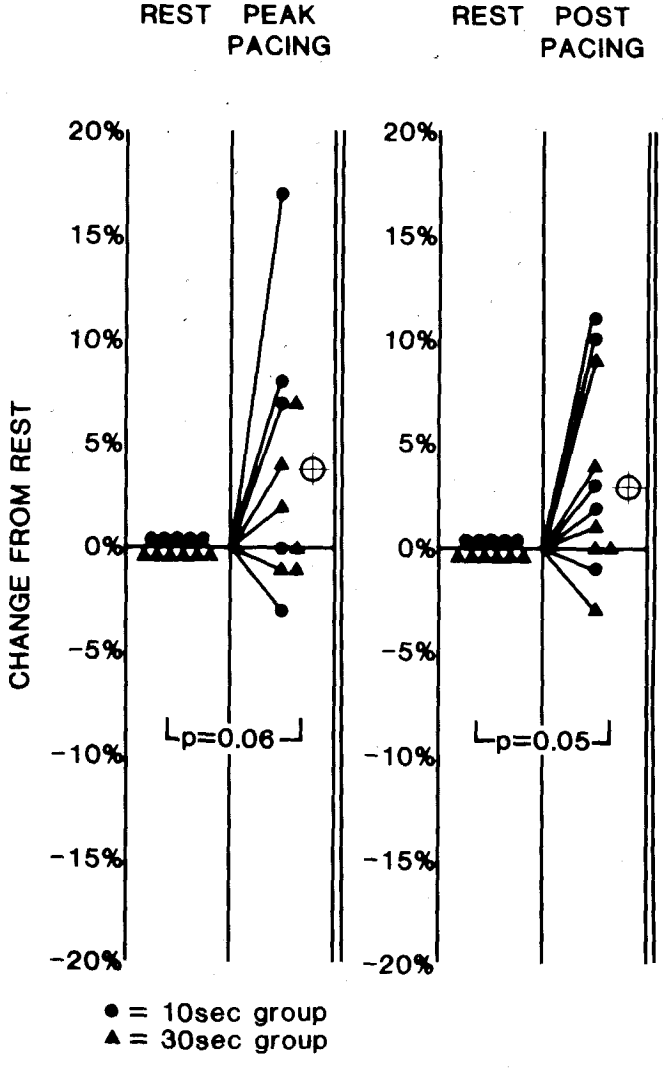

FIGURE 4. Ejection fraction (EF) response in patients without coronary artery disease. **Left,** change from rest to peak pacing; **right,** change from rest to the postpacing study. Mean EF increased mildly at both peak pacing and postpacing studies.

The purpose of this study was to determine the optimal time for recording left ventriculograms during atrial pacing stress tests. Digital subtraction angiography was chosen to study these issues because it allows 3 to 4 left ventriculograms to be recorded with the same amount of contrast typically used to obtain 1 standard film-based angiogram. Therefore, this technique can be used to answer physiologic questions concerning the time course of ischemic changes induced by atrial pacing in humans.[20]

In patients who undergo catheterization for chest pain and are found to have normal coronary arteries at angiography, the results of this study indicate that the response to atrial pacing at the peak heart rate consists of a marked decrease in end-diastolic and end-systolic volume. Global EF increased at least 2 percentage points in 6 of the 11 patients (55%) and did not change (i.e., was within 2 percentage points of the value at rest) in another 4 (36%). In 1 patient, EF decreased 3 percentage points. This decrease in EF is considered to be a false-positive response and has also been found during bicycle exercise in normal patients with a high EF at rest, in elderly patients and in patients with LV hypertrophy. In the normal patient with a high EF at rest, end-systolic volume is reduced at rest. Therefore, when the heart rate increases during pacing, the decrease in end-diastolic volume may not be matched by a proportional decrease in end-systolic volume, with the result that the EF inappropriately falls. However, in most

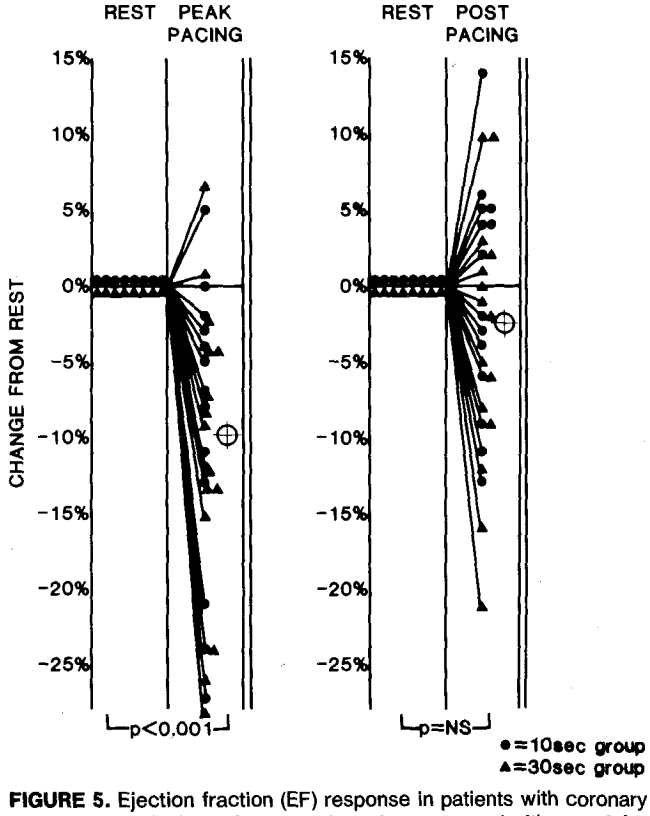

FIGURE 5. Ejection fraction (EF) response in patients with coronary artery disease. **Left,** studies at peak pacing compared with rest; **right,** postpacing study compared with rest. The mean decrease in EF was more pronounced during peak pacing, whereas the mean change in EF during the postpacing study was not significantly different (NS) from the value at rest.

patients with CAD, EF increases or does not change significantly during atrial pacing.

The volumetric changes in the patients without CAD quickly revert to rest values within 5 to 10 seconds after pacing is stopped, as does the double product. In addition, LVEDP is similar at rest or after the postpacing left ventriculograms. Thus, preload, afterload, LV volumes and heart rate were similar at rest and in the postpacing studies in patients without CAD.

In the 29 patients with angiographically documented CAD, mean EF decreased by 10 percentage points during peak atrial pacing and returned toward the value at rest either 5 to 10 or 30 seconds after pacing. Based on the findings in the patients without CAD, we defined a positive pacing test as a decrease in EF of 2 or more percentage points. When this criterion was applied to the 29 patients with CAD, a positive response occurred in 25 patients (86%). Thus, for the entire group of 40 patients studied, the sensitivity of the atrial pacing test performed at the peak pacing was 86% and the specificity was 91%. However, if the left ventriculograms performed 5 to 10 or 30 seconds after the peak pacing rate is used, then the sensitivity of the post pacing study diminishes to 58% and the specificity remains 91%. These data indicate that ventricular function should be evaluated at peak paced heart rate in order to detect ischemia induced by atrial pacing.

The change in end-systolic volume during pacing is also a good means of distinguishing patients with CAD from those without CAD. The decrease in EF that is seen in patients with CAD apparently occurs because of the inability of the myocardium to appropriately decrease the end-systolic volume during pacing and not because of differences in the changes in end-diastolic volume at peak pacing.

The data from this study were used to compare a qualitative analysis of segmental wall motion to the angiographic presence and distribution of coronary artery narrowing. In 25 of the 29 patients (86%), there was a good correlation between the presence of CAD and the development during pacing (23 patients) or after pacing (2 patients) of segmental wall motion contraction abnormalities in the myocardial distribution of the affected artery. In 4 patients significant wall motion abnormalities did not develop during or after the stress of atrial pacing. One of these patients had a 50% stenosis of the right coronary artery, which may not have been hemodynamically significant under the stress of atrial pacing to a heart rate of 142 beats/min. A second patient had an 80% stenosis of the mid-left anterior descending artery, but was not adequately stressed because of reluctance of the angiographer, who stopped the pacing study at a heart rate of 112 beats/min before chest pain developed. Another patient had disease only in the circumflex artery (75% stenosis). Because only RAO left ventriculograms were recorded in this patient, lateral wall motion abnormalities may have developed that were not visualized in the RAO projections. The fourth patient had 70% stenoses in both the right and circumflex coronary arteries. Although global EF decreased mildly, from 73% to 69%, no distinct wall motion abnormality was seen during or after pacing, again perhaps because the RAO projection was used.

This analysis of segmental wall motion highlights some of the issues associated with interpreting the effects of inducing myocardial ischemia by atrial pacing. First, the patient must be adequately stressed to ensure that ischemia develops. Second, if lesions in the circumflex distribution are to be analyzed, the left ventriculogram should be performed in the left anterior oblique projection so that the lateral wall appears on the periphery of the ventriculogram. Third, an angiographically significant lesion must be adequately defined hemodynamically. Even if a stenosis is measured as 50% or greater diameter narrowing, its functional significance may depend on several factors. Atrial pacing and other stressful interventions are independent tests of the functional significance of coronary stenosis and the result of the stress test can be used as a supplementary examination in the description of atherosclerotic lesions.

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