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Coronary Artery Atherosclerosis: Severity of the Disease, Severity of Angina Pectoris and Compromised Left Ventricular Function

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SUMMARY **To determine if the severity of angina pectoris and the degree of altered left ventricular function correlated with the severity and extent of the underlying coronary artery disease, a coronary scoring system was derived. The system was based on the severity of luminal diameter narrowing and weighted according to the usual flow to the left ventricle in each coronary vessel. Thus, the most weight was given to the left main coronary artery, followed by the left anterior descending, circumflex, and right coronary arteries. The resultant number was an indicator of the overall severity of the obstructive coronary artery disease. A coronary arterial system with no obstructive disease was scored as zero and the greater the degree of obstructive disease present, the higher the coronary score. From 202 subjects, four groups were evaluated: group 1 — coronary score = 0.5–4.5 (n = 10); group 2 — coronary score = 10.5–12.5 (n = 11); group 3 — coronary score = 17.5–20.5 (n = 11); and group 4 — coronary score = 25.0–36.0 (n = 11). All subjects had coronary artery bypass surgery and had preoperative and 1-year postoperative cardiac catheterization, including atrial pacing to maximal heart rate. The groups could not be separated on the basis of angina frequency, resting heart rate, cardiac index, left ventricular end-diastolic pressure, peak paced left ventricular end-diastolic pressure, dP/dt, V max, left ventricular end-diastolic volume index, left ventricular end-systolic volume index, stroke volume index, ejection fraction or mean circumferential fiber shortening velocity. Thus, based on this study, the severity of coronary artery disease does not statistically correlate with the frequency of angina pectoris or produce a predictable degree of altered left ventricular function. The frequency of angina pectoris cannot be used to predict prognosis or the adequacy of myocardial revascularization.**

IF THE SEVERITY of angina pectoris correlated with the severity of the underlying coronary artery atherosclerosis, one could better judge the appropriate time for invasive diagnostic and therapeutic interventions. One could also ascertain the prognosis of the disease, as it has been adequately shown that prognosis is in part dependent on the severity of coronary artery disease (CAD).[1-10] It would also be helpful if the severity of the CAD correlated with the degree of abnormal left ventricular function. This would be especially helpful in the postoperative state, when one could very simply ascertain the adequacy of the operation and judge the state of left ventricular function.

To evaluate these possibilities, a coronary score was derived to assess the degree of CAD. The coronary score was then related to the frequency of angina pectoris to determine if the extent of CAD could be predicted based on the clinical presentation of angina pectoris. It was also compared with resting and paced left ventricular function measurements to determine if the severity of CAD had a predictably deleterious effect on left ventricular function. The coronary score was also determined at 1 year after coronary bypass surgery and was again compared with the frequency of

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angina pectoris and the degree of altered left ventricular function.

Methods

Two hundred two patients with CAD underwent direct coronary revascularization surgery at the Erasmus University Hospital. All were evaluated before and 1 year after surgery. A coronary score was derived in all subjects, and 43 were further evaluated. All subjects were free of mitral valvular regurgitation and other cardiac valvular lesions and did not undergo left ventricular aneurysm resection. Thirty-seven subjects (86%) were taking therapeutic doses of β-blocking agents, which were discontinued at least 48 hours before cardiac catheterization. No patient was in clinical congestive heart failure. A detailed angina history was obtained and graded according to the frequency of attacks: 5 = more than five attacks/day; 4 = one to five attacks/day; 3 = more than one attack/week and less than one attack/day; 2 = seldom; and 1 = no angina. The angina history was obtained by an impartial observer not involved with the hemodynamic or angiographic evaluation. Angina pectoris was defined as substernal chest pressure that occurred with exercise and was relieved by rest and/or nitroglycerin. If the patient experienced a similar pain that occurred at rest, this was also considered to be angina pectoris. The angina frequency was determined at the time of the hospitalization for cardiac catheterization and represented the average frequency at that time and only attacks considered to be typical anginal attacks were counted. Most patients were on maximal medical therapy with nitrates and propranolol at that time. Cardiac catheterization and coronary arteriography was performed only when the patient was considered a "medical failure." Nine patients (21%) had a history of a preoperative myocardial infarction confirmed by left ventriculography. All of the infarctions were located on the diaphragmatic surface of the left ventricle. No additional myocardial infarctions occurred at postoperative cardiac catheterization as judged by serial ECGs and comparison of the pre- and postoperative left ventricular angiogram. Before surgery all 43 subjects underwent a complete right- and left-heart catheterization and selective coronary arteriography by the Sones-Shirey[11] or Judkins technique.[12] No premedication was given and nitroglycerin was not used prophylactically. One year later, the above procedures were repeated, except that four patients (9%) did not have right-heart catheterization. All subjects had selective injections of the aortocoronary bypass grafts.

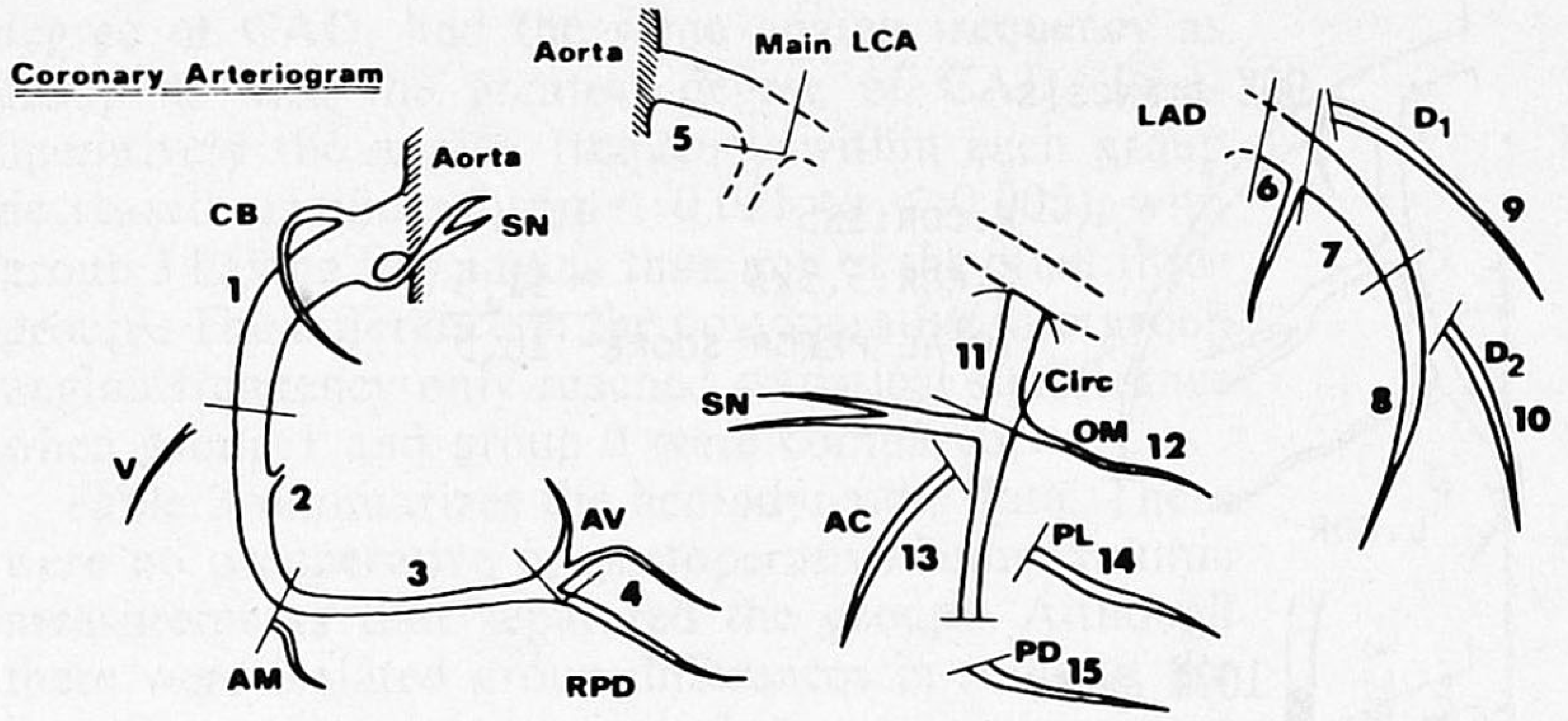
Preoperative and postoperative resting hemodynamic data included heart rate (HR), cardiac index by thermodilution,[13] and left ventricular end-diastolic pressure (LVEDP). Right atrial pacing was performed as previously described for this laboratory.[14] Briefly, a bipolar pacing electrode was positioned high in the right atrium. The left ventricular pressures were recorded by either a Dallons-Telco MMC or a Millar Instruments tip-manometer catheter. Right atrial pacing was commenced at 10 beats/min above the resting HR. Every 3 minutes the HR, LVEDP, peak dP/dt and V_{max} were recorded. The paced rate was then increased by 10 beats/min. This was continued until a rate of 180 beats/min, the onset of chest pain, or Wenckebach atrioventricular conduction (seven of 43 preoperatively, 13 of 43 postoperatively). The hemodynamic data at the highest HR before chest pain or Wenckebach atrioventricular conduction was used in the data calculations. All hemodynamic data were processed by a PDP 11-10 computer as previously described.[15-17] All subjects had pre- and postoperative simultaneous biplane left ventricular cineangiograms. The cineangiogram preceded the coronary arteriograms. Data derived from this portion of the study included the left ventricular end-diastolic volume index (LVEDVI), left ventricular end-systolic volume index (LVESVI), stroke volume index, ejection fraction (EF) and mean Vcf, as previously described.[18, 19]

Derivation of the Coronary Score

The purpose of the coronary score was to quantitate the severity of the underlying CAD. The different coronary vessels carry different volumes of blood to the left ventricle, and the coronary score was derived to take this into account. A review of the literature revealed the average left ventricular mass in the average male (free wall plus septum) is approximately 155 g.[20]

Numerous studies have been done to determine the actual flow to the left ventricle.[21-29] Most equate flow to muscle mass rather than to actual volume of flow.[23-29] Based on these studies, the average coronary blood flow is approximately 96 ml/100 g of left ventricular muscle, or essentially 150 ml/min to the average male left ventricle. Studies have shown that in the right dominant coronary artery system the right coronary artery supplies approximately 16% and the left coronary artery approximately 84% of the flow to the left ventricle.[20, 24, 29] Thus, the left coronary artery carries approximately five times the amount of flow to the left ventricle compared with the right coronary artery. It also has been shown that in the usual right-dominant coronary system, the flow in the left coronary artery is 66% into the left anterior descending (LAD) and 33% into the circumflex coronary artery.[20] Thus, the LAD carries approximately 3.5 times and the circumflex 1.5 times as much blood as the right coronary artery. This then was the basis for assigning different weighting values to the different coronary arteries. The coronary arteries were subdivided into segments according to the scheme devised by the American Heart Association.[30] The right coronary artery does not supply blood to the left ventricle until the posterior descending branch is given off, so only the segment with the most severe disease was analyzed. However, the LAD and circumflex coronary arteries were further subdivided and weighting factors were assigned to each segment (fig. 1).

Subjects who had a left-dominant coronary artery



SEGMENT	RIGHT DOMINANT	LEFT DOMINANT
1		
2	1.0	0.0
3		
4		
5	5.0	6.0
6	3.5	3.5
7	2.5	2.5
8	1.0	1.0
9	1.0	1.0
10	0.5	0.5
11	1.5	2.5
12	1.0	1.0
13	0.5	1.5
14	0.5	1.0
15	0.0	1.0

% LUMINAL DIAM. REDUCTION	WEIGHTING FACTOR
70-89	1
90-99	3
100	5

FIGURE 1. *Coronary artery segments and the weighting factor assigned to each segment in the right-dominant and left-dominant coronary artery system. Also, the percent luminal diameter reduction of the coronary artery segment and the weighting factor assigned to the specific percentage reduction. AC = atrial circumflex; AV = atrioventricular nodal branch; AM = acute marginal branch; CB = conus branch; CIRC = circumflex branch; D_1 and D_2 = diagonal branches; LAD = left anterior descending; LCA = left coronary artery; OM = obtuse marginal; PD = posterior descending branch; PL = posterolateral branch; RPD = right posterior descending; SN = sinus node branch; V = ventricular branch.*

system were graded differently. In this situation, the left ventricle receives its blood supply from the left coronary artery, so the right coronary artery was not weighted and its value was assigned to the main left coronary artery. Subsequently the circumflex coronary artery was also weighted heavier than in the right-dominant system (fig. 1).

The degree of stenosis was also considered in deriving the coronary score. If the vessel was totally occluded, the coronary artery segment value was multipled by 5.0. If there was a 90–99% occlusion, it was multiplied by 3.0. For a 70–89% obstruction, it was multiplied by 1.0. The segmental scores were then added to derive the total coronary score. A score of zero would indicate no obstructive CAD, and the higher the score, the greater the obstruction (fig. 1). Because the true significance of collateral flow is unknown, this was not taken into account in the derivation of the coronary score.[31] In deriving the postoperative coronary score, a vessel segment was considered normal if it was supplied by a patent aortocoronary bypass graft that was free of occlusive disease (fig. 2). If the aortocoronary bypass graft was occluded, the score was derived from the degree of disease in the native coronary system; that is, the score was derived as if no graft had been placed.

Four groups were analyzed, based on the preoperative coronary score: group 1 — 0.5–4.5; group 2 — 10.5–12.5; group 3 — 17.5–20.5; and group 4 — 25.0–36.0. The selection of the patients for each group was based entirely on their coronary score. Subjects whose coronary score fell within the predetermined score for that group were included in the study. Evidence of a preoperative myocardial infarction was found in none of the 10 group 1 patients, one of the 11 group 2 patients, five of the 11 group 3 patients and three of the 11 group 4 patients. No subject had evidence of an interim myocardial infarction at follow-up cardiac catheterization.

Statistical analysis of the pre- and postoperative data within the same group was performed using the paired *t* test, and the comparison between groups using the unpaired *t* test.[32]

Results

Table 1 and figure 3 are a summary of the preoperative and postoperative coronary scores for each group. Postoperatively the score was lower within each group than preoperatively, and was statistically significant for groups 3 and 4 ($p < 0.005$). The postoperative coronary score was not significantly different between groups.

Table 1 and figure 4 are summaries of the preoperative and postoperative angina frequency. The preoperative angina frequency was not significantly different between groups. Group 1, with the least

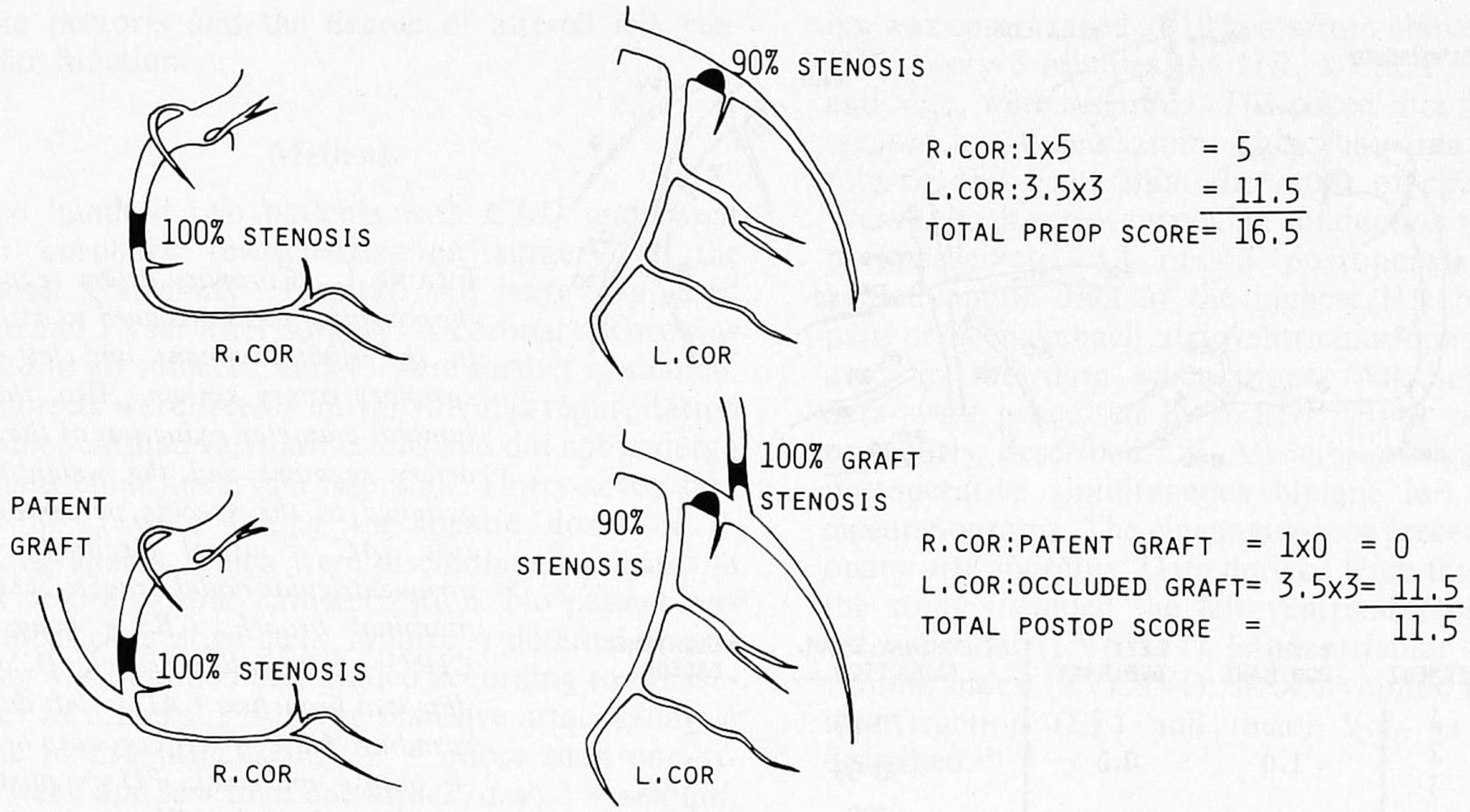


FIGURE 2. *The preoperative and postoperative coronary scoring system. (A) 100% stenosis of the right coronary artery (R. Cor.) (coronary segment weight = 1; stenosis weight = 5) and left anterior descending coronary, segment 6 (coronary segment weight = 3.5; stenosis weight = 3). One hundred percent (B) stenosis of right coronary with a patent aortocoronary artery bypass graft (no score) and left anterior descending coronary, segment 6, with an occluded graft. This segment (6) is thus scored the same as if no graft had been placed. L. Cor. = left coronary artery.*

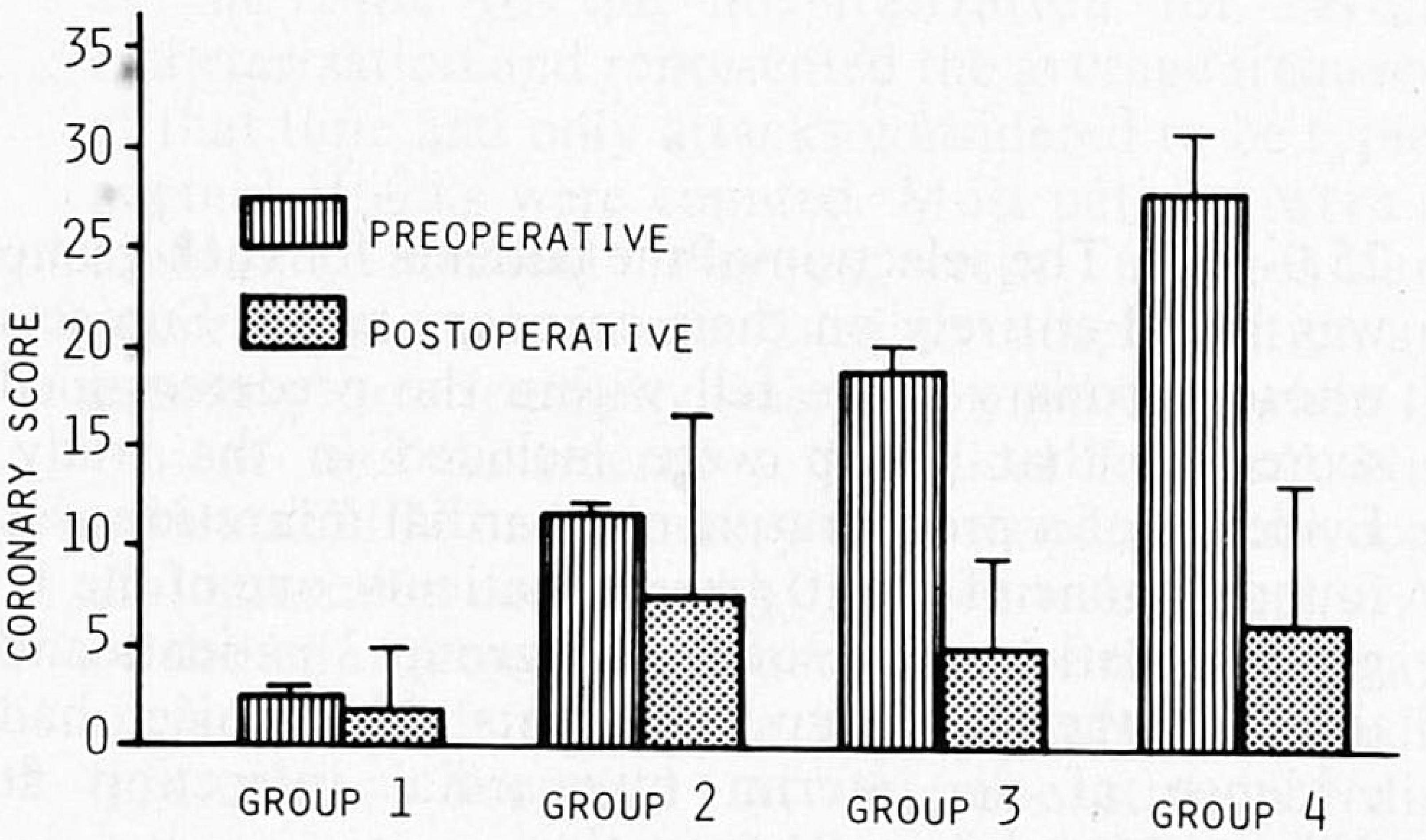


FIGURE 3. *Preoperative and postoperative coronary score with 1 standard deviation.*

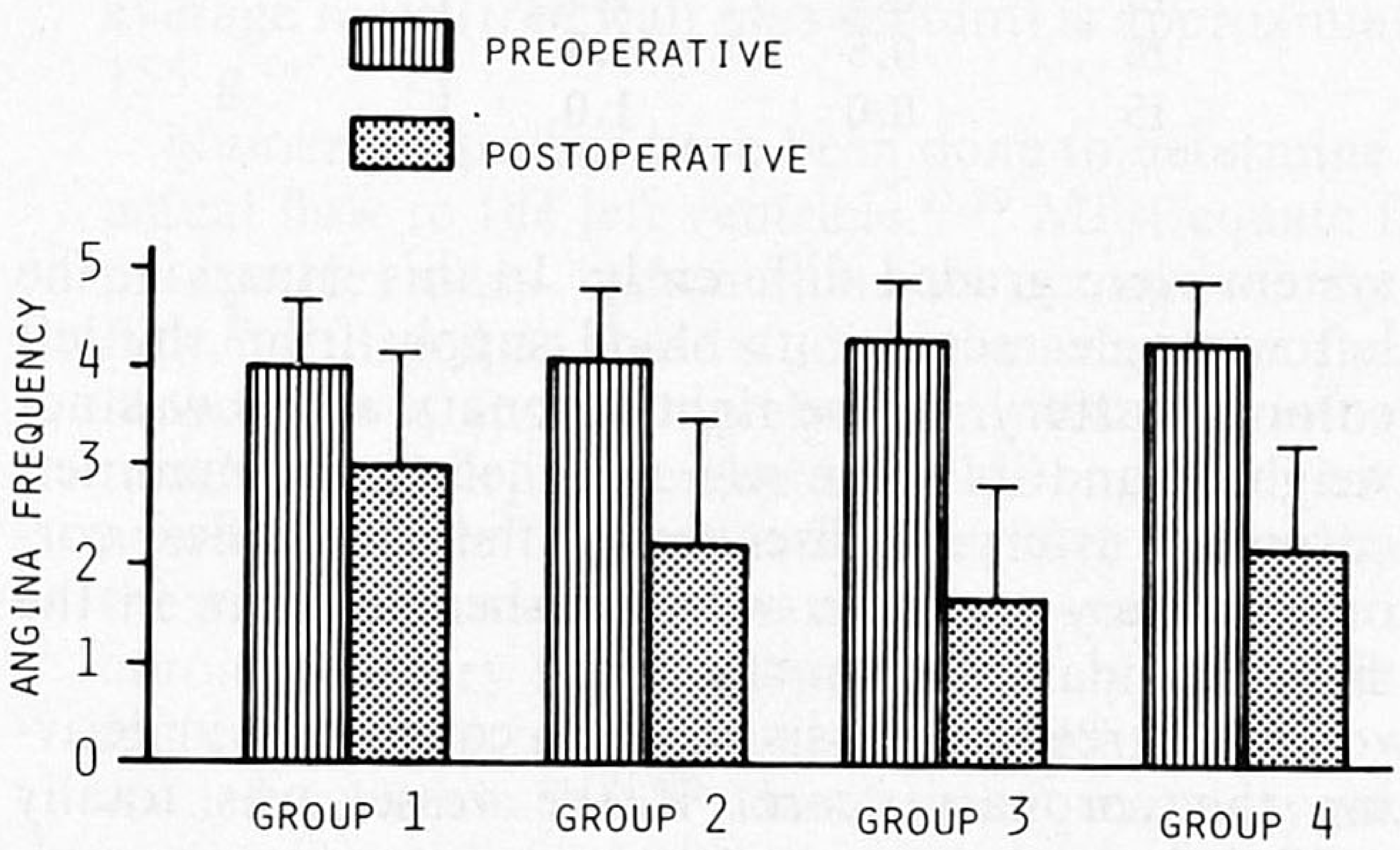


FIGURE 4. *Preoperative and postoperative angina frequency with 1 standard deviation.*

TABLE 1. *Preoperative and Postoperative Coronary Score and Angina Frequency*

	Group 1		Group 2		Group 3		Group 4	
	Preop	Postop	Preop	Postop	Preop	Postop	Preop	Postop
Coronary score								
Mean ± SD	2.55 ±0.93	2.00 ±3.08	11.73 ±0.65	7.64 ±9.22	19.23 ±1.06	5.18 ±4.42	27.86 ±3.18	6.41 ±6.95
Range	0.5–4.5	0.0–4.0	10.5–12.5	0.0–23.0	17.5–20.5	0.0–13.0	25.0–36.0	0.0–250
p		NS		NS		< 0.005		< 0.005
Angina frequency								
Mean ± SD	4.0 ±0.7	3.0 ±1.1	4.1 ±0.7	2.2 ±1.3	4.3 ±0.6	1.6 ±1.2	4.3 ±0.6	2.2 ±1.1
Range	3–5	1–4	3–5	1–5	3–5	1–4	3–5	1–4
p		< 0.025		< 0.005		< 0.005		< 0.005

degree of CAD, had the same angina frequency as group 4, with the greatest degree of CAD. Postoperatively the angina frequency within each group decreased significantly ($p < 0.025$ to < 0.005), with group 3 having less angina than any of the other three groups. The difference in the postoperative intergroup angina frequency only reached statistical significance when group 1 and group 3 were compared.

Table 2 summarizes the hemodynamic data. There were no preoperative or postoperative hemodynamic measurements that separated the groups. Although there were isolated group differences in some of the hemodynamic measurements, there was no consistent measurement that allowed for group separation (table 3).

Discussion

A coronary score was derived that reflected the expected volume of blood flow to the respective coronary artery and the severity of luminal diameter narrowing. The subjects all had aortocoronary bypass surgery and selective coronary arteriography both pre- and postoperatively. In deriving the postoperative coronary score, a diseased vessel segment was considered to be normal if it was supplied by a patent graft in which there was no occlusive disease. We could then compare the subjects' preoperative and postoperative coronary score, angina frequency and left ventricular hemodynamics.

A distinct separation of groups by coronary score, and thus severity of CAD, was deemed important to accentuate any differences in angina frequency and altered left ventricular hemodynamics. To maximize any potential difference between the groups, an atrial pacing stress test was performed at preoperative and postoperative cardiac catheterizations. This allowed us to evaluate the left ventricular hemodynamic variables of LVEDP, dP/dt and V_{max} under cardiac stress, as well as the usual resting hemodynamic variables at cardiac catheterization.

The coronary score within each group preoperatively was significantly different ($p < 0.0005$). However, there was no difference in the frequency of angina pectoris when any one group was compared with any other group. Thus, the group with the lowest coronary score (the least CAD) could not be separated on a historical basis from the group with the highest coronary score (the most severe occlusive CAD) (fig. 5).

Postoperatively the coronary score within each group decreased, especially in groups 3 and 4, so there was no statistical difference in coronary score between groups after surgery. There was also a significant intragroup decrease in the postoperative angina frequency ($p < 0.025$ to < 0.0005). When the postoperative intergroup angina frequency was compared, only groups 1 and 3 differed from each other. Thus, as in the preoperative state, there was no consistent correlation between the coronary score and the frequency of angina pectoris (fig. 6).

There was no consistent intergroup difference in angina frequency, so we could not correlate this

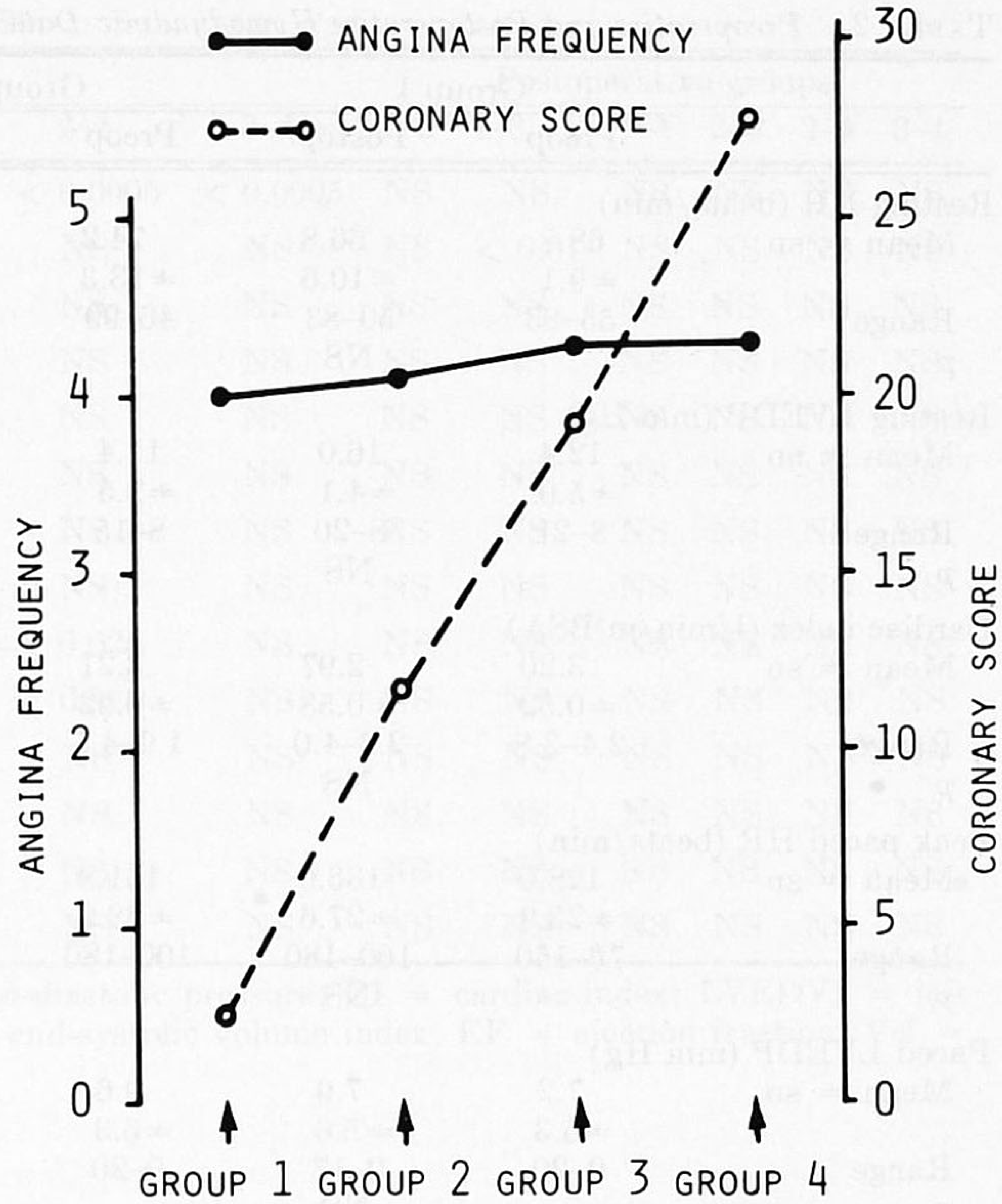


FIGURE 5. *Comparison of the preoperative angina frequency and coronary score. There was a large difference in coronary score with minimal difference in angina frequency.*

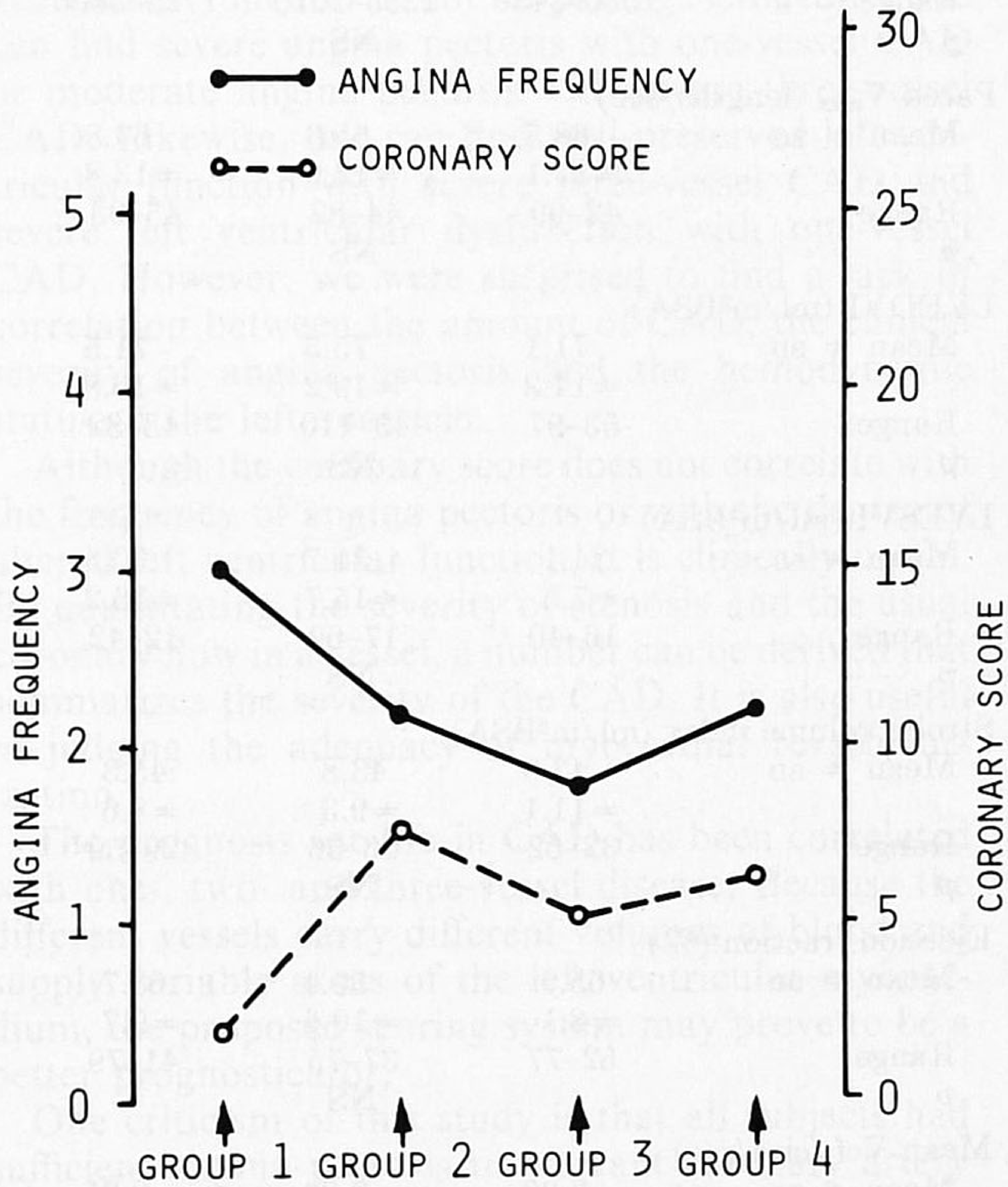


FIGURE 6. *Comparison of the postoperative angina frequency and coronary score.*

variable with the hemodynamic variables. There was no correlation between the coronary score and the hemodynamic measurements. Although there were occasional differences between the groups, no one

TABLE 2. *Preoperative and Postoperative Hemodynamic Data*

	Group 1		Group 2		Group 3		Group 4	
	Preop	Postop	Preop	Postop	Preop	Postop	Preop	Postop
Resting HR (beats/min)								
Mean ± SD	68.8	66.8	74.2	73.2	76.2	73.4	75.7	74.4
	±9.1	±10.6	±13.3	±5.1	±13.8	±13.8	±13.9	±14.6
Range	55–83	50–83	46–99	62–80	60–100	58–108	56–103	54–99
p		NS		NS		NS		NS
Resting LVEDP (mm Hg)								
Mean ± SD	12.4	16.0	11.4	15.7	14.4	12.1	15.9	16.2
	±5.0	±4.1	±2.5	±4.5	±7.9	±9.0	±7.8	±3.1
Range	3–21	8–20	8–15	10–23	10–34	8–24	8–31	12–21
p		NS		< 0.01		< 0.05		NS
Cardiac index (l/min/m²BSA)								
Mean ± SD	3.20	2.97	3.21	3.11	3.28	3.11	3.21	3.07
	±0.52	±0.58	±0.92	±0.63	±0.80	±0.68	±0.67	±0.82
Range	2.4–3.8	2.2–4.0	1.9–4.5	2.2–4.2	2.2–4.3	2.2–4.3	2.0–4.4	2.0–4.6
p		NS		NS		NS		NS
Peak paced HR (beats/min)								
Mean ± SD	128.0	136.0	151.8	138.2	124.5	148.2	133.6	138.2
	±23.9	±27.6	±28.2	±32.5	±34.7	±23.6	±25.4	±23.6
Range	70–150	100–180	100–180	80–180	60–170	110–180	100–170	100–170
p		NS		NS		< 0.025		NS
Paced LVEDP (mm Hg)								
Mean ± SD	7.2	7.9	9.6	9.9	14.4	8.4	14.8	12.1
	±5.3	±5.6	±6.3	±7.2	±7.9	±4.6	±11.4	±8.2
Range	0–20	0–17	0–20	0–22	6–31	3–19	1–36	2–23
p		NS		NS		< 0.025		NS
Paced peak dP/dt (mm Hg/sec)								
Mean ± SD	2099	2037	2226	2109	1994	2186	1945	1797
	±584	±408	±533	±446	±675	±874	±469	±402
Range	1530–3480	1265–2610	1725–3605	1465–2670	1070–3230	1300–4245	1330–2670	1370–2570
p		NS		NS		NS		NS
Paced V_{max} (lengths/sec)								
Mean ± SD	68.5	65.0	69.8	66.0	58.4	67.4	57.8	57.8
	±17.1	±12.9	±13.8	±14.1	±19.7	±17.7	±12.8	±12.8
Range	42–99	44–82	45–93	45–89	34–90	52–110	43–84	34–78
p		NS		NS		< 0.025		NS
LVEDVI (ml/m²BSA)								
Mean ± SD	71.1	75.5	71.5	74.4	83.4	85.3	85.5	97.3
	±14.3	±19.2	±10.8	±14.4	±24.1	±28.8	±19.2	±26.5
Range	53–97	49–110	43–82	59–103	49–123	63–153	59–112	58–117
p		NS		NS		NS		NS
LVESVI (ml/m²BSA)								
Mean ± SD	24.1	31.7	30.5	29.3	43.4	39.3	40.2	46.9
	±7.5	±15.7	±16.2	±11.3	±21.5	±23.1	±22.0	±30.8
Range	16–40	17–69	12–42	16–53	13–71	15–93	18–78	21–132
p		NS		NS		NS		NS
Stroke volume index (ml/m²BSA)								
Mean ± SD	47.0	43.8	45.3	45.1	40.1	45.4	45.4	50.4
	±11.1	±9.3	±8.6	±6.7	±9.2	±9.8	±10.7	±14.4
Range	32–62	26–56	29–59	36–50	22–55	23–60	34–68	21–74
p		NS		NS		NS		NS
Ejection fraction (%)								
Mean ± SD	65.9	59.4	63.7	61.7	50.9	55.4	55.4	55.0
	±8.1	±10.6	±9.7	±8.5	±14.8	±13.0	±15.9	±15.6
Range	52–77	37–75	41–79	49–74	24–73	36–77	30–71	13–66
p		NS		NS		NS		NS
Mean Vcf (circ/sec)								
Mean ± SD	0.90	0.83	0.95	0.93	0.71	0.87	0.82	0.77
	±0.25	±0.22	±0.27	±0.24	±0.33	±0.20	±0.35	±0.22
Range	0.51–1.35	0.35–1.10	0.45–1.56	0.53–1.32	0.11–1.26	0.56–1.13	0.32–1.50	0.20–0.93
p		NS		NS		NS		NS

Abbreviations: HR = heart rate; LVEDP = left ventricular end-diastolic pressure; BSA = body surface area; LVEDVI = left ventricular end-diastolic volume index; LDESVI = left ventricular end-systolic volume index; Vcf = velocity of circumferential fiber shortening.

TABLE 3. *Statistical Analysis of Preoperative and Postoperative Groups*

	Preoperative groups						Postoperative groups					
	1–2	1–3	1–4	2–3	2–4	3–4	1–2	1–3	1–4	2–3	2–4	3–4
Coronary score	< 0.0005	< 0.0005	< 0.0005	< 0.0005	< 0.0005	< 0.0005	NS	NS	NS	NS	NS	NS
Angina frequency	NS	NS	NS	NS	NS	NS	NS	< 0.01	NS	NS	NS	NS
Resting HR	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Resting LVEDP	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Resting CI	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Peak paced HR	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Paced LVEDP	NS	< 0.0125	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Paced dP/dt	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Paced V_{max}	NS	NS	NS	NS	< 0.025	NS	NS	NS	NS	NS	NS	NS
LVEDVI	NS	NS	NS	NS	< 0.025	NS	NS	NS	NS	NS	NS	NS
LVESVI	NS	< 0.01	< 0.025	NS	NS	NS	NS	NS	NS	NS	NS	NS
SVI	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
EF	NS	< 0.01	NS	< 0.025	NS	NS	NS	NS	NS	NS	NS	NS
Mean Vcf	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

Abbreviations: HR = heart rate; LVEDP = left ventricular end-diastolic pressure; CI = cardiac index; LVEDVI = left ventricular end-diastolic volume index; LVESVI = left ventricular end-systolic volume index; EF = ejection fraction; Vcf = velocity of circumferential fiber shortening.

hemodynamic measurement separated one group from another. Thus, the degree of CAD did not correlate with the left ventricular function.

There have been other attempts to correlate the number of diseased coronary vessels with altered left ventricular hemodynamics, angina severity and mortality. However, in none of these studies was there an attempt to weight the individual coronary arteries on the basis of the volume of flow to the left ventricle. Falsetti et al.[33] found no correlation between the number of vessels involved and the LVEDP, LVEDV and residual fraction. Hamby et al.[34] compared one-vessel CAD with diffuse CAD. They found a decreased LVEDP and LVEDV with an increased EF in the one-vessel CAD group compared with the diffuse CAD group. Our study compares favorably with Falsetti et al. Although we measured more variables and included an atrial pacing stress test, we could not correlate the degree of CAD and altered left ventricular function. Our low coronary score group is comparable to the one-vessel CAD group of Hamby et al., but we could not confirm their finding of a difference in hemodynamics in this group. Rowe et al.[35] measured resting coronary blood flow and hemodynamic variables. They could not correlate these variables with the degree of coronary artery disease. No studies have correlated a coronary score with hemodynamic data derived from atrial pacing. Levine et al.[36] devised an index of vascularization and compared left ventricular function before and after coronary artery bypass surgery. In their study, EF and segmental wall motion improved if the patient had an improved vascularization index. Although in our study, groups 3 and 4 had marked improvement in their coronary score (vascularization index), we could not detect any significant changes in their EFs.

That the degree of CAD does not correlate with the severity of angina pectoris or the degree of altered left ventricular function is not surprising. Clinically, one can find severe angina pectoris with one-vessel CAD or moderate angina pectoris with severe three-vessel CAD. Likewise, one can find well-preserved left ventricular function with severe three-vessel CAD and severe left ventricular dysfunction with one-vessel CAD. However, we were surprised to find a lack of correlation between the amount of CAD, the clinical severity of angina pectoris and the hemodynamic status of the left ventricle.

Although the coronary score does not correlate with the frequency of angina pectoris or with the degree of altered left ventricular function, it is clinically useful. By quantitating the severity of stenosis and the usual coronary flow in a vessel, a number can be derived that summarizes the severity of the CAD. It is also useful in judging the adequacy of myocardial revascularization.

The prognosis for life in CAD has been correlated with one-, two- and three-vessel disease. Because the different vessels carry different volumes of blood and supply variable areas of the left ventricular myocardium, the proposed scoring system may prove to be a better prognosticator.

One criticism of this study is that all subjects had sufficient angina pectoris to warrant coronary artery bypass surgery. However, this does not negate the lack of correlation of the coronary score with the frequency of the angina pectoris and the hemodynamic status. A second criticism involves inclusion of patients with a previous myocardial infarction. This actually makes our findings more significant. There were no previous infarctions in the group with the lowest coronary score and this should have accentuated any hemodynamic differences between this group and the other groups, all of which had subjects with previous infarction. However, no such differences were found.

From this study we conclude: (1) The clinical frequency of angina pectoris does not accurately reflect the degree of underlying CAD; (2) measurements of left ventricular function do not correlate with the frequency of angina pectoris or with the severity of the underlying CAD; (3) the postoperative improvement in a patient's angina pectoris does not mean that the patient has a revascularized left ventricular myocardium or improved left ventricular hemodynamics; and (4) one cannot predict a patient's postoperative success or prognosis based on postoperative angina frequency.

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