# Accelerated Angina Pectoris

## Clinical, Hemodynamic, Arteriographic, and Therapeutic Experience in 85 Patients

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SUMMARY

Eighty-five patients with accelerated (preinfarction) angina are reported. Six suffered acute myocardial infarction awaiting catheterization and coronary angiography, so were not studied. Seventy-nine had coronary arteriography and other angiographic and hemodynamic studies. Fifteen of these 79 patients had normal coronary arteriograms; 64 had significant coronary artery obstruction. The clinical manifestations in 64 abnormal patients did not differ from those with normal arteriograms. Hemodynamic abnormalities correlated with the severity of arteriographic abnormalities. Of 70 patients with coronary artery disease, including the six not studied because of infarction, 48 were treated surgically with a mortality of 12.5%. Mortality for those 22 patients treated without surgery was 27%. Mortality could be correlated with certain risk factors: (1) congestive heart failure; (2) more than three-vessel coronary disease; (3) left ventricular end-diastolic pressure $>$ 12 mm Hg; (4) cardiac index $<$ 2.7 liters/min/m$^2$; (5) stroke index $<$35 ml/beat/m$^2$; (6) estimated cardiac work (mean aortic pressure $\times$ cardiac index) $<$240 units; and (7) ejection fraction $<$0.50. Cardiac catheterization and angiography were performed without major complications in 97% of patients.

**Additional Indexing Words:**

Coronary artery surgery Impending myocardial infarction Myocardial revascularization
Preinfarction angina

UNSTABLE, accelerated angina pectoris, often referred to as preinfarction angina, impending myocardial infarction, or as the intermediate syndrome, was first described by Wearn.[1] Subsequent papers have discussed clinical features, natural history, and medical therapy, but the patient populations of these studies were not similar.[2-8] Each author defined the condition differently. Virtually no studies report hemodynamic and/or angiographic data, and only recently have a few of these patients had myocardial revascularization in an effort to relieve pain, prevent myocardial infarction, and spare myocardium.

We have seen 85 patients with the clinical syndrome of accelerated angina pectoris. This is a report of our experience.

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Accelerated angina pectoris is a clinical syndrome characterized by increasing frequency and severity of anginal attacks in a patient either with previously stable angina or without previous angina. The severity and frequency progress to incapacity, and rest or nocturnal angina is quite common. The pain often is associated with ischemic S-T- and T-wave changes, but ECG changes diagnostic of acute myocardial infarction are lacking. The serum enzyme values customarily are normal.

### Materials and Methods

#### Case Material

During the 21-month period from May, 1970 through January, 1972 the clinical diagnosis of accelerated angina pectoris was established in 85 patients, and all were scheduled for cardiac catheterization and coronary angiography. This group includes virtually all patients seen with this diagnosis during the period of study. Many of the patients were referred from other hospitals. The patients were included in the study only after confirmation of the diagnosis by one or more of the authors. Many patients referred during this period were excluded because their angina pectoris was stable or their chest pain was atypical and probably of noncardiac origin. Patients were included in this study on the basis of admission data.

Six patients developed myocardial infarction while awaiting cardiac catheterization and were not studied. One of these individuals died. Cardiac catheterization was performed in 79 patients. Two of 79 patients studied suffered acute myocardial infarction on the way to the laboratory. Seventy-seven patients were still in the preinfarction phase at the onset of catheterization, two of whom progressed to infarction during the procedure.

### Methods

Coronary arteriography was performed on 79 patients. The Sones technic was utilized in 69, the Judkins technic in seven, and both technics in three. Films were considered adequate for analysis in 71 patients; in eight, film quality was not adequate for evaluation of collateral circulation. Significant coronary obstruction was defined as an area showing greater than 60% narrowing of the coronary artery lumen. The vessels included for study were the right coronary artery (RCA), the main left coronary artery (LCA), the left anterior descending artery (LAD) and its diagonal branch, and the left circumflex artery and its obtuse marginal branch. Thus, six vessels were included in analysis, in that the diagonal and obtuse marginal branches are often major vessels.

Left heart catheterization was performed in 77 patients and right heart catheterization in 69. Pressures were determined through a fluid-filled catheter system, utilizing Hewlett-Packard 1280 pressure transducers and Hewlett-Packard model 350-1100C carrier preamplifiers. Pressures were recorded at rest.

Cardiac output was obtained by the Fick method in 69 patients. Arterial and venous $O_2$ saturations and contents were determined by either Van Slyke analysis or with a model 182 IL cooximeter. Stroke index was obtained by dividing cardiac index by heart rate. A simple estimate of cardiac work (in units) was determined as the product of cardiac index and mean aortic pressure. Peak left ventricular dp/dt was obtained with the aid of a Hewlett-Packard 8814 A derivative computer.

Left ventricular angiography was performed in 57 patients. In most cases 40 cc of meglumine diatrizoate (Renografin-76) was injected, with the patient at the 30° right anterior oblique position. A gross assessment of left ventricular contractility was made in all of these 57 patients. An ejection fraction was determined in 50 patients. Utilizing the method of Kasser and Kennedy[9] for single-plane cineangiographic study, left ventricular volumes were estimated.

## Results

### Clinical Findings

The 85 patients were separated into 70 "abnormal" patients and 15 "normal" patients on the basis of coronary arteriograms. The average age of the abnormal group was 53 years with a range of 30–70 years, while the average age of our normal group was 45 years with a range of 35–57 years (table 1). There were 49 males and 18 females in the abnormal group and 12 males and three females in the normal group. There was a previous history of ischemic heart disease in virtually all patients. All patients had a history of exertional angina pectoris. Thirty of the abnormal group (43%) and four (27%) of the normal group had been treated for a myocardial infarction. The patterns of accelerated angina included pain at rest, pain of increasing intensity, nocturnal angina, diaphoresis, and/or postprandial angina. Virtually all patients had pain at rest or pain of increasing intensity and/or frequency. Nocturnal angina was present in 55% of patients, diaphoresis in 35%, and postprandial angina in 18%.

**Table 1**

*Clinical Data for 85 Patients with Accelerated Angina Pectoris*

| Parameter | Abnormal coronary angiogram (N = 70) | Normal coronary angiogram (N = 15) |
|---|---|---|
| Average age (yr) | 53 | 45 |
| Range (yr) | 30 – 70 | 35 – 57 |
| Sex | 49 M | 12 M |
| | 18 F | 3 F |
| Average blood pressure (mm Hg) | 138/84 | 138/75 |
| Hypertension: | 12 | 3 |
| Systolic > 140 | 12 | 3 |
| Systolic and diastolic >140/90 | 11 | 2 |
| Atrial gallop ($S_4$) | 56 | 7 |
| Ventricular gallop ($S_3$) | 10 | 3 |

The duration of stable angina prior to acceleration averaged approximately 2 years in both groups. The duration of accelerated angina pectoris averaged 17 days in the abnormal group, ranging from less than 1 day to approximately 3 months. Thirty patients had had an accelerated phase for less than 2 weeks. Duration of accelerated angina was similar in the normal group, averaging 14 days.

There were 22 patients with exertional angina for less than 20 weeks and accelerated angina for less than 2 weeks. Four of these had myocardial infarction before cardiac catheterization could be done and were not studied. Of the remaining 18 patients studied, nine were found to have single-vessel disease. Fourteen of the 18 had completely normal hemodynamics, and 16 of the 18 had no demonstrable collateral coronary artery circulation.

Physical examination did not permit discrimination between patients with abnormal and normal arteriograms. As seen in table 1, the blood pressure in the abnormal group averaged 138/84 mm Hg and in the normal group, 138/75 mm Hg. There

**Table 2**

*Electrocardiographic Findings*

| ECG | Abnormal pt (N = 70) | Normal pt (N = 15) |
|---|---|---|
| Normal | 13 (19%) | 11 |
| Ischemic ST-T wave changes | 22 (31%) | 4 |
| Acute myocardial infarction | 6 | 0 |
| Old myocardial infarction | 21 (30%) | 0 |
| Left ventricular hypertrophy | 4 | 0 |
| Left-axis deviation | 2 | 0 |
| Right bundle-branch block | 2 | 0 |

were 12 patients with systolic hypertension in the abnormal and three in the normal group. Eleven patients in the abnormal group and two of the normal group had systolic and diastolic hypertension, defined as pressures greater than 140/90 mm Hg. Fifty-six of the 70 abnormal patients and seven of the 15 normal patients had an audible atrial gallop sound. Ventricular gallop sounds were heard in 10 and three patients of the respective groups.

Chest X-rays were not helpful in discriminating abnormal from normal subjects since they were virtually all normal. Sixty-four of 70 patients had normal chest X-rays; six showed some degree of cardiomegaly.

The results of electrocardiography are listed in table 2. Nineteen percent of the abnormal group had normal electrocardiograms. Ischemic ST-T wave changes were by far the most common electrocardiographic finding. Such changes were also present in four normal patients. Electrocardiographic evidence of previous myocardial infarction was present in 30% of patients in the abnormal group.

### Angiography

Of 79 patients having coronary arteriography, 15 (19%) were found not to have significant coronary artery obstructions (12 were completely normal arteriographically and three had up to 30% obstruction of the proximal right coronary artery). These patients comprised our group of "normal" patients. Among 64 "abnormal" patients with significant obstruction (table 3), 18 had single-vessel disease, 14 had two diseased vessels, 17 had three diseased vessels, and 15 had more than three diseased vessels. Arteriographically 86% of patients were considered to have at least one operable vessel. A vessel was considered operable if it was visibly patent distal to the obstruction and if it was not diffusely diseased. Five of nine patients deemed inoperable had more than three-vessel involvement. The LAD artery was most often involved (50 patients). The RCA had significant obstruction in 47, the circumflex in 30, the diagonal in 17, the obtuse marginal in 11, and the main LCA in nine.

Table 3 demonstrates that visible collateral circulation was found in only one of 18 patients with single-vessel disease, a significantly lower incidence compared to seven of 14 with two-vessel disease ($P < 0.01$), to 10 of 17 with three-vessel disease ($P < 0.001$), or to 12 of 15 with more than three-vessel disease ($P < 0.001$). Though a speculative conclusion, the low incidence of collaterals in patients with single-vessel disease might explain the rapid clinical course of many of these patients.

Left ventricular contractility, as estimated grossly from left ventricular angiograms, correlated directly with the number of vessels involved (table 3). It is interesting that 47% of patients with only one vessel involved had abnormality of left ventricular contraction even though they had no evidence of prior myocardial infarction.

### Hemodynamic Findings

The physiologic findings (reported in mean values ± standard deviation) are listed in table 4 according to the number of vessels involved. There are no significant differences among groups with three or fewer vessels involved except for a lower ejection fraction in those with three involved compared to those with one ($P < 0.05$). However, the group with more than three-vessel involvement

**Table 3**

*Abnormal Coronary Arteriographic Findings*

| Significant obstruction | No. | Operable No. | Operable % | Inoperable | Collaterals No. | Collaterals % | Abnormal ventriculogram (%) |
|---|---|---|---|---|---|---|---|
| One vessel | 18 | 16 | 89 | 2 | 1 | 5.5 | 47 (7/15) |
| Two vessels | 14 | 13 | 93 | 1 | 7 | 50 | 78 (7/9) |
| Three vessels | 17 | 16 | 94 | 1 | 10 | 59 | 86 (12/14) |
| > Three vessels | 15 | 10 | 67 | 5 | 12 | 80 | 83 (10/12) |
| Total | 64 | 55 | 86 | 9 | 30 | 47 | 76 |

**Table 4**

*Hemodynamic Findings Related to the Number of Coronary Arteries with Significant Obstruction*

| Vessels involved | LVEDP (mm Hg) | LVEDP post LV (mm Hg) | A-V $O_2$ diff (vol %) | CI (liters/min/m²) | SI (ml/beat/m²) | Cardiac work (units) | Peak dp/dt (mm Hg/sec) | EF |
|---|---|---|---|---|---|---|---|---|
| Normal | 9.5 | 22.0 | 4.3 | 3.1 | 40.4 | 299 | 1631 | 0.64 |
| | ± 5.1 | ± 7.8 | ±0.9 | ±0.6 | ±10.9 | ± 75 | ± 199 | ±0.14 |
| One | 9.4 | 26.6 | 4.6 | 2.8 | 37.6 | 272 | 1543 | 0.64 |
| | ± 3.9 | ± 7.4 | ±1.0 | ±0.7 | ± 7.7 | ± 70 | ± 308 | ±0.10 |
| Two | 7.2 | 20.9 | 4.5 | 2.8 | 39.0 | 271 | 1527 | 0.60 |
| | ± 3.5 | ± 7.8 | ±0.9 | ±0.5 | ± 8.5 | ± 65 | ± 397 | ±0.10 |
| Three | 12.8 | 32.11 | 4.9 | 2.9 | 39.9 | 286 | 1540 | 0.45 |
| | ±10.2 | ±13.8 | ±2.6 | ±0.8 | ±11.1 | ± 78 | ± 383 | ±0.23† |
| >Three | 13.3 | 35.9 | 4.8 | 2.6 | 34.2 | 245 | 1401 | 0.41 |
| | ± 6.0†‡ | ± 9.6*†‡ | ±0.7 | ±0.4* | ± 8.9 | ± 39* | ± 369* | ±0.18*†‡ |

*Significantly different from normal.
†Significantly different from one vessel.
‡Significantly different from two vessel.

showed evidence of significant hemodynamic abnormalities, namely higher left ventricular end-diastolic pressure, and lower cardiac index, cardiac work, peak left ventricular dp/dt, and ejection fraction. Since right heart pressures, including pulmonary wedge pressure, showed no significant variation between groups, they are not detailed.

### Complications of Catheterization

No deaths occurred during the course of the study (table 5). The one bout of ventricular fibrillation occurred in one of the two patients who suffered myocardial infarction during catheterization. Both the patients were taken to surgery immediately following the procedure and underwent successful bypass procedures. Two other patients who had infarcted just before catheterization survived study and went to surgery, although one died shortly after surgery. The other complications listed were transient and of no real significance. No major complications occurred in 77 of the 79 studies (97%).

**Table 5**

*Complications of Cardiac Catheterization and Arteriography (79 Patients)*

| Complications | No. |
|---|---|
| Major: | |
| Death | 0 |
| MI | 2 |
| Ventricular fibrillation | 1 |
| Minor and transient: | |
| Angina | 7 |
| Hypotension | 4 |
| Conduction abnormality during coronary arteriography | |
| Two A-V block | 2 |
| Left bundle-branch block | 1 |
| Right bundle-branch block | 2 |
| Left anterior hemiblock | 10 |
| Left posterior hemiblock | 9 |
| No complications | 55 (75%) |
| No major complications | 77 (97%) |

### Clinical Course

Of the 70 patients in this study with coronary artery disease, 48 were treated surgically and 22 were treated without surgery. No attempt at randomization of mode of therapy was made.

### Medical Management

The 22 patients not operated on included six who sustained acute myocardial infarction awaiting catheterization, seven who refused surgery although they were deemed operable, and nine considered inoperable due to distal disease in obstructed vessels.

Medical therapy included variable periods of bed rest, monitoring in most cases, sedation, and various drugs. Nitrates and propranolol were used in most patients. Digitalis and antiarrhythmic agents were utilized for complications of congestive heart failure and/or arrhythmias. Since the literature is replete with references extolling the benefits of anticoagulation, heparin and/or coumarin agents were given to most patients.

Despite aggressive medical therapy, 13 (59%) of these 22 patients suffered acute myocardial infarction during the period of observation, and six died (27%). Another nine patients similarly treated suffered infarction while awaiting surgery. If these nine are considered to have infarcted on medical management, the incidence of infarction on medical management was 71% (22 of 31 patients). Comparison with surgical treatment, however, is not valid since these are not truly comparable groups.

### Surgical Management

Of 48 patients treated surgically, nine had infarcted during the preoperative period and were operated upon as emergencies. Thirty-nine were still preinfarction at the time of their procedure.

Table 6 lists the procedures performed. There was an inverse relationship between the number of vessels with significant obstruction in a patient and the feasibility of total surgical revascularization. With three exceptions, all patients had at least one direct revascularization procedure. In two patients only bilateral internal mammary artery implantation could be performed, and in one patient with severe coronary artery calcification no procedure could be performed. The latter patient survived surgery but died suddenly 2 months later. These three patients are included in the surgical group because they were considered candidates for direct revascularization by all available evidence prior to operation.

Surgical mortality was 10% (four of 39) for those not recently infarcted and 22% (two of nine) for those with preoperative infarction, for a total mortality rate of 12.5%. Mortality correlated with the completeness of myocardial revascularization. Of 18 patients with complete revascularization (bypass to all arteriographically obstructed vessels) only one (5.6%) died, where six of 30 (20%) with incomplete revascularization died.

Myocardial infarction occurred subsequent to surgery in 10 (25%) of 39 patients still preinfarction at the time of operation. This incidence is high, though lower than the infarction rate of 59% for those patients treated without surgery. Again, because of lack of randomization, this comparison is not really accurate.

### Late Follow-up

There were 41 surgical survivors and 16 medical survivors with follow-up. In the surgical group 24 patients (59%) were angina-free, and an additional 12 had improvement of angina. Angina was the same or worse in five patients, and five had episodes of congestive heart failure. All of the patients with persistent angina and all patients with heart failure had more than three-vessel disease. The medically treated patients fared less well; only three patients were free of angina (19%), and three others showed improvement.

### Discussion

Prodromal symptoms often precede acute myocardial infarction, but may not be recognized as such by the patient or physician. Feil, and later Sampson and Eliaser[2, 3] reported that 50% of their patients with acute infarction could recall prodromal symptoms. Others have reported prodromal pain in from 10 to 65% of infarction patients.[8, 10–14] Thus, many patients with myocardial infarction progress through a preinfarction phase of accelerated angina.

Authors describing patients with "preinfarction angina" have reported subsequent myocardial infarction during medical management in 3–93% of patients, and mortality in as many as 73% of patients.[2–8, 11, 15] Several studies have evaluated anticoagulant therapy in this clinical syndrome and almost without exception patients treated with anticoagulants fared better than did those in control groups.[5–8, 16] Earlier reports can be criticized because definitions of "preinfarction angina" varied from author to author, but particularly because patients were included without arteriographic confirmation. Our definition can be criticized, and we have inadequate controls, but arteriography was performed and this gives weight to our findings. If 19% of patients with accelerated anginal pain pattern have normal coronary anatomy, earlier studies must have included similar patients with resultant false-low morbidity and mortality rates.

An accelerated anginal pain pattern in patients with normal coronary arteriograms is difficult to explain. One of our 15 "normal" patients suffered a nontransmural myocardial infarction 4 days after study, and another had a form of cardiomyopathy,

**Table 6**

*Revascularization Feasibility*

| Vessels involved | Pt | Obstructed arteries | Bypass | | Implantation | Gas endarterectomy |
|---|---|---|---|---|---|---|
| | | | No. | % | | |
| One | 14 | 14 | 14 | 100 | 2 | |
| Two | 12 | 24 | 15 | 63 | 4 | 1 |
| Three | 12 | 36 | 15 | 42 | 3 | 1 |
| >Three | 10 | 45 | 13 | 29 | 3 | |
| Total | 48 | 119 | 57 | 48 | 12 | 2 |

but the remaining 13 had no significant cardiovascular abnormalities, and their subsequent clinical course has been benign. The clinical manifestations in this "normal" group differed in no significant manner from those with coronary artery disease, except that ECG patterns of old myocardial infarction were always associated with coronary obstructions.

The data from patients with coronary artery disease were evaluated to see if any clinical parameters had prognostic significance. There were no significant correlations between eventual outcome and pain patterns, history of old myocardial infarctions, blood pressures, chest X-rays, and/or electrocardiograms. The only clinical finding with prognostic significance was the presence of congestive heart failure. In patients with congestive failure the overall mortality rate was 70% (seven of 10) compared to 10% (six of 60) for the remainder ($P < 0.001$).

Few references are found to the arteriographic and/or hemodynamic findings in patients with accelerated angina.[17, 18] Our data show close correlation to exist between the number of vessels involved and the presence of certain hemodynamic abnormalities.

Analysis of our data yielded five hemodynamic (risk) factors related to mortality (table 7): (1) left ventricular end-diastolic pressure (LVEDP) > 12 mm Hg; (2) cardiac index (CI) < 2.7 liters/min/m$^2$; (3) stroke index (SI) < 35 ml/beat/m$^2$; (4) cardiac work <240 units; and (5) ejection fraction < 0.50 (50%).

There was a significantly lower overall mortality of 9.1% and a surgical mortality of 5.7% in those patients with a normal LVEDP compared to incidences of 39% ($P < 0.01$) and 36% ($P < 0.01$) in similar groups with LVEDP above 12 mm Hg. Among patients with normal LVEDP, the mortality rate was lower with surgical than nonsurgical management, though the difference did not reach statistical significance.

Similar relationships hold true to a greater or lesser degree when one compares patients with a normal cardiac index to those with a CI of less than 2.7 liters/min/m$^2$. Among those with normal CI, patients treated surgically had a 3.7% mortality, significantly lower than the 27% mortality for the medical group ($P < 0.05$).

Similar relationships hold true upon comparing patients with a stroke index of 35 ml/beat/m$^2$ to those whose SI is below this figure, though this cutoff point is not quite as significant.

**Table 7**

*Mortality Related to Hemodynamic Risk Factors*

| Variable | Total no. | Deaths No. | Deaths % | Total no. | Deaths No. | Deaths % | P |
|---|---|---|---|---|---|---|---|
| LVEDP (mm Hg) | | *<12* | | | *>12* | | |
| Surgery | 35 | 2 | 5.7 | 11 | 4 | 36 | <0.01 |
| No surgery | 9 | 2 | 22 | 8 | 3 | 38 | |
| Total | 44 | 4 | 9.1 | 19 | 7 | 37 | <0.01 |
| CI (liters/min/m$^2$) | | *2.7 or >* | | | *<2.7* | | |
| Surgery | 27 | 1 | 3.7 | 14 | 5 | 36 | <0.01 |
| | | | (*P* <0.05) | | | | |
| No surgery | 11 | 3 | 27 | 4 | 2 | 50 | |
| Total | 38 | 4 | 10.5 | 18 | 7 | 39 | <0.05 |
| SI (ml/beat/m$^2$) | | *35 or >* | | | *<35* | | |
| Surgery | 27 | 2 | 7.4 | 14 | 4 | 29 | |
| No surgery | 10 | 2 | 20 | 5 | 3 | 60 | |
| Total | 37 | 4 | 10.8 | 19 | 7 | 37 | <0.05 |
| Cardiac work (units) | | *240 or >* | | | *<240* | | |
| Surgery | 29 | 2 | 6.9 | 12 | 4 | 33 | <0.05 |
| No surgery | 12 | 2 | 17 | 3 | 3 | 100 | <0.05 |
| Total | 41 | 4 | 9.8 | 15 | 7 | 47 | <0.01 |
| EF | | *0.50 or >* | | | *<0.50* | | |
| Surgery | 20 | 1 | 5 | 11 | 3 | 27 | |
| No surgery | 9 | 1 | 11 | 5 | 3 | 60 | |
| Total | 29 | 2 | 6.8 | 15 | 6 | 40 | <.01 |

**Table 8**

*Mortality Related to Hemodynamic Risk Factors*

| Treatment | < 4 risk factors | | | 4 or > risk factors | | | P |
|---|---|---|---|---|---|---|---|
| | Total no. | Deaths No. | Deaths % | Total no. | Deaths No. | Deaths % | |
| Surgery | 37 | 2 | 5.4 | 5 | 4 | 80 | <0.001 |
| No surgery | 12 | 2 | 17 | 4 | 3 | 75 | <0.05 |
| Total | 49 | 4 | 8.2 | 9 | 7 | 78 | <0.001 |

A cardiac work (mean aortic pressure × cardiac index) of 240 units or above is associated with a significantly lower mortality whether one considers patients treated medically, surgically, or all together. There is a trend toward better survival among the surgical patients than the medical whether the cardiac work is abnormal or not.

For ejection fraction, 50% was found to be a significant dividing line. Those patients with an ejection fraction above this figure had an overall mortality of 6.8%, versus a 40% mortality for those patients whose left ventricles ejected less than 50% of their diastolic volumes.

Even more striking is the difference in survival between patients who had at least four abnormal risk factors compared to those with fewer than four risks. It appears that this high-risk group will do very poorly regardless of the form of treatment, and so surgery is probably hopeless (table 8).

On the other hand, those patients without any of these risks have the best results (no deaths in 18 such patients).

Arteriographically, the presence of obstruction in more than three coronary arteries can be considered a surgical risk factor, in that operative mortality in this group was 40% compared to 5.3% for those with three or fewer involved arteries ($P < 0.01$).

The medical therapy of our patients was standard, employing nitrates, propranolol, and anticoagulation. Some may contend our medical group was weighted with poor-risk patients since it included patients considered inoperable. Yet, it also included seven patients refusing surgery, and nine other patients could be included who were operable but infarcted awaiting surgery.

A few reports have appeared relating experience with direct revascularization surgery for "impending myocardial infarction."[17–22] The most impressive results are those of Auer et al.[19] who reported 0% mortality in 41 patients. Our results are not as good but illustrate that surgical results depend not only on excellent surgical technic, but also on the severity of the coronary artery disease, on the completeness of myocardial revascularization, and on the degree of left ventricular dysfunction as reflected by the hemodynamic studies and left ventricular angiograms.

The follow-up periods for the survivors of hospitalization are obviously too short to permit valid conclusions, but the studies suggest that surgical patients have an improved quality of life in the early postoperative period. Whether this trend will persist will only be known after longer observation of larger numbers of patients. A controlled study must be performed to answer these questions.

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