

Coronary angioplasty of the unstable angina related vessel in patients with multivessel disease

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This study is a retrospective analysis of the efficacy of percutaneous transluminal coronary angioplasty of the ischaemia-related vessel in patients with unstable angina. Forty-three patients had multivessel disease with dilatation of the ischaemia-related vessel only (group I; partial revascularization) while 111 patients had single vessel disease only (group II; total revascularization). The initial success rate in both groups was identical (88 versus 88%). The need for emergency coronary artery bypass surgery was similar in the two groups (group I 12% versus group II 9%; NS). The total post PTCA myocardial infarction rate (despite urgent CABG) was also similar in the two groups (group I 9% versus group II 10%; NS).

The results of electrocardiographic exercise testing and Thallium-201 scintigraphy provide objective evidence for incomplete revascularization in group I. The maximum workload achieved was lower, and the frequency of exercise induced angina, ST-segment depression and reversible perfusion defect was higher than in group II. Moreover, at 6 months follow-up the recurrence rate of angina pectoris rate was higher in group I than in group II (29% versus 16% $P < 0.05$).

It is concluded that dilatation of the ischaemia related vessel only in patients with unstable angina and multivessel disease is as effective in the management of the acute phase of unstable angina as is dilatation of the ischaemia related vessel in patients with single vessel disease. However, due to only partial revascularization the recurrence rate of angina pectoris is higher.

Percutaneous transluminal coronary angioplasty is effective in the treatment of selected patients with stable angina^[1] and has been advocated in patients with unstable angina^[2-5], particularly when they are refractory to pharmacological therapy. Although potentially hazardous because of coexistent ischaemia, the early results of percutaneous transluminal coronary angioplasty indicate an acceptable risk-benefit ratio^[2-5]. However, the majority of patients with unstable angina have multivessel disease^[6] and multiple dilatations during the same percutaneous transluminal coronary angioplasty procedure carries an increased risk. It is therefore, as yet, not widely practised^[7]. In this retrospective study we contrast our experience with dilatation of the ischaemia related vessel only, in

patients with multivessel disease and unstable angina, i.e. partial revascularization, with the efficacy of total revascularization, i.e. dilatation of the affected vessel, in patients with single vessel disease only. Objective evidence of relief of overall myocardial ischaemia was sought by electrocardiographic exercise testing and Thallium-201 scintigraphy.

Patient selection

Between February 1983 and the end of December 1984, percutaneous transluminal coronary angioplasty was performed in a consecutive series of 154 patients who satisfied the following criteria:

(1) Chest pain at rest lasting for at least 15 min accompanied by reversible electrocardiographic ST-T changes, and no evidence of myocardial infarction (CPK less than twice normal, no Q wave development);

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- (2) adequate left ventricular function ($EF > 0.45$);
 (3) potentially suitable for coronary artery bypass surgery;
 (4) an ischaemia-related vessel with a lesion suitable for percutaneous transluminal coronary angioplasty.

These 154 patients were selected from a total of 2243 patients admitted to our CCU in 1983 and 1984. Three hundred and forty five patients of the 2243 (16%) had unstable angina. The extent of coronary artery disease and the management of these patients is shown in Table 1. All patients were monitored in the coronary care unit. Initial treatment included administration of beta-adrenergic blockers, calcium-antagonists and intravenous nitroglycerine. Sixty-nine patients were stabilized and had elective angiography followed by percutaneous transluminal coronary angioplasty. Eighty-five patients remained unstable and had emergency angiography with percutaneous transluminal coronary angioplasty. In patients with multivessel coronary artery disease, only the ischaemia-related vessel was dilated. Thus two study groups were formed: group I—43 patients with multivessel disease and dilatation of the ischaemia related vessel, 'partial revascularization' and group II: 111 patients with single vessel disease and dilatation of that vessel 'total revascularization'. In patients with multivessel disease the ischaemia related vessel was identified by the correlation of the electrocardiographic changes during the attack with the angiographic findings. Electrocardiographic changes in leads V_1-V_5 were related to coronary artery lesions of the left anterior descending artery (LAD) while electrocardiographic

changes in leads V_6, I, aVL or II, III, aVF reflected either a lesion of the circumflex (CX) or of the right coronary artery (RCA). The baseline clinical characteristics of group I and II are shown in Table 2. A history of previous myocardial infarction was more frequent in group I than in group II (53% versus 35%; $P < 0.05$).

Angiography was performed in multiple views and included hemiaxial angulations. The angiograms were interpreted by at least two experienced angiographers. A significant stenosis was defined as one causing at least a 50% luminal diameter narrowing. The angiographic characteristics of the ischaemic related vessels in group I and II were comparable as shown in Table 3. The characteristics of the non-dilated vessels and lesions are shown in Table 4.

Percutaneous transluminal coronary angioplasty was performed with preformed guiding catheters and Schneider or Meditech balloon dilating catheters using the method described by Grüntzig^[1]. Before the procedure 250 mg acetylsalicylic acid and 100 mg heparin *i.v.* were given: during the procedure a continuous drip of Rheomacrodex was given. To prevent or exclude coronary spasm, nifedipine or nitroglycerin was given into the coronary artery^[8]. Initial balloon inflation pressure was 2.0 atmospheres, with subsequent inflations ranging to 12 atmospheres. The mean, transstenotic gradient was calculated on-line before and after each dilatation, and divided by the mean aortic pressure to give a gradient index. Percutaneous transluminal coronary angioplasty was considered successful if the luminal diameter narrowing was reduced to less than 50% or the

Table 1 Selection of patients with unstable angina pectoris and dilatation of only the ischaemia-related vessel in multivessel or single vessel disease (total number of admissions to CCU, Thoraxcenter, from 1 Jan 1983 to 1 Jan 1985 was 2243)

Unstable angina	No. of pts	Extent of coronary artery disease				
		0 V	1 V	2 V	3 V	LM
Refractory to pharmacological treatment						
emergency PTCA	85	—	63	13	9	—
emergency CABG	67	—	3	14	33	17
Initially stabilized with pharmacological treatment						
elective PTCA	69	—	48	17	3	1
elective CABG	59	—	1	14	38	6
pharmacological treatment	65	11	7	17	22	8
Total no of pts	345	11	122	75	105	32

Table 2 Baseline clinical characteristics of patients with unstable angina and dilatation of ischaemia-related vessel in multivessel or single vessel disease

	Multivessel disease (N=43)	Single vessel disease (N=111)
Mean age (years)	58.0	57.5
range (years)	(39–72)	(35–74)
Percent males	86	82
Prior myocardial infarction	53%	35% (0.05)
History unstable angina:		
Recent onset AP at rest	21%	15%
Worsening of pre-existing AP	39%	46%
Post MI (within 4 weeks)	39%	39%
Initially stabilized with pharmacological treatment	49%	43%

Table 3 Angiographic characteristics of patients with unstable angina and dilatation of ischaemia-related vessel in multivessel or single vessel disease

	Multivessel disease (N=43)	Single vessel disease (N=111)
Ischaemia-related vessel		
LAD	63%	68%
RCA	12%	17%
CX	19%	15%
graft	5%	—
left main stem	2%	—
Left ventricular ejection fraction, mean \pm SD	0.56 \pm 0.11	0.59 \pm 0.10

gradient index to <0.20 , with stabilization or abolition of angina and no progression to myocardial infarction or death^[9]. After the procedure, all patients were monitored for 24 hours in the coronary care unit where electrocardiograms and enzyme levels were measured. The patients were usually discharged 3 days after the procedure. They were kept on treatment with nifedipine 40–60 mg daily and enterosarine 500 mg daily during a period of 6 months.

During the procedure a surgical team was available to carry out CABG. A perioperative myocardial infarction was diagnosed when a new Q-wave developed in the 12-lead electrocardiogram.

Clinical follow-up information was obtained, either from a visit to the outpatient clinic or by information obtained from the referring physician. Cardiac death, occurrence of myocardial infarction, and recurrence of angina pectoris were

Table 4 Characteristics of stenosis of non-dilated vessels in 43 patients with unstable angina, multivessel disease and PTCA of ischaemia-related vessel only

Status of non-dilated vessels	No.	%
Total no. of vessels	56	100
Technically suitable for CABG	55	98
Technically suitable for PTCA	27	48
Non-dilated; LAD	6	11
Non-dilated; RCA	23	41
Non-dilated; CX	27	48
Non-dilated 1-vessel	30	54
Non-dilated 2-vessel	13	23
Total occlusion	15	28
Stenosis to infarcted myocardium	12	21
Distal stenosis	15	27
Non-severe stenosis	35	62
Filled by collaterals	23	41

tabulated. A symptom limited exercise test was performed on a bicycle ergometer with stepwise increments in loads of 20 W each minute. Heart rate was measured at one and blood pressure at 2-min intervals with continuous electrocardiographic monitoring with three orthogonal leads X, Y, Z. An ischaemic response was defined as at least 1 mm of ST-segment depression, 0.08 s after the J point. The maximum workload achieved was expressed as a percentage of the normal workload predicted for age, sex and length.

For the exercise thallium 201 myocardial scintigraphy, 1.5 m Ci of Thallium 201 was injected intravenously approximately 1 min prior to maximum exercise. Sequential imaging was performed immediately after exercise and again 4 h later in the anterior, left anterior oblique 45°, and left anterior oblique 65° views. A Searle Phogamma V camera with a 25 cm field of view and a low energy, all purpose, parallel hole collimator, interfaced to a DEC Gamma 11 nuclear medicine computer system was used for data collection and processing^[10]. Basically, early and late circumferential profiles were computed within the automatically detected contour of the left ventricle following background subtraction. From the early and late profiles a washout circumferential profile was computed as percent washout from the early post exercise profile. Analysis of the Tl uptake, redistribution and washout profiles was performed by comparison of these data from a given patient with a set of normal reference curves. These normal values were defined by the upper and lower 10th percentile at each point of the profile derived from normal subjects. Defects with redistribution were considered to represent exercise induced ischaemia. Persistent defects without redistribution were considered as

scars. For prediction of the presence of significant disease in the left anterior descending artery, the anterolateral region in the anterior view, the septum in left anterior oblique 45° and the anterior region in left anterior oblique 65° were studied. Significant disease in the left circumflex artery and/or right coronary artery was predicted when the abnormal profile occurred in the postero septal and inferior region of the anterior view, in the postero lateral region in left anterior oblique 45° or in the inferior region left anterior 65°. If an abnormal region was limited to the apex, no prediction of the location of disease was made.

Repeat angiograms were obtained in multiple views including hemiaxial angulation and were interpreted by observers without knowledge of the patient's clinical status. Restenosis was defined as an increase of the luminal diameter narrowing of the dilated lesion to more than 50%.

Chi-square statistics were used to compare proportions.

Results

The coronary angioplasty success rate was not significantly different in multivessel disease versus single vessel disease (Table 5). All 5 patients with unsuccessful coronary angioplasty and multivessel disease underwent emergency coronary bypass surgery; nevertheless a myocardial infarction developed in 9% (4/43). Emergency bypass surgery was necessary in 10 of the 13 patients with single vessel disease and unsuccessful PTCA; nevertheless 8 patients developed a myocardial infarction. Three other patients had a technically successful PTCA, but developed a myocardial infarction nonetheless.

Table 5 Success rate PTCA of ischaemia-related vessel in patients with unstable angina and single- or multi-vessel disease

Ischaemia related vessel	Multivessel disease		Single vessel disease	
	Total No.	Success rate (%)	Total No.	Success rate (%)
LMCA	1	100	—	—
LAD	27	93	75	90
LCX	8	75	17	94
RCA	5	80	19	79
Bypass	2	100	—	—
Total	43	88% (38/43)	111	88% (98/111)

Table 6 Cardiac events after PTCA of ischaemia-related vessel in unstable angina with single or multivessel disease

	Multivessel disease		Single vessel disease	
<i>Successful PTCA</i>				
Follow-up period (months)	6	12	6	12
No of pts followed-up	38	30	98	75
Death	—	1	—	—
Myocardial infarction	—	—	—	1
Angina pectoris	11	—	14	2
Re-PTCA	3	—	8‡	2
CABG	5*	—	3	—
Pharm. treatment	3†	—	3	—
<i>Unsuccessful PTCA</i>				
Follow-up period months	6	12	6	12
No. of pts followed-up	5	4	13	10
Angina pectoris	-1	—	-2	—
Pharm. treatment	-1	—	-2	—

*In 2 patients CABG was performed due to undilated stenosis; †all 3 patients suffered from angina pectoris due to undilated stenosis; ‡in 1 patient redilatation was performed due to progression of disease in another segment.

This latter was not caused by reocclusion or occlusion of a side branch or prolonged angina during the procedure; apparently the onset of infarction was related to the attacks of chest pain immediately prior to the procedure. The reasons for failure and complication in group I were total occlusion of the vessel during attempts to cross in 2 patients and dissection with slow run off after dilatation in 3 patients. The reasons for failure and complications in group II were total vessel occlusion during the attempts to cross the lesion in 5 patients, dissection with slow run off after dilatation in 5 patients and broken guide wire embedded in a side branch in 1 patient.

Exercise electrocardiographic testing and thallium 201 scintigraphy was performed 2.6 ± 2.3 months after a successful percutaneous transluminal coronary angioplasty. Both tests were available in 82% (31/38) of the patients with multivessel disease and in 89% (87/98) of the patients with single vessel disease. The baseline clinical characteristics of both groups with exercise testing were comparable. The maximum heart rate achieved was 138 ± 22 beats min^{-1} in multivessel disease and 144 ± 22 beats min^{-1} in single vessel disease (NS). The number of patients who achieved 90% of the maximum workload predicted for age, sex and height was lower in patients with multivessel

disease than in single vessel disease (61% vs 75%; NS). The frequency of exercise induced ischaemic ST-segment depression (39% vs 15%; <0.05), exercise induced angina pectoris (16% vs 7%; <0.05) and of a reversible perfusion defect (33% vs 23%; NS) was higher in multivessel disease than in single vessel disease. The frequency of a reversible defect in the myocardial area perfused by the dilated vessel was similar in both groups; in multivessel disease 17% and in single vessel disease 23%. However, a reversible perfusion defect was also present in a non-dilated area in 20% of the patients with multivessel disease. The frequency of cardiac events during follow-up after successful and unsuccessful percutaneous transluminal coronary angioplasty is shown in Table 6. All patients were followed for at least 6 months; nearly all cardiac events occurred within that period. Recurrence of angina pectoris within 6 months after successful percutaneous transluminal coronary angioplasty was more frequent in multivessel disease (11 patients) than in single vessel disease (13 patients) (29% vs 16%; $P < 0.05$). In 5 patients with multivessel disease angina pectoris was due to a remaining undilated stenosis; in all other cases this was due to restenosis. In all patients with single vessel disease, recurrence of angina was due to restenosis except in 1 patient (Table 6).

Repeat angiography after successful percutaneous transluminal coronary angioplasty in multivessel disease was available in 84% (32/38) and was performed at a mean of 3.1 ± 2.5 months after percutaneous transluminal coronary angioplasty; in single vessel disease this was available in 86% (84/98) and it was performed at a mean 2.7 ± 1.8 months after percutaneous transluminal coronary angioplasty. The angiographic restenosis rate was not statistically different in multivessel disease and single vessel disease (32% vs 24%; NS).

Discussion

Unstable angina pectoris, in this study defined as transient ischaemic chest pain lasting at least 15 min and accompanied by reversible ST-T changes, requires aggressive management because it carries an increased risk of myocardial infarction or death^[11-16]. Percutaneous transluminal coronary angioplasty as an alternative to bypass surgery has been advocated in the treatment of unstable angina initially stabilized with pharmacological treatment and in patients in whom pharmacological therapy proves insufficient^[1-5]. PTCA compares favourably with bypass surgery for therapy of selected patients with unstable angina and single vessel coronary artery disease^[4,5]. While the exact percentage of patients with unstable angina and single vessel disease suitable for percutaneous transluminal coronary angioplasty is unknown, it is presumably in the same range as for chronic stable angina which is estimated to be between 5-10%^[1,17]. The majority of patients with unstable angina however have multivessel disease^[6,18]. Successful multiple dilatations in one percutaneous transluminal coronary angioplasty procedure have been reported with acceptable complication rates, in patients with chronic stable angina^[7]. However, this procedure is not yet widely practised and there is even less acceptance of and experience with multivessel dilatation in unstable angina, since the risks inherent in single vessel dilatation might be compounded by multiple vessel dilatation. Minimizing these procedural risks by dilating only the ischaemia related vessel was part of our initial rationale for investigating this treatment in patients with unstable angina and multivessel disease. This view is supported by the recent observation of Smith *et al.*^[22] that major complications are more likely to occur in patients with multivessel disease requiring emergency operation after failed

coronary angioplasty than in patients with single vessel disease and failed angioplasty.

That only partial revascularization might result in an asymptomatic condition is inferred from the fact that many patients with coronary artery disease with or without myocardial infarction are asymptomatic^[19-21].

Furthermore, a compromised left ventricular function, partly or wholly resulting from rest ischaemia may recover over a few days after restoration of adequate perfusion^[23]. In reversing unstable angina to a more stable condition, single vessel dilatation in multivessel disease may allow this improvement in left ventricular function.

If angina recurs, further medical management or repeat percutaneous transluminal coronary angioplasty can then be performed at lesser risk. It turned out that the success rate of percutaneous transluminal coronary angioplasty of the ischaemia related vessel in multivessel disease and in single vessel disease was identical at 88%. The major complications, the occasional need for urgent bypass surgery, and the post PTCA myocardial infarction rate were also comparable. The procedure-related major complication rate of about 10% is in agreement with the rate observed in the treatment of unstable angina with coronary angioplasty reported from the NHBLI PTCA Registry^[4]. This rate is substantially higher than the 2.6% rate reported for elective procedures^[24] which may be related to the unstable condition of our patients. The clinical improvement after single vessel dilatation in multivessel disease was substantiated by evidence obtained from electrocardiographic exercise testing and exercise thallium scintigraphy. Sixty-one per cent of the patients with successful dilatation achieved a workload of more than 90% of the predicted workload on testing at follow up. In the majority of these patients (80%) thallium 201 scintigraphy showed no reversible defects in the myocardial areas supplied by non-dilated significantly stenotic vessels. A similar incidence of reversible perfusion defects in the myocardium supplied by dilated vessels was found in multivessel disease and single vessel disease. However, the maximum workload achieved was lower and the frequency of exercise induced angina, ST-segment depression and reversible perfusion defect was higher in patients with partial revascularization than those with total revascularization. At 6 months follow-up after successful percutaneous transluminal coronary angioplasty, the incidence of angina pectoris in patients with

multivessel disease was 29%, whereas this was only 16% in single vessel disease. This could be attributed to significant disease of non-dilated vessels in patients with multivessel disease. Repeat angiography revealed a comparable incidence of restenosis of the dilated vessel. Since the emphasis was on investigating the symptomatic patients, the true incidence of restenosis is probably less than our figures suggest.

In summary, the results of this study apply only to a selected group of patients and certainly not to all patients with unstable angina and multivessel disease. In this study patients with multivessel disease were only selected if they had good left ventricular function with suitable coronary anatomy for percutaneous transluminal coronary angioplasty and bypass surgery. In our experience (Table 1), the majority of patients with unstable angina and single vessel disease are suitable candidates for PTCA. Approximately 20% of patients with multivessel disease have an ischemia related vessel suitable for PTCA. The results of the present study indicate that dilatation of the ischemia related vessel only, in a selected group of patients with unstable angina and multivessel disease, is effective in 88% of the patients in relieving acute ischaemic symptoms. At 6 months follow-up angina pectoris had recurred in 29%, a higher recurrence rate than in single vessel disease. Thus the consequence of partial revascularization in patients with multivessel disease and dilatation of only the ischaemia related vessel manifests itself in the first 6 months after the PTCA. Also the angina recurrence rate is higher than would be expected following complete revascularization with CABG. Yet, the ischaemia-related vessel dilatation stabilized most patients and the mortality and morbidity of major surgery could be avoided, at least in the acute phase. We now believe that PTCA of only the ischaemia related vessel should be regarded as an initial strategy which in most patients will have long-term success but in some, further dilatations or even CABG will be required. In these, the subsequent interventions can be performed on a more elective basis. This strategy of single vessel dilatation in patients with multivessel disease warrants a randomized controlled clinical investigation.

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References

- [1] Grüntzig AR, Senning A, Siegenthaler WE. Non-operative dilatation of coronary artery stenosis. Percutaneous transluminal coronary angioplasty. *N Engl J Med* 1979; 301: 61-7.
- [2] Williams DO, Riley RS, Singh AK, Gewirtz H, Most AS. Evaluation of the role of coronary angioplasty in patients with unstable angina pectoris. *Am Heart J* 1981; 102: 1-9.
- [3] Meyer J, Schmitz H, Kiesslich T *et al.* Percutaneous transluminal coronary angioplasty in patients with stable and unstable angina pectoris: Analysis of early and late results. *Am Heart J* 1983; 106: 973-80.
- [4] Faxon DP, Detre KM, McGabe CH *et al.* Role of percutaneous coronary angioplasty in the treatment of unstable angina. Report from the National Heart, Lung and Blood Institute Percutaneous Transluminal Coronary Angioplasty and Coronary Artery Surgery Study Registries. *Am J Cardiol* 1983; 5:II: 131C-35C.
- [5] de Feyter PJ, Serruys PW, Wijns W, van de Brand M. Emergency PTCA in refractory unstable angina. *N Engl J Med* 1985; 313: 342-6.
- [6] Rahimtoola SH, Nunley D, Grunkemeier G, Tepley J, Lambert L, Starr A. Ten-year survival after coronary bypass surgery for unstable angina. *N Engl J Med* 1983; 308: 676-81.
- [7] Dorros G, Stertz SH, Cowley MJ, Myler RK. Complex coronary angioplasty: multiple coronary dilatations. *Am J Cardiol* 1984; 53: 126C-30C.
- [8] Serruys PW, van den Brand M, Brower RW, Hugenholz PG. Regional cardioplegia and cardioprotection during transluminal angioplasty, which role for nifedipine? *Eur Heart J* 1983; 4: 115.
- [9] Wijns W, Serruys PW, Reiber JHC *et al.* Quantitative angiography of the left anterior descending coronary artery: Correlations with pressure gradient and exercise Thallium Scintigraphy. *Circulation* 1985; 71: 239-9.
- [10] Reiber JHC, Lie SP, Simoons ML, Wijns W, Gerbrands JJ. Computer quantification of location, extent and type of thallium 201 myocardial perfusion abnormalities. In: Proceedings of the 1st International Symposium on medical imaging and image interpretation. ISM III 1982. IEEE Cat. No 82 CH1084-4: 1982: 123-8.
- [11] Cairns JA, Fantus IG, Klassen GA. Unstable angina pectoris. *Am Heart J* 1976; 92: 373-86.
- [12] Scanlon PH. The intermediate coronary syndrome. *Prog Cardiovasc Dis* 1981; 23: 351-64.
- [13] Russell RO, Moraski RE, Kouchoukos N. Unstable angina pectoris: National cooperative study group to compare surgical and medical therapy. *Am J Cardiol* 1978; 42: 839-48.
- [14] Plotnick GD. Approach to the management of unstable angina. *Am Heart J* 1979; 98: 243-55.
- [15] Silverman KJ, Grossman W. Angina pectoris: National history and strategies for evaluation and management. *N Engl J Med* 1984; 310: 1712-7.
- [16] Michels R, Hugenholz PG, Haalebos M, van den Brand M, Serruys PW, Balakumaran K. Management of unstable angina pectoris. In: Adelman AG, Goldman BS, eds. Unstable angina — recognition and management. Littleton, MA:PSG Publishing Company 1981: 143-63.
- [17] Rapaport E. Percutaneous transluminal coronary angioplasty. *Circulation* 1979; 60: 969-71.
- [18] Alison HW, Russell RO, Mantle JA, Kouchoukos NT, Moraski RE, Rackley CE. Coronary anatomy and

- arteriography in patients with unstable angina pectoris. *Am J Cardiol* 1978; 41: 204-9.
- [19] Cohn PF. Severe asymptomatic coronary artery disease. A diagnostic, prognostic and therapeutic puzzle. *Am J Med* 1977; 62: 565-8.
- [20] de Feyter PJ, van Eenige MJ, Dighton DH, Visser FC, de Jong J, Roos JP. Prognostic value of exercise testing, coronary angiography and left ventriculography 6-8 weeks after myocardial infarction. *Circulation* 1982; 66: 527-36.
- [21] Kannel WB, Abbott RD. Incidence and prognosis of unrecognized myocardial infarction. An update on the Framingham Study. *N Engl J Med* 1984; 311: 1144-7.
- [22] Smith CW, Hornway CA, Sutton JP, Allen WB, Yarbrough JW, Tarner J. Emergency and elective surgical intervention for failed PTCA after unstable angina. In: Hugenholz PG, Goldman BS, eds *Unstable angina. Current concepts and management*. Stuttgart: Schattauer, 1985: 265-78.
- [23] Braunwald E, Kloner RA. The stunned myocardium: prolonged, postischemic ventricular dysfunction. *Circulation* 1982; 66: 1146-9.
- [24] Bredlau CE, Roubin GS, Leimgruber PP, Douglas JS, King SB, Gruentzig AR. In hospital morbidity and mortality in patients undergoing elective coronary angioplasty. *Circulation* 1985; 72: 1044-52.