

Restenosis and repeat coronary angioplasty in Geneva

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KEY WORDS: Coronary angioplasty, restenosis after angioplasty.

Methods

Between July 1983 and August 1985, a total of 400 patients underwent coronary angioplasty on 488 vessels at the Cardiology Centre. There were 358 men (89%) and 42 women (11%) with a mean age (\pm SD) of 55 ± 9 years. There were 83 multi-vessel angioplasties, 56 angioplasties for chronic total occlusion, and 27 angioplasties for evolving myocardial infarction. Clinical follow-up was available in 318 of 400 patients (80%). Of these, 230 patients also underwent control angiography.

The technique of angioplasty which was used has been described previously¹¹. The degree of stenosis was estimated and averaged by two experienced angiographers in at least two projections. Primary success was considered to be present if the following conditions were fulfilled: angiographically improved stenosis, improvement of transstenotic pressure gradient, absence of electrocardiographic or enzymatic evidence of myocardial infarction or need for bypass surgery, and improvement of NYHA functional class and/or stress test. In multi-vessel coronary angioplasty, angiographic success was considered to be present if at least the dilatation of the strategic lesions was successful. Restenosis was defined as a $> 50\%$ narrowing of the site of angioplasty. All patients received aspirin before angioplasty, most of them were given intracoronary nifedipine and/or nitrates immediately before angioplasty and aspirin 100 mg day^{-1} after the procedure. The pressure used for balloon inflations ranged from 4 to 14 bar, and the inflation time from 30 to 90 s. Repeat inflations were performed until the transstenotic pressure gradient had diminished, and the angiographic aspect of the lesion appeared satisfactory.

Clinical follow-up consisted of an interview and/or exercise stress test. A small number of patients were followed by a questionnaire. Control angiography was solicited in all patients. Repeat angio-

graphy was performed promptly in any patient who referred for recurrence of angina or deterioration in functional class.

Results

Primary success was obtained in 348 of 400 patients (87%). Major complications were encountered in 20 patients (5%): one death, 17 (4%) myocardial infarctions, 11 (3%) emergency operations.

Clinical follow-up was available in 318 of 348 patients (91%) with primary success at 10 ± 6 months (Fig. 1). Including repeat angioplasty, 80% of the patients (253/318) with primary success were improved by at least one NYHA functional class and/or had an improved exercise test: 19% (61) were improved by three classes, 31% (97) by two classes, and 30% (95) by one class. Among the 65 patients with no improvement, there was one sudden death at 7 months, one patient had a myocardial infarction at 4 months, 16 patients had bypass surgery, and 47 patients were treated medically.

Seventy patients had repeat angioplasty at 6 ± 4 months (range 3-24 months) after the first procedure. At the time of their second procedure, 61% (43) were still clinically improved by at least one functional class: 3% (2) were improved by three classes, 17% (12) by two classes, and 41% (29) by one class. Among the other patients 24% (17) were again in the same class as before angioplasty and 14% (10) were worse than before angioplasty. The 61% with clinical improvement have to be compared to the 88% with clinical improvement among the 146 patients without recurrence ($P < 0.01$).

Angiographic follow-up in 230 patients with primary success revealed a recurrence in 84 patients (37%) at 12 ± 6 months. In the subgroup with multi-vessel angioplasty, recurrence was found in 21 of the 45 patients (47%) restudied. Recurrence per artery was 40% (56/140) for the left anterior

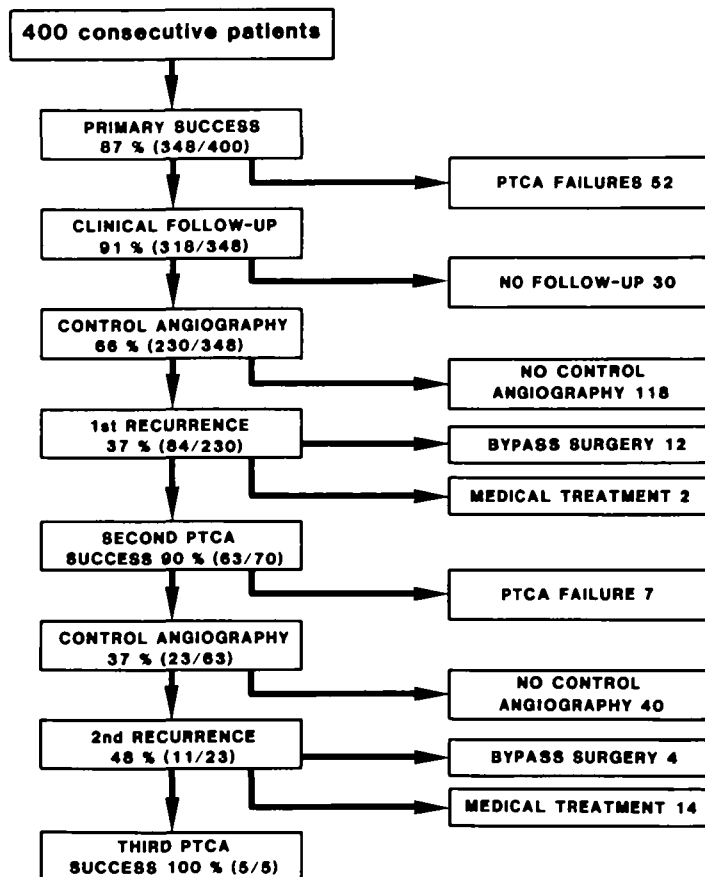


Figure 1 Follow-up after successful coronary angioplasty.

descending artery, 33% (24/73) for the right coronary artery, 30% (18/61) for the left circumflex artery, and 29% (4/14) for diagonal branches. Of these, 70 patients underwent repeat angioplasty, 65 on one vessel, and five on two vessels (Fig. 1). Primary success was 90% (63/70). There was one myocardial infarction, and no emergency surgery. Figure 2 depicts the interval between the first and the second angioplasty. A control angiography was performed in 23 of 63 patients with successful repeat angioplasty. Recurrence was found in 11 patients, representing 17% of the patients with primary success of repeat angioplasty or 48% of the patients with angiographic follow-up. In five of these patients, a third angioplasty was performed after 7 ± 3 months. It was successful and without complications in all patients. Medium-term clinical follow-up (8 ± 6 months) after the last procedure,

available for all 59 patients with repeat angioplasty who did not undergo surgical revascularization, showed improvement of at least one NYHA functional class in 84% (50/59). This percentage is identical to that of the whole group with initial primary success without subsequent bypass surgery where long-term clinical improvement was present in 84% of patients (253/302) after 10 ± 6 months.

Discussion

The accuracy of recurrence rates is subject to the completeness of follow-up angiography. In our study, as in most others^[2], control angiography was done in only 2/3 of the population studied, showing recurrence in 37%. One might speculate that most of the symptomatic patients were restudied and therefore the real recurrence rate would be less if the

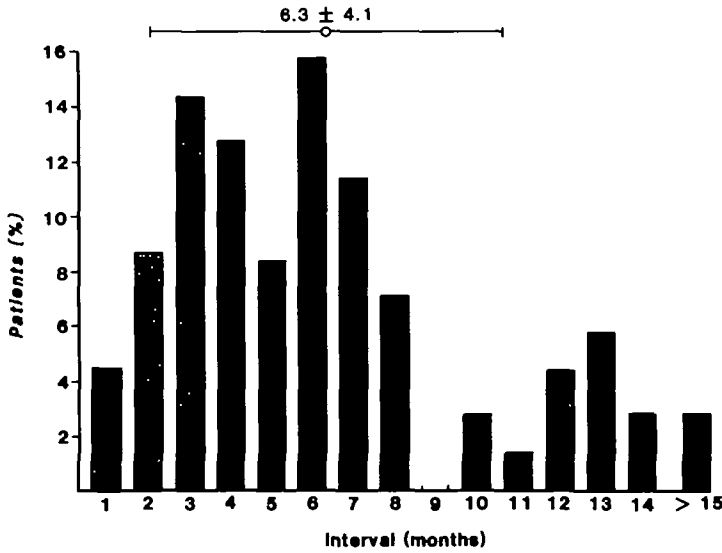


Figure 2 Distribution of intervals between first and second coronary angioplasty in 70 patients.

entire population had been subjected to control angiography.

In patients with angiographically documented recurrence, there was still a clinical improvement in 61% as compared to the pre-angioplasty status. This indicates that recurrences in general cause less symptoms than the initial lesions. Even though recurrences may be only partial and may be treated by repeat angioplasty at a low risk, they are frequent and represent a serious problem. This fact justifies control angiography as well as any effort

towards a better understanding of their pathophysiologic mechanism and new methods to diminish restenosis.

References

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