European Heart Journal (1983) **4**, 678–686

Angina pectoris, one to 10 years after aorto-coronary bypass surgery

K. LAIRD-MEETER, H. J. TEN KATEN*, R. W. BROWER*, M. J. B. M. VAN DEN BRAND, P. W. SERRUYS, M. M. P. HAALEBOS, E. BOS AND P. G. HUGENHOLTZ

*University Hospital Dijkzigt, Thorax Center, Erasmus University, Rotterdam, The Netherlands, and *Interuniversity Cardiology Institute Amsterdam, The Netherlands*

KEY WORDS: Coronary artery bypass surgery, angina pectoris, graft-patency, progression coronary sclerosis.

The incidence of angina pectoris (AP) after bypass surgery was assessed in 1041 patients operated on consecutively between 1971 and 1980. Of the 977 survivors, 920 (94%) participated in the study with a followup time varying from 1 to 10 years (mean 3·5 years). Post-operative angina pectoris was present at 1 year in 277 patients (30%), at 3 years in 46%, at 8 years in 50%. The pain limited usual physical activities in 17·5%, 30% and 25%, respectively at these times. Nonetheless, 89% of the respondents felt improved by surgery. Factors without predictive value for late outcome were sex, number of pre-operative diseased vessels, and pre-operative ejection fraction. A correlation was found between post-operative AP and younger age at surgery in the males only ($P<0·001$); between AP and patency rate of the bypass graft ($P<0·005$) and with the status of the coronary arterial tree at three years post-operatively ($P<0·001$) in both sexes. The percentage of patients with recurrent AP increased with time after surgery up to 3 years, but remained stable thereafter. In conclusion, post-operative AP seems initially related to decreased functioning of the bypass graft, later to progression of coronary sclerosis in the native circulation.

Introduction

Aorto-coronary bypass surgery as treatment for angina pectoris was introduced in 1968[1,2]. Since then it has become an accepted procedure, particularly when pharmacological management has failed to relieve the symptoms. Today, large numbers of patients have been operated upon with more than 85% symptomatic improvement in the first year after surgery[3–5]. But, as more time has elapsed after the operation, angina pectoris has recurred in a appreciable number of patients[6–10]. This study reports on the reoccurrence of chest pain during a post-operative follow-up, of up to 10 years in 920 patients out of 1041 patients consecutively operated upon in the Rotterdam University Hospital. Pre-operative characteristics determining late outcome were sought for. Moreover, an analysis was made of specific causes, which might explain or predict the return of symptoms in the post-operative period.

Received for publication on 26 November 1982; and in revised form on 18 April 1983.

Requests for reprints to: Karin Laird-Meeter, Thorax Center, Erasmus University P.O. Box 1738, 3000 DR Rotterdam, The Netherlands. Telephone: 010-635356.

0195–668X/83/100678+09 $02.00/0

Materials and methods

PATIENT SELECTION

All 1041 patients who underwent isolated aorto-coronary bypass surgery from the time of opening of our institution in February 1971 to May 1980 were included in the study. All patients were symptomatic before operation despite intensive pharmacological therapy. The anginal complaints could be either stable or unstable. Those patients undergoing left ventricular aneurysmectomy or valve repair in addition to the bypass operation, were excluded. At the reference time, May 1981, 64 (6·2%) of the patients had died. The 977 survivors were sent a questionnaire to ascertain their post-operative condition. Of these, 920 (94%) returned their forms. They comprise the study population (Table 1). This group consisted of 109 women, aged 55·5 ± 8·2 years, and 811 men aged 52·6 ± 7·7 years. All

Table 1 Responders to questionnaire constituting the study population, shown in relation to the total number of patients operated consecutively from 1971 to June 1980

Responders	Total		Men		Women	
Alive	977		861		116	
Responders	920	(94·2%)	811	(94·2%)	109	(94·0%)
Died	64		54		10	
Peri-operative	12	(1·2%)	9	(1·0%)	3	(2·4%)
Late	52	(5·0%)	45	(4·9%)	7	(5·5%)
Total operated	1041		915		126	

Responders to questionnaire shown as percentage of those alive in May 1981.

patients were operated upon by the same surgical team. Follow-up time varied from 1 to 10 years with a mean of 3·5 years.

PRE-OPERATIVE CATHETERIZATION

All patients underwent a pre-operative right- and left-heart catheterization with selective coronary angiography. Coronary artery stenosis was judged to be present when a luminal narrowing was seen of 50% or more in the diameter of one of the major coronary arteries in more than one view. The main stem of the left coronary artery was graded separately from the anterior descending and circumflex branch of this artery.

POST-OPERATIVE CATHETERIZATION

Recatheterization was performed at 1 and 3 years postoperative in 169 patients with near consecutive operations between 1976 and 1979. This recatheterization was carried out as part of an ongoing national study, irrespective of complaints[13]. The coronary angiograms were first analysed for patency of the aorto-coronary bypass. In addition the coronary score was calculated as described by Leaman *et al.* in 1981[14], which was based on a scheme accepted by the American Heart Association[15]. This score takes into consideration the amount of luminal diameter narrowing both in the native coronary arteries and in the aorto-coronary bypass. Moreover it is weighted according to the expected flow to the left ventricle in each coronary vessel. The score thus represents the 'resistance' to the expected blood flow to the myocardium. The place of anastomosis of the bypass on the coronary artery determines which segments of the coronary tree are reached with flow from the bypass as well as the segment which cannot be reached because of intervening stenoses, thus providing the opportunity to correct the score after bypass surgery. The coronary score varies between 0 in normals and 50 in severe three vessel diseasé.

Ejection fraction (EF) was calculated from the left ventricular angiogram in the right anterior oblique projection with the area length method adjusted for single plane views[11,12]. A normal EF was considered present if it was $\geqslant 55\%$, a poor EF if $\leqslant 30\%$ and a moderately decreased EF when $> 30\%$ but $< 55\%$.

DATA STORAGE AND ANALYSIS

Data concerning pre-operative catheterization, number of grafts placed at operation, post-operative catheterization, when available, and subjective data from the questionnaire were stored using the MUMPS system on a PDP-11 computer. The nature of this data handling system makes it possible to combine data originating in various subdepartments[16] of the cardiology and the thoracic surgery division. For each patient, 46 items were stored. Analysis was performed with sorting programs written in BASIC and executed on a Hewlett Packard 85 microcomputer. Statistical testing was done with the Hewlett Packard statpac Chi-square contingency test[17], the Mantel-Haenszel test[18], the Friedman's two-way analysis by rank[19] and the Wilcoxon's rank sum test[19].

Results

At follow-up, angina pectoris (AP) was present in 434 (47%) individuals. Of the 109 women, 60 or 55% experienced AP and of the 811 men, 374 or 46%. The difference in the occurrence of post-operative angina pectoris between men and women, 46% versus 55%, was not statistically significant ($\chi^2 = 3{\cdot}07$, $P = 0{\cdot}079$). The data for both sexes were therefore pooled for further analyses unless stated otherwise.

With regard to the severity of the pain in the chest: of the 434 individuals with post-operative angina pectoris, 332 (or 36% of the total population) had less severe complaints after the operation than before. The complaints were reported to be 'as bad or worse than before' by 81 patients. These 81 and the 21 patients, who provided non-specific replies, constitute 11% of the total population. They were regarded as unimproved by surgery. On the other hand, we found that 486 patients were pain free while 332 patients described their

Table 2 Circumstances during which angina pectoris was experienced by the 434 patients reporting symptoms

Event	No. of patients	Percent of responders $n = 920$	Percent of group with AP $n = 434$
At rest	148	16%	34%
Light physical work (dressing, bathing)	91	10%	21%
Medium physical work (walking outside, shopping)	315	34%	73%
Heavy physical work (bicycling, gardening)	318	35%	73%
Emotion	352	38%	81%

AP = Angina pectoris.

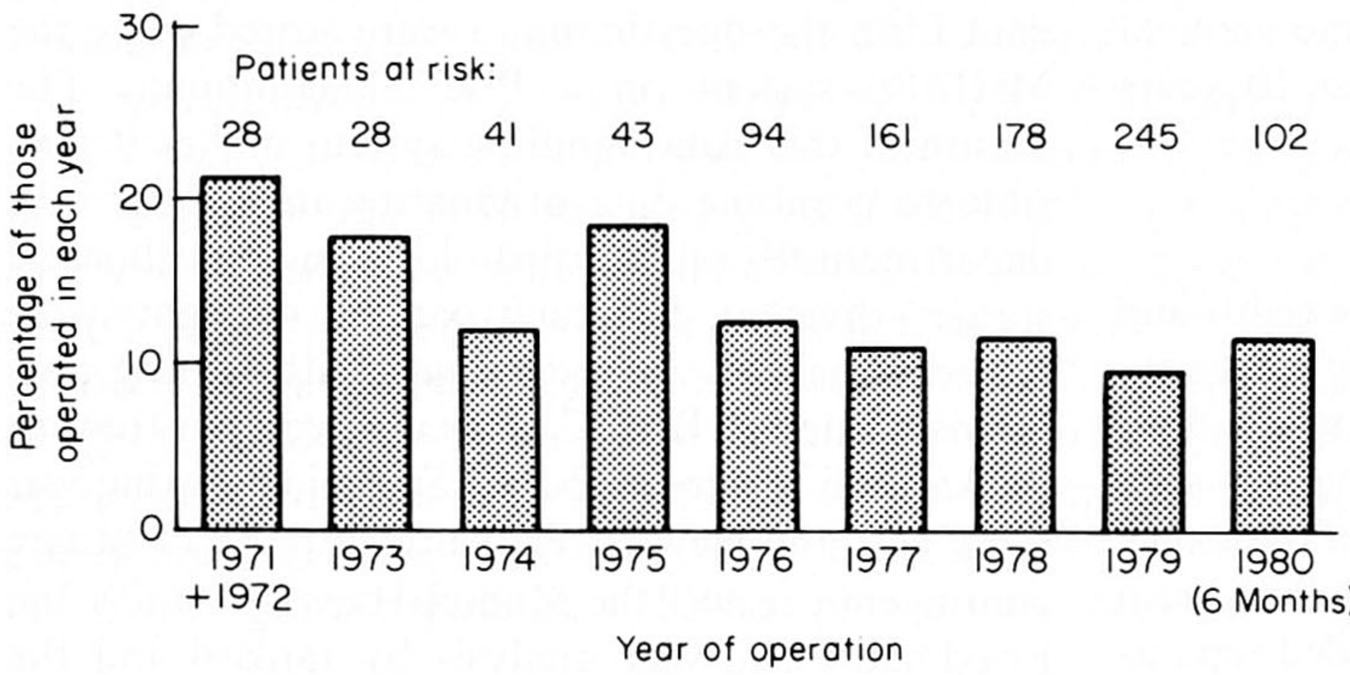

Figure 1 Patients with post-operative angina pectoris, persisting after surgery, expressed as a percentage of those operated on in that year. In the 140 patients operated before 1976, pain persisted in 24 (17%), of the 780 patients operated thereafter this occurred 87 times (11%). The difference is significant at $P < 0{\cdot}05$.

anginal complaints as less severe. Thus, 818 (89%) of the total group can be regarded as improved.

The various circumstances under which post-operative AP was experienced are listed in Table 2. It is possible for an individual to have complaints provoked by more than one event, causing the absolute number of patients to be larger than the total who reported pain. The high fraction with pain on emotion is noteworthy: 38% of the total and 81% of those with pain in the chest. Angina pectoris 'never disappeared' after the operation in 111 of the 920 patients (12%), as shown in Fig. 1. This fraction was slightly higher in the early years of this study. In 24 (17%) of the 140 patients operated in the years 1971 through 1975, AP never disappeared, while of the 780 operated on between 1976 and 1980 this was the case in 87 (11%). The difference is significant ($\chi^2 = 4{\cdot}012$, $P < 0{\cdot}05$).

The incidence of AP in the years following operation increased steadily from 16% at 3 months to 46% at 3 years. No further increase in the number of symptomatic patients in the population at risk is evident after three years (Fig. 2). It was not only the occurrence of AP which showed a trend in time, the severity of complaints did so as well. Angina pectoris, sufficiently severe to limit physical exercise, is shown as a percentage of the population at risk. The fraction of the individuals experiencing pain, which did not influence their daily activities, was rather constant at 15%. The increase in the number of patients with pain in the first three post-operative years is seen in the group which feels it is limiting their activities.

To study the influence of age at operation on the post-operative results, the total population was subdivided in four age groups: younger than 40

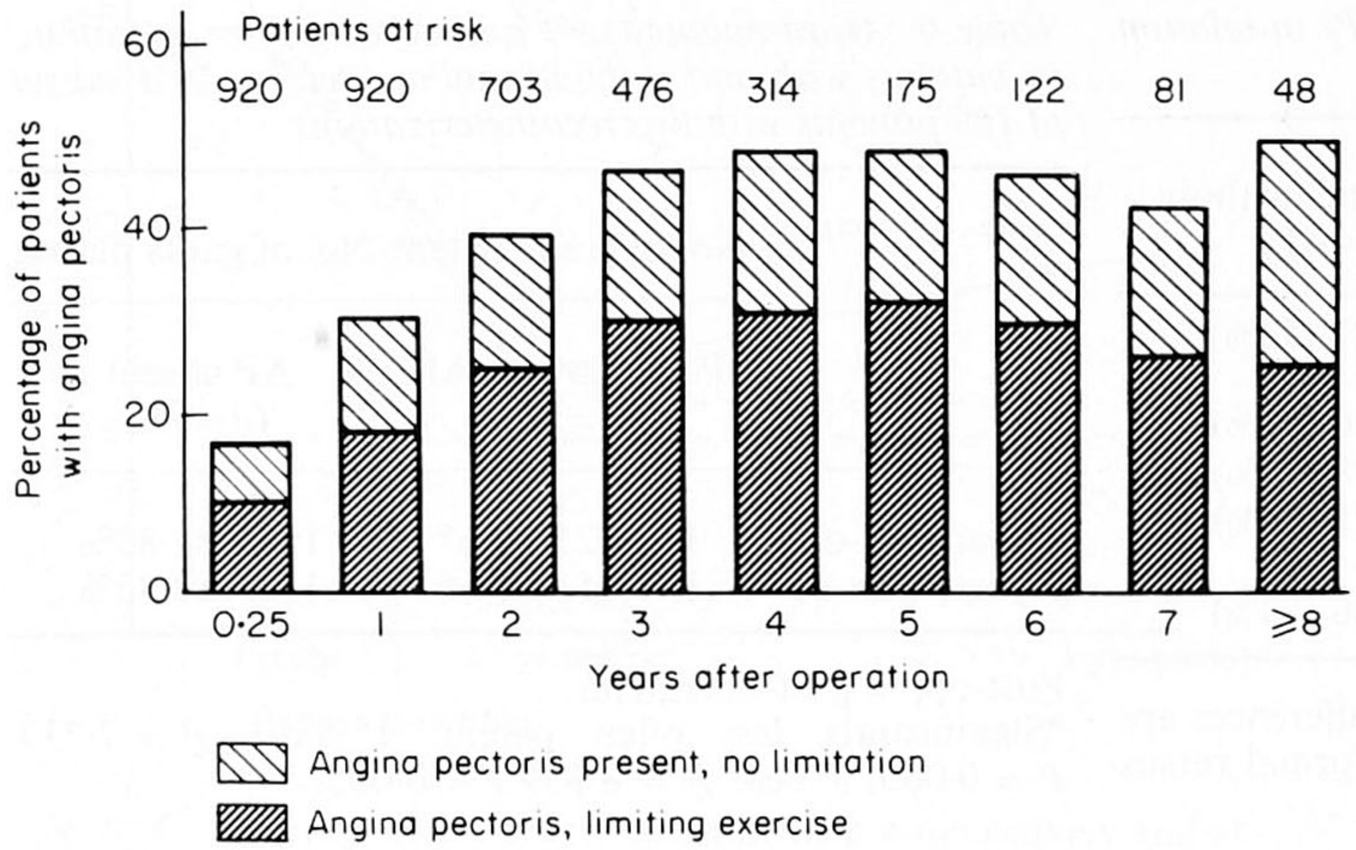

Figure 2 Number of patients with angina pectoris at yearly intervals after aorto coronary bypass surgery. The number of patients at risk indicates those followed up to the end of that year. Symptoms are subdivided into pain limiting physical activities, and pain experienced but not limiting activities.

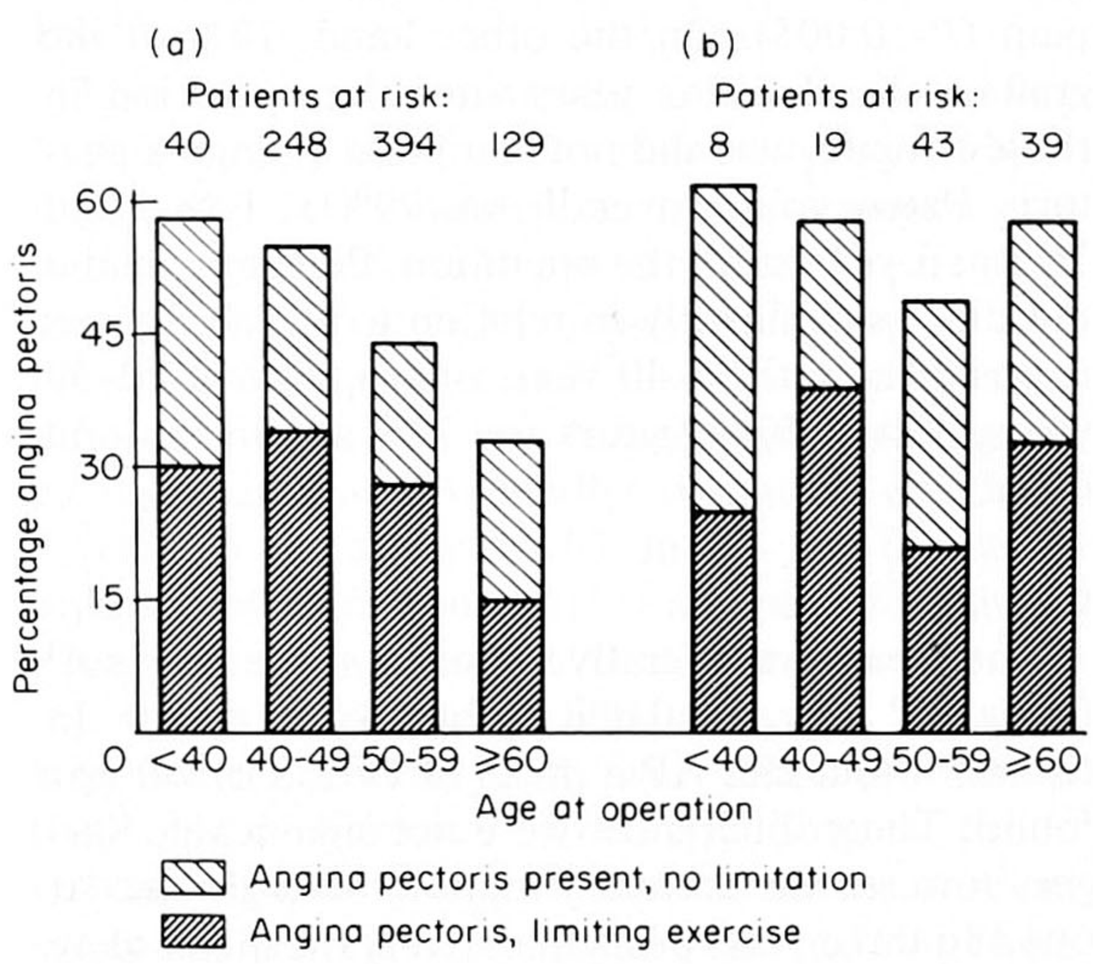

Figure 3 Post-operative angina pectoris at mean follow up of 3·5 years, occurring in men and women, with a subdivision according to age at operation. Angina pectoris present and angina pectoris limiting physical activities shows an age trend in men ($P<0·001$) but not in women. (a) Men; (b) women.

years, 40–59 years, 50–59 years and 60 years and older. The incidence of AP in men was 58% for the youngest, 55% and 44% for the two middle groups, against 33% in the oldest. This age trend was highly significant ($P<0·01$)[18]. No trend could be discerned in the women (see Fig. 3). In those males who feel limited by pain, there is a trend with age as well: the younger the age at operation the higher the incidence of AP limiting physical activities ($P<0·005$). This age trend is, again, not seen in women ($P=0·84$).

ANATOMICAL CORRELATES

Extent of pre-operative vascular lesion

Pre-operative vascular disease varied from involvement of one vessel in 171 (18%) of the patients, to two vessels in 291 (32%), three vessels in 386 (42%) and the left main stem in 72 (8%). In Table 3 it can be seen that post-operative angina

Table 3 Post-operative angina pectoris (AP) in relation to pre-operative number of stenosed vessels, as reported by 920 patients

Stenosed vessels	Patients with AP	Patients without AP
1 vessel	81 (47%)	90 (53%)
2 vessels	141 (48%)	150 (52%)
3 vessels	182 (47%)	204 (53%)
LM	30 (42%)	42 (58%)
Total	434 (47%)	486 (53%)

These differences are not statistically significant (χ^2 1·07 $P = 0·78$). AP = angina pectoris at follow-up. LM = left main coronary artery.

Table 4 Post-operative angina pectoris (AP) in relation to pre-operative ejection fraction

Ejection Fraction	Patients with AP	Patients without AP
Normal	256 (47%)	287 (53%)
Moderately impaired	92 (43%)	120 (57%)
Poor	15 (65%)	8 (35%)
Unknown	71 (50%)	71 (50%)
Total	434 (47%)	486 (53%)

AP = angina pectoris at follow up. These differences are not statistically significant when tested for Normal versus Poor ($P = 0{\cdot}09$).

Table 5 Post-operative angina pectoris (AP) and number of grafts placed at operation in relation to the number of stenosed vessels pre-operatively

Stenosed vessels	Number of grafts/Patient	
	Patients with AP	Patients without AP
1 vessel	1·44	1·27
2 vessels	2·32	2·10
3 vessels	3·36	3·52
LM	3·17	3·26

LM = Left main coronary artery. AP = Angina pectoris at follow-up

pectoris ranges from 48% in patients with two vessel disease to 42% in those with a lesion in the main stem of the left coronary artery. Analysed with the Chi-square contingency test, these observed differences could only be attributed to random variation ($P=0{\cdot}78$).

Pre-operative ejection fraction

In Table 4, the occurrence of post-operative AP is demonstrated between groups with various ejection fractions. There appeared to be no statistically significant difference, even when the worst group, those with a poor EF, were tested versus the group with a normal EF ($P=0{\cdot}09$).

Number of grafts placed at operation

There is no significant difference in the number of grafts placed at surgery in those who later experienced angina pectoris compared to those who did not (Table 5).

Table 6 Graft patency at 1 and 3 years after operation, in patients with and without angina pectoris, in a subset of 169 patients with two recatheterizations

	No. of grafts patent/No. of grafts placed	
	Patients with AP ($n=98$)	AP absent ($n=71$)
1 year post-op.	189/225 74%*	155/182 85%
3 years post-op	181/255 71%*	151/182 83%

Post-op. = post-operative.
*Significantly less often patent. 1 Year $\chi^2 = 7{\cdot}737$ $P = 0{\cdot}005$. 3 Year $\chi^2 = 8{\cdot}359$ $P < 0{\cdot}005$.

Patency of the grafts

In Table 6 it is shown that the patency rate in the 169 recatheterized patients is 74% at one and 71% at 3 years for those with AP after the operation. This is significantly lower than the observed 85%, respectively 83% in the patients who did not have pain ($P<0{\cdot}005$). On the other hand, 17% of the grafts were closed 3 years after the operation in those patients who did not complain of angina pectoris. Patency rate, overall, was 79% at 1 year and 76% at 3 years after the operation. Patency rate did not differ significantly in relation to age: at 3 years it was 78% in those 40 years of age, 79% at 40–49 years, 76% at 50–59 years and 74% at 60 years and older.

Coronary score

The mean pre-operative score in those later suffering AP was calculated to be 13·8, S.D. 8·0. In those without late AP a mean of 14·2, S.D. 7·0 was found. These differences were not significant. Surgery lowered the coronary score in both groups. At one and three years post-operatively the mean score in the AP group was 6·4, S.D. 4·0 and 9·3, S.D. 6·5, respectively. In the asymptomatic group, these values were 4·7, S.D. 4·0 and 4·8, S.D. 5·1, as illustrated in Fig. 4. With two-way analysis of variance it could be concluded that surgery lowered the coronary score significantly at 1 and 3 years post-operatively in both the symptomatic and asymptomatic group ($P<0{\cdot}001$). At the first post-operative study, the difference in coronary score between groups did not reach statistical significance ($P=0{\cdot}07$); however at the second post-operative study the coronary score in the group with AP was significantly higher than in the asymptomatic group ($P<0{\cdot}001$, by Wilcoxon's rank sum test[19]).

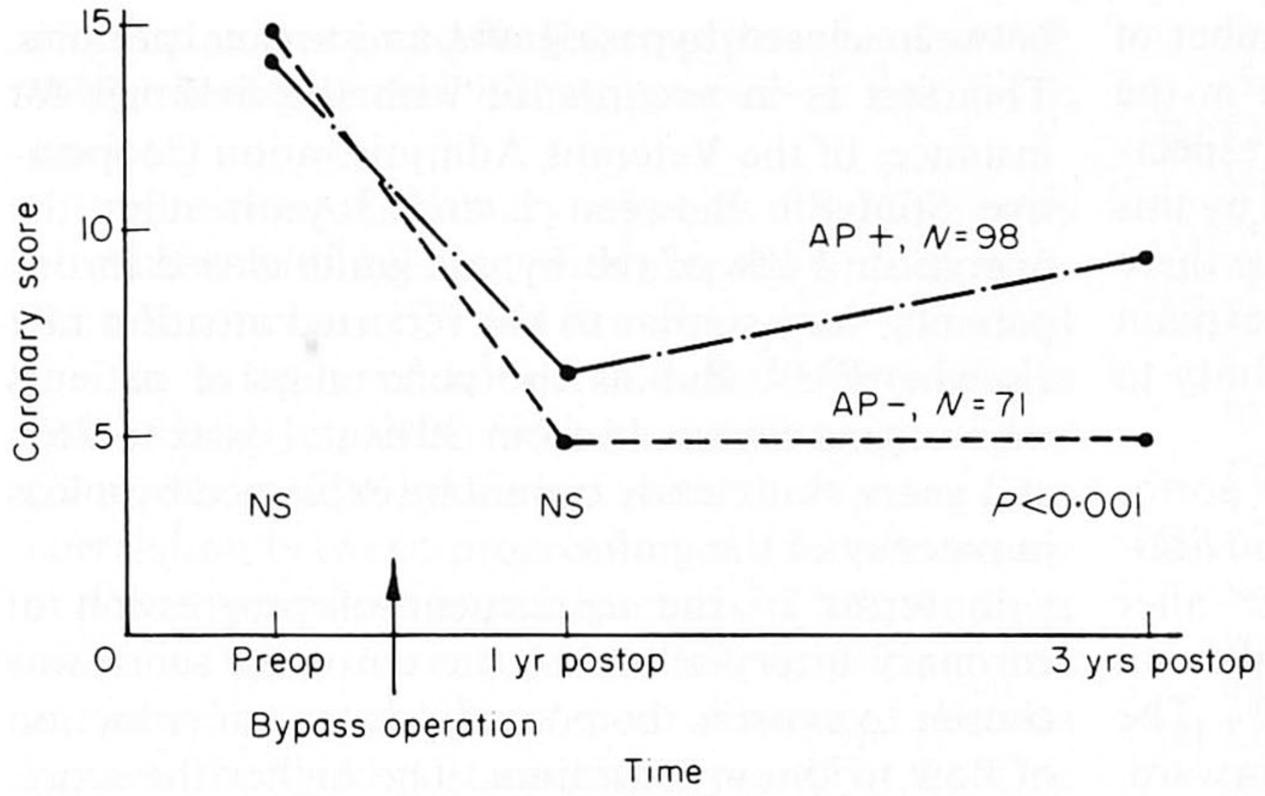

Figure 4 Mean coronary score, determined pre-operatively and at 1 and 3 years post-operatively, in the group experiencing AP and the group without AP at the reference date. Number of patients indicates those catheterized three times, with follow-up data on AP via questionnaire. The coronary score worsened significantly in AP group between 1 and 3 years post-operatively ($P < 0{\cdot}001$), which did not happen in the group without AP.

Discussion

Loss of improvement late after aorto-coronary bypass surgery was first commented on by McNeer in 1974[20]. He reported a drop in angina free patients from 62% at 1 year to 53% at 2 years post-operatively, as seen by yearly follow-up of 260 patients. Campeau *et al.*[6] also found a deterioration of results. The percentage of patients experiencing angina at 1 year after operation was 38 at 3 years 45 and at 7 years 63. In a 7-year follow-up reported by Cameron *et al.*[21], angina recurred at a linear annual rate from 21% at 1 year to 37% at 6 years with an increase to 50% at 7 years. The only large-scale European study with follow-up data is that of the European Coronary Surgery Study Group[22] showing angina pectoris in the surgical group at 1 year in 42% and at 3 years in 51%. These results, seen in the first three post-operative years, are similar to ours and are, given the initially favorable results, in fact disappointing. However we did not see a continuation of the increase in the proportion of symptomatic patients beyond the third post-operative year.

One possible shortcoming in this study was the fact that a questionnaire was used to assess the post-operative state. As all patients suffered angina pectoris before the operation, it could be expected that after surgery these same patients would be able to recognise their old complaints. Therefore, if a patient reported symptoms similar to those prior to surgery, angina pectoris was assumed to be present. Nevertheless we realize that a questionnaire rather than an interview could be the cause of some misinterpretations. A second shortcoming might be that this is not a longitudinal study of individual patients, but a sample of a large patient group at one moment. Thus it is possible that a patient, at first, experiences mild recurrence of angina and only later severe symptoms. As a consequence, the symptoms are classed as severe, and the mild period is missed. Similarly, the 53 patients who required reoperation were categorized according to their condition after the second operation, thus missing cross-over from severe symptoms to their present improved state. In this respect the late mortality in our population is noteworthy as well. In total 52 patients (5%) died during the follow-up period of a mean of 3·5 years—an average death rate of 1·4% per year. As it is not known if these patients had post-operative AP or not, they have been omitted from the analysis. Assuming that they did have AP, a bias has been introduced, albeit not a very large one, favoring the group without complaints.

A third criticism might be that the timing of the questionnaire led to separation in time between the answers to the questionnaire and the moment of re-catheterization. We elected to do this, because it made a comparison possible with patients who were not re-catheterized, as the history was obtained in the same manner in all 920 studied patients. The mean interval between the return of the questionnaire and the moment of the last re-

catheterization was 7·7 months. As the number of patients reporting recurrence of symptoms in the third and fourth post-operative year was, respectively, 7 and 2% (Fig. 2), the error introduced by this time gap seemed acceptable. Neither of these three possible sources of errors can, however, explain why the loss of improvement was limited only to the first three post-operative years.

The exact causes of angina pectoris after aorto-coronary bypass are still unclear. Several investigators have commented on early failure after surgery[8,9] and later loss of improvement after an apparently successful, initial operation[6,7,10]. The following five possibilities have been put forward. (1) Poor functional state of the blood vessels, (2) impaired ventricular function prior to surgery, (3) insufficient revascularization at surgery, (4) closure of the bypass graft, or (5) progression of coronary sclerosis in either the bypassed artery or the un-bypassed arteries resulting in a loss of blood flow to the myocardium during the post-operative period. These possibilities will be discussed in relation to the findings of this study.

The first three factors could not be shown to influence late symptoms, since the difference in prevalence of post-operative AP did not reach statistical significance in respect to the pre-operative number of stenosed vessels, pre-operative ejection fraction, or number of grafts placed at operation. However, the experience of the cardiac surgical team did seem to influence results as evidenced by the slightly higher percentage of patients in which the pain persisted after surgery in the years before 1976 compared with later. Thus, insufficient revascularization due to the technique may bear a relation to post-operative angina pectoris. Also related to the surgical technique is the closure of the bypass graft. As Bourassa *et al.*[23] have shown, early closure, within one month of surgery, is clearly influenced by technical factors and due to thrombosis in the graft. It commonly occurs in 12–15% of the grafts. Late closure, from 1 to 12 months post-operatively, is caused by fibrous intimal proliferation which is seen in 5–15% of all grafts. Closure after 1 year is rare, an attrition rate of 1–2% per year has been reported. It is often seen as a progression of a formerly localized stenosis or, rarely, as a new atherosclerotic lesion[23,24].

In our patients, no data are available on the patency shortly after operation. At one year, the overall patency rate in the recatheterized subset of patients was 79%, 74% in those with AP and 85% in those without. Thus showing a correlation between closed bypass grafts and angina pectoris. This fact is in accordance with the findings, for instance, of the Veterans Administration Cooperative Study[25]. Between 1 and 3 years after the operation 2–3% of the bypass grafts closed in our patients, very similar to the reported attrition rate elsewhere[23,24]. But as the percentage of patients with angina increased from 30% at 1 year to 46% at 3 years, this clearly cannot be explained by a loss in patency of the grafts.

In terms of the assessment of progression of coronary artery-sclerosis, the coronary score was chosen to express the potential extent of reduction of flow to the myocardium. The higher the score, the higher the degree of stenosis in the more proximal arteries. As reported previously[14], the level of the score did not correlate directly with the severity of angina pectoris but the score could be used to evaluate the effect of surgery by comparing the pre- and post-operative figures in the same patient, while it also indicated progression or regression of coronary sclerosis in the vessels not touched by the surgeon. In our study, the coronary score was lowered markedly by surgery both at 1 year and at 3 years after surgery, suggesting that surgery indeed improved blood flow to the myocardium and that this result was maintained over the course of 3 years. In those patients with post-operative AP, the score increased significantly between the two post-operative studies, while this did not happen in those without AP. The change in score at that time cannot be explained by the attrition rate of the bypass: respectively 3% and 2% of the grafts closed in the two intervening years in the group with and without AP. Thus, only progression of atherosclerosis in the native coronary vessels can explain the increased score. This leads to the conclusion that late post-operative AP is related to the progression of coronary artery sclerosis.

The influence of age at the time of operation on post-operative angina pectoris has not been commented on by others. In this study, younger men fared worse regarding post-operative angina pectoris than older men. It has been shown by Kramer *et al.*[26] that coronary sclerosis progresses faster in men below the age of 50 years than in older individuals. This could be the reason why post-operative angina pectoris was more often seen in the younger age groups. But it has to be realized that the complaint of angina pectoris is an entirely subjective one. No objective data on exercise tolerance are available in these patients. It is not unreasonable to presume that older individuals undertake less

physical activity and therefore experience less AP. Why this age-trend did not occur in women is unclear.

Progression of coronary sclerosis not only shows a relation with age, but with elapsed time as well. Both Kramer *et al.*[26] and Bruschke *et al.*[27] showed this in a study of unoperated patients, while in post-operative patients it has been demonstrated by Campeau *et al.*[6]. In this last study there was a clear correlation between progression of sclerotic disease and post-operative angina pectoris. These findings make it the more remarkable that we did not find an increase in the incidence of angina pectoris beyond the third post-operative year (Fig. 2). One would have expected that, as the time elapsed after surgery increased, new stenoses would be formed with more angina pectoris as a result.

In conclusion, aorto-coronary bypass surgery remains a significant addition to the management of patients with severe angina pectoris, even with present day pharmacological therapy. Improvement after surgery is common, but angina pectoris at late follow-up occurred in nearly half of the patients, especially younger men. When angina pectoris persists after surgery, incomplete revascularization is usually the reason. On the other hand, deterioration after the first post-operative year is mainly caused by progression of coronary sclerosis.

These findings should give cause to rethink the present day tendency to operate on young(er) patients, often with minimal symptoms, to 'prevent' future complaints. As long as progression of coronary sclerosis cannot be arrested, 'preventive' surgery would seem to be unwarranted.

We thank H. Wolf and Louise van Solkema for completing the data file and Sandra Lonnee for preparing the manuscript.

References

[1] Favaloro RG. Saphenous vein autograft replacement of severe segmental coronary artery occlusion-operative technique. Ann Thorac Surg 1968; 5: 334–9.

[2] Johnson WD, Flemma RJ, Lepley D Jr, Ellison EH. Extended Treatment of severe coronary artery disease: a total surgical approach. Ann Surg 1969; 170: 460–70.

[3] Effler DB, Favaloro R, Groves LK *et al.* The simple approach to direct coronary artery surgery: Cleveland clinic experience. J Thorac Cardiovasc Surg 1971; 62: 503–10.

[4] Hall RJ, Dawson JJ, Cooley DA *et al.* Coronary artery bypass. Circulation Suppl 1973; 48: 146–50.

[5] Sheldon WC, Rincon G, Effler DB *et al.* Vein graft surgery for coronary artery disease: survival and angiographic results in 1,000 patients. Circulation (Suppl 3) 1973; 38: 184–9.

[6] Campeau L, Lespérance J, Hermann J, Corbara F, Grondin CM, Bourassa MG. Loss of the improvement of angina between 1 and 7 years after coronary bypass surgery. Circulation (Suppl 1) 1975; 60: 1–5.

[7] Seides SF, Borer JS, Kent KM, Rosing DR, McIntosh CL, Epstein SE. Long-term anatomic fate of coronary bypass grafts and functional status of patients five years after operation. N Engl J Med 1978; 298: 1213–7.

[8] Culliford AT, Girdwood RW, Isom OW, Krauss KR, Spencer FC. Angina following myocardial revascularization. J Thorac Cardiovasc Surg 1975; 77: 889–95.

[9] Loop FD, Cosgrove DM, Kramer JR *et al.* Late clinical and arteriographic results in 500 coronary artery reoperations. J Thorac Cardiovasc Surg 1981; 81: 675–85.

[10] Tecklenberg PL, Alderman EL, Miller DC, Shumway NE, Harrison DC. Changes in survival and symptom relief in a longitudinal study of patients after bypass surgery. Circulation (Suppl 1) 1975; 98: 51–2.

[11] Dodge HT, Sandler H, Baxley WA, Hawley RR. Usefulness and limitations of radiographic methods for determining left ventricular volume. Am J Cardiol 1966; 18: 10–24.

[12] Heintzen PH, Brennecke R, Bursch JH, Longe P, Malerczek V, Moldenhauer K, Onnasch D. Automated video-angiocardiographic image analysis. Computer 195; 55–64.

[13] Brower RW, ten Katen HJ, Meester GT. Interim data processing in the Netherlands study on coronary bypass graft surgery. Comp and Biomed Res 1980; 13: 87–101.

[14] Leaman DM, Brower RW, Meester GT, Serruys P, v.d. Brand M. Coronary artery atherosclerosis: severity of the disease, severity of angina pectoris and compromised left ventricular function. Circulation 1981; 63: 285–92.

[15] Report of the Ad Hoc Committee for grading of coronary artery disease: A reporting system on patients evaluated for coronary artery disease. Council on Cardiovascular Surgery. Dallas. American Heart Association, document; 73–315–A.

[16] Laird-Meeter K, Hoare MR. A diagnostic coding system as used in a cardiology out-patient clinic with access to diagnoses generated in clinical departments. Longbeach California: Computers in Cardiology 1981; 133–6.

[17] HP-85 General Statistics Pack: Hewlett-Packard Company, 1980.

[18] Mantel N, Haenszel W. Statistical aspects of the analysis of data from retrospective study of disease. J Natl Cancer Int 1959; 22: 719–48.

[19] Ganz SA. Primer of Biostatistics. New York: McGraw Hill, 1981.

[20] McNeer JF, Starmer CF, Bartel AG, *et al.* The nature of treatment selection in coronary artery disease. Circulation 1974; 49: 606–14.

[21] Cameron A, Kemp HG, Shimomura S, *et al.* Aorto coronary bypass surgery. A 7 years follow-up. Circulation 1979; 60 (Suppl 1): 9–13.

[22] European Coronary Surgery Study Group. Prospective randomised study of coronary artery bypass surgery in stable angina pectoris. Lancet 1980; 11: 491–5.
[23] Baurassa MG, Campeau L, Lesperance J, Grondin CM. Changes in Grafts and Coronary arteries after saphenous vein aorto-coronary bypass surgery: results at repeat angiography. Circulation 1982, 65 (Supple II); 90–7.
[24] Lawrie GM, Morris GC, Chapman DW, Winters WL, Lie JT. Patterns of patency of 596 vein grafts up to seven years after aorto-coronary bypass. J Thorac Cardiovasc Surg 1977; 73: 443–8.
[25] Peduzzi P, Hultgren HN. Effect of medical vs Surgical Treatment on symptoms in stable angina pectoris. Circulation 1979; 60: 888–900.
[26] Kramer JR, Matsuda Y, Mulligan JC, Aronow M, Proudfit WL. Progression of Coronary Atherosclerosis. Circulation 1981; 63 (Suppl 3): 519–26.
[27] Bruschke AVG, Wegers TS, Kolsters W, Landman J. The anatomic evolution of coronary arteriography in 256 non-operated patients. Circulation 1981; 63: 527–36.

Comment by D. J. Wheatley (Glasgow, U.K.)

Aorto-coronary bypass surgery is now one of the commonest and most expensive forms of surgery in Western countries. Its value is widely accepted by the medical profession, and the general public has come to have very high expectations of this form of surgery. The authors of this paper are therefore to be congratulated on a valuable and appropriate review of results.

The quality of the surgery reported here is excellent—overall perioperative mortality of 1·2% and commendable grafting rates attest to this. It is important to emphasise that 89% of the respondents were improved by surgery and 46·7% (almost half the total group) were pain free at a mean period of 3·5 years after surgery. The authors point out a difficulty of their study—evaluation of angina is undertaken by questionnaire. This makes it a little difficult to evaluate such a subjective symptom as angina. No mention is made of the need for postoperative pharmacologic therapy and this may have a bearing on the severity of symptoms. Nevertheless, the authors have drawn attention to a rather disappointingly high incidence of recurrent angina (30% at one year, 46% at three years, and 50% at eight years) in a group having good surgery.

It is notable that recurrence of angina and closure of bypass grafts correlate well in this study at one year, but that at three years the increased incidence of angina cannot be ascribed to further graft occlusion. The evidence that progression of coronary disease is occurring is important, and only serves to emphasise the need for continued effort in finding measures to arrest the progression of coronary atherosclerosis.

An important aspect of this study is that the late death rate (average 1·4% per year) is reassuringly low. There is also confimatory evidence from this report that coronary disease progresses more rapidly in younger men. The suggestion by the authors that 'preventive' surgery in younger patients may be unwarranted must remain open to debate, as these may be the very patients in whom improvement of longevity can be achieved by surgery.

The two major reasons for undertaking aorto-coronary bypass surgery are currently the expectation of relief of angina and the hope of improving natural history. This important paper gives a timely reminder that angina pectoris may well recur in a disappointingly high proportion of patients, and it emphasises the need for continued endeavours to find means of arresting the progress of disease.