Balloon dilatation of the aortic valve for inoperable aortic stenosis

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Abstract

The place of balloon dilatation of the aortic valve in the treatment of calcific aortic stenosis is controversial. Thirty two patients (mean age 76) in whom valve replacement was contraindicated were followed up for three to 24 months (mean 8); 25 were in functional class III or IV according to the New York Heart Association's classification. Major complications of the procedure occurred in four patients. Echocardiography and Doppler studies were performed before operation and before discharge in 28 patients, and the area of the valve was measured again six to 50 (mean 23) weeks after operation in 11 patients. The peak to peak aortic pressure gradient fell from a mean of 65 (SD 24) to 46 (20) mm Hg, but the area of the aortic valve, measured by Doppler echocardiography, in 18 patients showed a modest but significant increase, from 0.61 (0.16) to 0.74 (0.23) cm². One month after dilatation, 29 patients were alive, of whom 17 had improved symptoms. Only two had lasting clinical benefit. Sixteen patients died, 12 of a cardiac cause. The estimated one year survival rate was 49%. Six patients underwent or required valve replacement because of persisting symptoms.

In view of its limited long term efficacy balloon dilatation of the aortic valve should be used only for patients with severe symptoms whose life expectancy is limited by other disease or who are considered to be unsuitable for valve replacement. It may have a role in improving the condition of patients who present with cardiogenic shock or pulmonary oedema before valve replacement is undertaken.

Introduction

Patients aged 70 and over form a large proportion of those with severe aortic stenosis.¹ The mortality associated with elective aortic valve replacement in this age group is roughly 5%,^{2,3} but that associated with emergency operation may be as high as 31%.³ Valve replacement may not be undertaken in some elderly patients (those considered to have an unacceptably high operative risk or limited life expectancy because of accompanying non-cardiac disease), but data on this have not been included in reports of surgical series.

The use of percutaneous balloon dilatation of the aortic valve to treat calcific aortic stenosis has attracted wide interest since the first report of the technique in 1986.⁴ Although it improves valve function and symptoms in the short term,⁵⁻¹⁰ its long term efficacy remains unclear. We report on 32 patients who were followed up for up to two years.

Patients and methods

Patients were selected for balloon dilatation of the aortic valve if calcific aortic stenosis had been con-

firmed by cross sectional echocardiography, Doppler ultrasound, and measurements of intracardiac pressure; they had symptoms of angina, breathlessness, or syncope (New York Heart Association's functional classes II-IV¹¹); appreciable aortic regurgitation (no more than grade 2+ on supravulvar aortography) and left main stem coronary artery disease were absent; and one or more major contraindications to valve replacement had been agreed with a cardiac surgeon. Contraindications included advanced age and infirmity, malignancy, or other non-cardiac disease associated with an increased operative risk. One patient (a 73 year old man) had refused valve replacement.

Coronary arteriography was performed by Judkins's technique through the right femoral artery,¹² followed by supravulvar aortography to assess the degree of aortic regurgitation and identify the orientation of the valve orifice. The aortic valve was crossed with a straight guide wire; an Amplatz left coronary catheter was used as a guide if necessary. Left ventricular pressure was recorded with simultaneous aortic pressure by either a catheter inserted through the left femoral artery or a long sheath in the right femoral artery.

If the aortic valve was suitable for balloon dilatation the patient was given heparin 5000 units, the right femoral artery was progressively dilated, and a 14 French gauge sheath with a haemostatic valve was inserted. The catheter balloon was introduced by an exchange guide wire (the end of which had been bent into a large J to reduce the risk of perforating the ventricle) and positioned across the aortic valve.

Before the balloon was inflated atropine 1 mg was given intravenously. The balloon was inflated by hand with a mixture of saline and contrast to the manufacturer's maximum recommended pressure (400-600 kPa (4-6 atmospheres)). Inflation was maintained for 10-15 seconds and aortic pressure monitored continuously. Although other workers have used longer periods of inflation,^{6,7} we found that inflation for longer than 15 seconds often resulted in a substantial fall in aortic pressure, which required an infusion of dopamine. Usually several balloons of increasing diameter were used and each inflated two or three times. Cinefilm was taken to confirm full inflation of the balloon and loss of indentation by the stenotic valve.

After each inflation and at the end of the procedure, when the patient's haemodynamic state was stable, simultaneous left ventricular and aortic pressures were recorded. Aortography was not routinely repeated. The arterial sheath was removed, and haemostasis was achieved by pressure.

In one patient (a 70 year old woman with diabetes and peripheral arterial disease) dilatation was performed by a trans-septal approach through the right femoral vein.

Patients were admitted to the coronary care unit for

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observation for 24 hours and then transferred to a general ward.

Echocardiography and Doppler study were carried out with a standard Hewlett Packard system with a 2.5 MHz duplex transducer for echocardiographic imaging and pulsed wave Doppler studies and a 1.9 MHz transducer for continuous wave Doppler studies. A full study was performed from all conventional views. The peak aortic pressure gradient was calculated from the simplified Bernoulli equation (peak aortic gradient (mm Hg) = $4 \times \text{peak aortic velocity}^2$ (m/s)).^{12 13}

The area of the aortic valve was calculated by a modification of the continuity equation (area of aortic valve = subaortic area $\times$ peak subaortic velocity / peak aortic velocity); this method has been validated against measurement of the area of the valve by the Gorlin formula.¹⁴⁻¹⁷ To measure the subaortic velocity the sample volume was placed in the left ventricular outflow tract in both the apical long axis view and the apical four chamber view, and the maximum velocity was taken. Subaortic area was calculated from the subaortic diameter just below the aortic valve (from the leading edge of the left septal border to the leading edge of the anterior mitral leaflet) on freeze frame images recorded in the parasternal long axis view in early systole (subaortic area = $(\text{diameter}/2)^2 \times \pi$). Echocardiography was performed before the valve was dilated and again before the patient was discharged. When possible further studies were made at intervals of three months during follow up. Echocardiographic and Doppler measurements were made from prints by one observer (DCS).

Patients were questioned about their symptoms and effort tolerance before and one month after operation.

TABLE 1—Clinical characteristics of patients selected for balloon dilatation of aortic valve

Case No	Age (years)	Sex	Functional class*	Other disease
1	70	F	IV	Rheumatoid arthritis
2	81	M	III	
3	73	F	IV	Chronic airways obstruction, lung resection
4	76	F	IV	
5	73	M	III	Chronic airways obstruction
6	77	F	III	Polymyalgia rheumatica
7	68	F	IV	Coronary artery disease of one vessel†
8	73	M	III	Bronchial carcinoma
9	67	F	II	Chronic airways obstruction
10	84	F	III	Coronary artery disease of one vessel†
11	82	M	II	
12	83	F	III	Coronary artery disease of two vessels†
13	76	F	II	
14	72	M	III	Chronic airways obstruction
15	75	M	III	Chronic airways obstruction
16	79	M	III	Coronary artery disease of two vessels,† myocardial infarction
17	80	F	IV	Mitral regurgitation
18	68	F	III	Hypothyroidism; coronary artery disease of three vessels†
19	83	M	IV	Diabetes mellitus, coronary artery disease of one vessel,† myocardial infarction, chronic airways obstruction
20	86	F	III	Mitral regurgitation
21	65	M	II	Chronic airways obstruction, coronary artery disease of one vessel†
22	77	M	II	Colonic carcinoma
23	78	F	III	Diabetes mellitus, coronary artery disease of one vessel,† myocardial infarction, peripheral vascular disease
24	73	M	III	
25	70	F	III	Diabetes mellitus, coronary artery disease of two vessels†, peripheral vascular disease
26	78	F	III	
27	79	M	III	
28	81	F	III	Colonic angiodysplasia
29	83	F	IV	Chronic osteomyelitis
30	85	F	IV	
31	78	F	II	Coronary artery disease of one vessel†
32	67	M	II	Chronic airways obstruction

*New York Heart Association's classification.¹¹

†Reduction of $>70\%$ in luminal diameter of major vessel.

TABLE 2—Pressures before and after dilatation (mm Hg)

Case No	Left ventricular peak systolic pressure		Aortic peak systolic pressure		Peak to peak gradient		Diameter of balloons (mm)
	Before	After	Before	After	Before	After	
1	180	160	112	120	68	40	8, 12
2	224	210	118	118	106	92	8, 10, 12
3	230	130	114	76	116	54	4, 8, 10
4	222	222	132	144	90	78	8, 10
5	158	154	74	80	84	74	10
6	178	138	128	90	50	48	8, 10, 12, 15
7	194	158	120	104	74	54	12
8	178	136	150	114	28	22	8, 15
9	232	232	156	188	76	44	8
10	178	164	128	106	50	58	10
11	196	190	136	146	60	44	20
12	268	182	188	118	80	64	10, 15, 20
13	230	122	180	104	50	18	20
14	204	176	146	134	58	42	18, 20
15	184	180	140	156	44	24	20
16	218	216	128	136	90	80	15, 20
17	164	174	98	128	66	46	10, 20
18	204	152	148	104	56	48	20
19	166	146	136	120	32	26	10, 18
20	150	154	130	126	20	28	10
21	202	178	132	112	70	66	10+10, 10+10*
22	232	154	150	108	82	46	20
23	188	212	140	190	48	22	10
24	166	150	102	110	64	40	20
25	134	138	110	126	24	12	10, 20
26	238	226	136	176	102	50	15, 20
27	154	174	124	164	30	10	10, 20
28	202	178	138	128	64	50	10, 19, 20
29	178	140	102	110	76	30	20
30	208	164	110	122	98	42	20
31	258	238	198	188	60	50	20
32	208	244	144	170	64	74	20
Mean	198	175	133	129	65	46	
SD	32	34	26	30	24	20	

*Two balloons were used in this patient.

Their functional state was determined on 31 January 1988. Patients referred from other hospitals were contacted by telephone, or information about them was obtained from their doctor.

Measurements before and after dilatation were compared by Student's *t* test for paired data. The estimated one year survival rate and its standard error were calculated by the method of Cutler and Ederer.¹⁸ The χ^2 test was used to compare categorical variables in the patients who survived during follow up and those who died.

Results

Balloon dilatation of the aortic valve was performed on 32 patients between 31 January 1986 and 14 August 1987. Table 1 gives clinical characteristics of the patients. Their mean age was 76 (SD 6) years (range 65-86). Seven were in the New York Heart Association's functional class II, 17 in class III, and eight in class IV. Eleven had been referred from other hospitals.

The catheter balloon was satisfactorily placed in all patients. One to four balloons were used in each. The largest balloon used was 20 mm in diameter for 19 patients (including one patient in whom a dual balloon technique with 10 and 15 mm balloons was used) and 8-18 mm in diameter in 13. Cineangiography during inflation showed complete inflation and loss of the indentation by the stenotic valve in all cases.

Table 2 gives data on pressures before and after dilatation. Left ventricular systolic pressure fell from 198 (32) mm Hg to 175 (34) mm Hg after dilatation ($p < 0.001$). Aortic systolic pressure did not change significantly (133 (26) mm Hg before and 129 (30) mm Hg after dilatation; $p = 0.54$). The peak to peak gradient was reduced from 65 (24) mm Hg to 46 (20) mm Hg ($p < 0.001$). Aortography was repeated in eight patients; it showed no change in the degree of aortic regurgitation in seven and a slight increase (from 1+ to 2+) in one. The procedure (including left heart

catheterisation) took 104 (41) minutes, and screening time was 23 (15) minutes.

Major complications occurred in four patients, three of whom developed cardiac tamponade during or shortly after the procedure. In several patients the balloon had been seen to oscillate to and fro in the ventricle during inflation before it locked in the valve; presumably this movement drove the tip of the guide wire through the wall of the ventricle. In all cases tamponade was successfully treated by pericardiocentesis. One patient (a 73 year old woman who was in cardiogenic shock before dilatation) died four hours after the procedure after cardiac arrest; echocardiography had not shown recurrence of tamponade. The two other patients made uncomplicated recoveries.

Dissection of the femoral artery occurred in an 83 year old woman who presented in cardiogenic shock; because of the urgency the standard protocol of progressive dilatation of the femoral artery was not followed. Though the dissection was successfully repaired at operation, she remained in low output heart failure and died four days later.

Two patients developed haematomas in the groin but did not require operation. No patient sustained a stroke, and no embolisms were seen.

Table III shows the results of Doppler echocardiography. Recordings of aortic velocity were obtained

TABLE III—Doppler and aortographic measurements before and after dilatation

Case No	Peak aortic velocity (m/s)		Valve area (cm ²)		Grade of aortic regurgitation	
	Before	After	Before	After	Before	After
1	4.5	3.2	0.44	0.58	2+	2+
2	5.0	5.0	NA	NA	2+	NA
3	4.8	NA	NA	NA	1+	NA
4	5.6	5.3	0.43	0.45	2+	2+
5	3.9	3.7	NA	NA	1+	NA
6	4.1	2.7	NA	NA	0	0
7	4.8	4.6	NA	NA	2+	NA
8	3.0	2.9	NA	NA	1+	NA
9	4.0	4.6	0.81	0.85	1+	NA
10	4.0	NA	NA	NA	2+	NA
11	4.6	4.0	0.65	0.64	1+	1+
12	4.8	5.0	0.34	0.33	1+	NA
13	4.7	4.9	0.61	0.72	2+	NA
14	4.7	4.8	0.62	0.60	2+	NA
15	3.6	3.5	0.89	1.17	2+	NA
16	3.9	NA	NA	NA	1+	1+
17	4.5	3.8	0.58	0.52	2+	NA
18	4.5	4.4	NA	NA	0	NA
19	2.5	2.7	NA	NA	1+	NA
20	3.3	3.3	0.54	0.84	1+	2+
21	4.5	3.9	0.99	1.17	1+	1+
22	3.5	2.9	0.58	0.73	1+	NA
23	3.6	NA	NA	NA	1+	NA
24	3.9	3.1	0.61	0.93	2+	NA
25	2.8	2.9	0.69	0.88	2+	NA
26	4.9	4.7	0.57	0.53	1+	NA
27	2.5	3.2	NA	NA	1+	NA
28	4.7	4.0	0.53	0.73	0	NA
29	NA	2.5	NA	NA	NA	NA
30	5.0	3.4	NA	NA	1+	NA
31	3.9	4.0	0.71	0.90	1+	1+
32	4.9	4.0	0.43	0.55	NA	NA

NA=Not assessed.

TABLE IV—Area of aortic valve (cm²) before and after dilatation and at follow up (mean 23 weeks) in 11 patients

Case No	Before	After	At follow up
1	0.44	0.58	0.52
4	0.43	0.45	0.46
9	0.81	0.85	0.69
13	0.61	0.72	0.59
14	0.62	0.60	0.83
15	0.89	1.17	0.83
25	0.69	0.88	0.85
26	0.57	0.53	0.52
28	0.53	0.73	0.58
31	0.71	0.90	0.57
32	0.43	0.55	0.52
Mean	0.61	0.72	0.63
SD	0.15	0.21	0.14

both before and after dilatation of the valve (mean four days) in 28 patients. Peak aortic velocity fell from 4.2 (0.8) m/s to 3.9 (0.8) m/s ($p<0.05$), representing a fall in the estimated peak instantaneous aortic gradient from 71 to 61 mm Hg.

The area of the aortic valve was assessed by Doppler echocardiography in 18 patients and increased from 0.61 (0.16) cm² to 0.74 (0.23) cm² ($p<0.001$) (figure 1). Dilatation had a variable effect on the area. In six patients the area changed by $\leq 10\%$ and in 12 patients it increased by 16–56%, becoming >0.7 cm² in 11 patients but >1.0 cm² in only two. In 11 of 18 patients the area of the valve was measured again six to 50 (mean 23) weeks after dilatation (table IV; figure 2). The mean area had increased from 0.61 (0.15) cm² to 0.72

FIG 1—Area of aortic valve (measured by Doppler echocardiography) before and 1-7 (mean 4) days after dilatation in 18 patients. Vertical bars indicate mean (SD)

FIG 2—Area of aortic valve before dilatation, before discharge (1-7 days after operation), and at follow up (6-50 (mean 23) weeks after dilatation) in 11 patients. Vertical bars indicate mean (SD)

(0.21) cm² before discharge but fell to 0.63 (0.14) cm² at follow up. In seven patients the area was less than before dilatation. The mean area at follow up did not differ significantly from that before dilatation ($p=0.6$).

Among the 29 patients alive one month after the procedure symptoms had improved in 17, were unchanged in 11, and were worse in one. Four patients in whom the area of the valve changed by $\leq 10\%$ reported an improvement in symptoms. Among the 12 patients in whom the area of the valve increased, symptoms were improved in six, unchanged in five, and worse in one.

Symptoms recurred or worsened in most patients during follow up, and the mortality of the group was high. On 31 January 1988, 16 patients were alive; the mean duration of follow up was 10 months (range 3–24). Two patients had had the aortic valve replaced and four were awaiting operations. Only two of the remaining 10 patients had lasting clinical benefit from balloon dilatation. A 73 year old man, previously in the New York Heart Association's class III, had an unlimited effort tolerance after eight months; the area of the valve

had increased from 0.61 to 0.93 cm² after dilatation. A 76 year old woman had improved effort tolerance after 13 months, though she was still in the New York Heart Association's class II; the area of the valve had increased from 0.61 to 0.72 cm² after dilatation but had fallen to 0.59 cm² at eight months. Neither was willing to undergo valve replacement.

Sixteen patients died (table V). The estimated one year survival rate was 49% (SE=0.1015, 95% confidence interval 29% to 69%). Death was attributable to a cardiac cause in 12 patients (including three who died suddenly). Patients who died did not differ from those who survived in mean age (76 (6) years in both groups), but a greater proportion who died were in the New York Heart Association's class III or IV before dilatation (15/16 v 10/16 who survived; $\chi^2=2.9$, df=2, p=0.08) or had a blood urea concentration >10 mmol/l (13/16 v 4/16; $\chi^2=8.0$, df=2, p=0.005).

TABLE V—Causes of death after dilatation

Case No	No of months survived	Cause of death
3	0	Cardiac arrest*
29	0	Heart failure
17	0	Heart failure
19	2	Gastrointestinal haemorrhage*
27	3	Heart failure
16	3	Heart failure
25	3	Not established
5	4	Sudden death (cardiac cause)
23	4	Cardiac arrest
20	4	Not established
8	6	Bronchial carcinoma
4	7	Heart failure
1	9	Heart failure*
12	12	Sudden death (cardiac cause)*
9	13	Sudden death (cardiac cause)
7	13	Myocardial infarction*

*Necropsy performed.

Discussion

Our results show that balloon dilatation of the aortic valve achieved only a modest, variable, and temporary improvement in valve function in elderly patients who had severe calcific aortic stenosis. Degenerative calcific disease is probably the commonest cause of aortic stenosis in this age group.^{19,21} The area of the valve is reduced because rigidity of the cusps (due to calcium deposits) prevents full opening. In contrast with rheumatic or congenital valvular stenosis, commissural fusion is absent or unimportant; thus one of the principal mechanisms of balloon dilatation, the splitting of fused commissures, cannot operate. Necropsy and intraoperative studies have shown that balloon inflation may fracture nodular calcific deposits and stretch cusp tissue,²² although the extent to which the area of the valve is increased is disputed.^{23,24}

Other workers have obtained greater increases in the area of the valve in populations that closely resembled ours; they used a similar proportion of catheter balloons of 20 mm diameter.⁶⁻¹⁰ In the largest series from a single centre Cribier *et al* reported an increase in the area of the valve from 0.49 to 0.93 cm² in 92 patients; the resultant area was >0.7 cm² in 79% and >1.0 cm² in 32% of patients.⁷ The French registry of 507 patients undergoing valvar dilatation in 27 centres, however, reported a resultant area of >0.7 cm² in only 55% of patients.²⁵

Restenosis after dilatation may reflect the "healing" of fractures in calcific nodules, stretched cusp tissue resuming its former shape, or (as degenerative calcific aortic stenosis is progressive²⁶) further deposition of calcium in the cusps. The high incidence of restenosis that we observed differs appreciably from the incidence found in previous studies. McKay *et al* reported that measurement of aortic cusp excursion on M mode echocardiography six weeks after dilatation of the

aortic valve showed no evidence of restenosis in 17 of 32 patients.⁶ This method of assessing valve function in aortic stenosis has important limitations.²⁷ Cribier *et al* catheterised 12 of 92 patients between four and 24 weeks (mean 14) after dilatation and found that the reduction in peak to peak gradient had been maintained; measurement of the area of the valve by the Gorlin formula in 10 patients showed no evidence of restenosis (mean area 0.76 cm² immediately after dilatation and 0.75 cm² after reinsertion of the catheter).⁷ Clinical information was not given about this small subset of patients, who may not have been representative.

Most of our patients reported an improvement in symptoms and effort tolerance one month after dilatation. Progression or treatment of accompanying cardiac or non-cardiac disease, both of which were common in our patients, may have accounted for the lack of a relation between this response of symptoms and the effect of dilatation on the area of the valve. In addition, patients' reporting of symptoms may have been influenced by their expectations of a new form of treatment. The limitations of a history in assessing effort tolerance are recognised,^{28,29} and a suitable objective measurement (such as the distance walked in six minutes³⁰) would be preferable for future studies.

The management of patients whose symptoms recur or worsen after balloon dilatation is difficult. Dilatation was repeated (without complication) in six patients, all of whom were in the New York Heart Association's class III or IV. The interval between procedures ranged from three to 12 (mean six) months. The mean area of the valve increased slightly but not significantly (from 0.61 (0.22) to 0.68 (0.23) cm²). Symptoms improved in only one patient.

Even though our patients were elderly and many had other disease, unrelieved aortic stenosis probably made an important contribution to the high mortality seen during follow up. The poor prognosis of symptomatic aortic stenosis is well recognised.³¹⁻³³ The area of the valve was less than 0.7 cm² (that is, in the range defined as severe stenosis) in seven of 18 patients shortly after dilatation and in eight of 11 at follow up. Unless dilatation can consistently create an orifice with an area comparable with that of a prosthetic aortic valve (in most cases 1.0-1.5 cm² or more)^{34,37} it is unlikely to confer the improved life expectancy that follows successful valve replacement.² This cannot be achieved at present, and we therefore believe that valve dilatation should be used only for patients with severe symptoms in whom life expectancy is limited by other disease. Dilatation may have a place in the management of patients with aortic stenosis who present with cardiogenic shock or severe pulmonary oedema. The high mortality after emergency operations to replace aortic valves in the elderly³ might be reduced if left ventricular function could be improved before operation.

We thank the nursing staff of the catheter laboratory and acute cardiac ward and Marilyn Moore, superintendent radiographer, for skilled help. DCS is funded by a donation to the cardiac department from Mr Kerry Packer. JBC holds a British Heart Foundation junior fellowship.

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(Accepted 14 July 1988)

Cardiac predictors of death after non-cardiac surgery evaluated by intention to treat

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Abstract

Cardiac risk factors were studied among patients who were admitted to hospital with appendicitis or a fracture of the proximal femur less than one year after being admitted with myocardial infarction. Of 99 patients with myocardial infarction and appendicitis, 87 underwent appendicectomy; and of 221 with myocardial infarction and hip fracture, 179 were operated on. The patients were studied on an intention to treat basis. The mortality within one month was 9% and 16% respectively. A history of congestive heart failure was the dominating risk factor, while ischaemic heart disease (recent myocardial infarction or angina pectoris) had no independent association with mortality.

If the ventricular function is known additional preoperative information about the heart is of negligible value when estimating the mortality of non-cardiac surgery.

Introduction

Congestive heart failure and ischaemic heart disease are considered to be the most important predictors of death after non-cardiac surgery.^{1,2} Of the ischaemic heart diseases, a recent myocardial infarction is regarded as the most ominous condition, while an older infarction or angina pectoris is less important.^{3,5} A recent myocardial infarction is usually defined as one occurring within the preceding six months, and mortality after surgery is typically found to be 10 times

higher in patients with recent infarction.⁴ Earlier studies did not take into account the possibility that awareness of risk factors may influence the selection of patients for operation. They were based on a broad range of diseases and many different surgical procedures, including elective operations. They also presented information only about patients who had been operated on—that is, they did not contain any data on patients who for various reasons (for example, recent myocardial infarction) were treated conservatively.

The purpose of the present study was to evaluate cardiac risk factors as predictors of operative death in an adequate number of patients with one specific disease who were all candidates for the same operation, thus minimising the variability associated with differences in disease and operation. We selected two acute conditions (appendicitis and hip fracture) that usually need urgent surgical treatment and analysed data according to the principle of intention to treat. Furthermore, to ensure a high frequency of cardiac risk factors in the study population we confined the study to patients who had suffered myocardial infarction in the year preceding the appendicitis or hip fracture.

Patients and methods

The national registry of hospitalised patients was established in Denmark in 1976.⁶ The data provided by this registry include diagnoses and operations

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