European Heart Journal (1988) **9**, 782–794

Percutaneous balloon valvuloplasty for calcific aortic stenosis. A treatment 'sine cure'?

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KEYWORDS: Aortic valvuloplasty, complications, follow-up.

Twenty-five elderly patients with calcific aortic stenosis, 12 male (48%) and 13 female (52%), mean age 74·8 ± 7·6 years, underwent percutaneous aortic balloon valvuloplasty between March 1986 and September 1987. Twenty-two patients (88%) were in class III–IV of the New York Heart Association, 13 (52%) had a history of previous angina and 7 (28%) of syncopal attacks. All patients had been considered either unsuitable or high-risk candidates for aortic-valve replacement because of age or associated diseases. Balloons of increasing size (area ranging from 1·3 to 3·8 cm^2 during inflation) were successively passed retrogradely from the femoral artery and manually inflated to 3–7 atmospheres. Inflation duration ranged from 15 to 260 s (mean 40 s). Post-dilatation there were significant changes in left ventricular peak-systolic and end-diastolic pressures ($P < 0·00001$ and $P < 0.01$, respectively), mean systolic aortic transvalvular gradient (from 73 to 43 mmHg, $P < 0·000001$), mean systolic aortic flow (from 176 to 208 ml s^{-1}, $P < 0·0001$) and aortic valve area (from 0·47 to 0·72 cm^2, $P < 0·000001$). Major complications included: in-hospital deaths of two patients (8%) admitted in cardiogenic shock; left haemiplegia (4%); transient haemianopia (8%); development of grade III aortic insufficiency (4%); and persistent complete atrioventricular block (4%). Complications at the puncture-site occurred in 7 patients (28%)—including two femoral pseudoaneurysms and the need for surgical removal of a balloon remnant after rupture in one patient. No local haemorrhagic complications were observed in the latter eight procedures, performed using a 16·5 French 100-cm long arterial introducer. At a mean follow-up of 13·0 ± 5·0 months, an important functional improvement persisted in 14 patients (56%), no major changes in pre-valvuloplasty symptoms were observed in 3 patients (12%), while five patients (20%) required surgical treatment after a successful valvuloplasty because of recurrence of symptoms (late valve restenosis). Percutaneous aortic balloon valvuloplasty is a possible palliative therapy in elderly patients with calcific aortic stenosis. However, its inherent immediate risk, limited haemodynamic result and the possible development of valve restenosis at medium-term follow-up, suggest that the application of this technique should be limited to poor surgical candidates.

Introduction

In September 1985, Cribier *et al.* first applied balloon dilatation to inoperable elderly patients with calcific aortic stenosis[1]. Although hundreds of percutaneous aortic valvuloplasties have been performed up to now[2–11], and more follow-up data have become available[8,12], continuing technical improvements and rapidly changing indications have precluded a definite assessment of the therapeutic role of this procedure. In particular, the application of aortic valvuloplasty in young adult patients, and in older patients who have a relatively low risk of elective valve replacement, is still controversial[13,14]. In this paper we describe complications and immediate and medium-term results of 25 patients who have undergone aortic balloon valvuloplasty at the Thoraxcenter since March 1986. The present indications and future expectations for this new invasive cardiological treatment are also discussed.

Submitted for publication on 16 December 1987 and in revised form 25 March 1988.

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H. E. Luijten is the recipient of a Fellowship from the Dutch Heart Foundation (85-118). K. J. Beatt is the recipient of the Joint Fellowship from the British and Netherlands Heart Foundation.

0195–668X/88/070782 + 13 $02.00/0

Methods

PATIENT POPULATION

Since March 1986, percutaneous balloon valvuloplasty has been performed in 25 elderly patients with calcific aortic stenosis, all severely disabled with dyspnoea [16 (64%) in class III and 6 (24%) in class IV of the New York Heart Association], angina pectoris (13 patients, 52%), or syncope on effort (7 patients, 28%). In two patients the procedure was performed immediately after being admitted to our Intensive Care Unit in cardiogenic shock, requiring treatment with intravenous dopamine and dobutamine. The mean age was 74·8 ± 7·6 years (range 60–88 years). Twelve patients were male (48%) and 13 female (52%). The aetiology was inflammatory (rheumatic) in one patient and degenerative in the remaining patients. Angiographic findings suggestive of a congenitally bicuspid aortic valve were present in 3 patients (12%). Most of the patients presented with associated cardiovascular or non-cardiovascular diseases, including: (a) coronary artery diameter reduction ⩾ 50% (12%)—a complete occlusion of the right coronary artery and a 60% luminal narrowing of the main stem of the left coronary artery was observed in one patient; (b) systemic hypertension (8%); (c) respiratory insufficiency (16%); (d) diabetes mellitus (12%); (e) arterial claudication (12%); (f) chronic renal insufficiency (8%); (g) severe anemia (8%); (h) rheumatoid arthritis (8%); and (i) previous mitral commissurotomy because of rheumatic mitral valve stenosis (4%).

It was felt that, balloon valvuloplasty was justified because of the existent, age-related, high surgical risk (14 patients > 75 years old, 56%) and the presence of concomitant diseases. Informed consent was obtained from all the patients.

VALVULOPLASTY PROCEDURE

Percutaneous balloon aortic valvuloplasty immediately followed the diagnostic catheterization in 8 patients (32%) while valvuloplasty was performed during a separate session in the remaining patients. Left ventricular and aortic pressures, and mean systolic transaortic gradients were averaged and calculated on-line, after acquisition periods of 20 s. Cardiac output was computed from duplicate thermodilution measurements. A computerized system also provided instant calculation of the mean systolic aortic flow and aortic valve area[15]. Basal haemodynamic data are reported in Table 1.

Left ventricular end-diastolic and end-systolic volume indices were 98 ± 20 ml m^{-2} and 57 ± 26 ml m^{-2}, respectively. Left ventricular ejection fraction ranged from 14 to 65% (mean 42%), and left ventricular weight index ranged from 136 to 241 g m^{-2} (mean 173 g m^{-2}).

A trace of aortic insufficiency was present in 9 patients (36%), and mild insufficiency in 8 (32%).

Before starting aortic ballon valvuloplasty, 1·0 mg of atropine and 5000 I.U. of heparin were administered intravenously. The percutaneous femoral route was used in all patients. In the first 17 patients, a straight 0·038 inch, 260-cm long, guidewire (Cordis Corporation, Miami) with its soft tip manually fashioned to a large J shape, was advanced into the left ventricle. A balloon catheter was then advanced over it and positioned across the aortic valve. Before insertion, particular care was taken to remove air from the balloon. In the remaining 8 patients, a 0·035 inch, 300-cm long, 'back-up' guide-wire (Schneider Shiley, Zürich) was used. Over the distal stiffer part of this guide-wire a 16·5 French 100-cm long valvuloplasty introducer (Schneider Shiley, Zürich) was positioned immediately above the aortic valve, in an attempt to facilitate introduction and removal of multiple, large balloon catheters. Inflations were performed by hand injection of a mixture of contrast medium and saline (30/70), with pressures of 3–7 atmospheres. Balloons of increasing diameter, varying from 13 mm (area 1·3 cm^2) circular balloons to 3 × 12 mm (3·8 cm^2, equivalent area) trefoil balloons, were used in an attempt to reduce the mean aortic transvalvular gradient to < 40 mmHg, and to increase the aortic valve area to ⩾ 1·0 cm^2. Inflation duration ranged from 15 to 260 s (mean 40 s), depending upon the individual tolerance. In most patients only a moderate decrease of systemic arterial pressure, continuously monitored throughout the procedure, was observed during balloon inflation. However, the maximal systolic aortic pressure transiently fell to ⩽ 60 mmHg in 13 patients (52%), resulting in dizziness and/or promptly reverisble syncopal attacks in six cases (24%). The mean number of balloons used per patient was 2·6 (range 1–6). In some of the first patients, 80-mm long balloons were used in order to achieve a stable position during inflation. In the latter patients shorter balloons could be used because to-and-fro motion across the aortic valve during dilatation was prevented by positioning the long sheath immediately above the inflated balloon (Fig. 1). At the end of the procedure, haemodynamic results and aortic regurgitation were systematically re-evaluated.

Table 1 Haemodynamic data before and after aortic valve dilatation in 25 patients

Case No.	LVPSP		LVEDP		PSG		MSG		AF		AA	
	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After
1	247	229	24	11	56	28	57	39	179	245	0·53	0·88
2	275	222	31	16	98	68	78	56	234	270	0·59	0·81
3	264	177	15	2	66	35	81	31	134	197	0·33	0·80
4	230	188	16	19	73	22	67	28	136	166	0·38	0·71
5	204	210	23	23	54	36	48	41	152	163	0·49	0·57
6	238	212	12	2	105	70	80	58	136	120	0·34	0·36
7	203	188	28	15	47	33	53	37	231	259	0·71	0·96
8	191	177	30	27	90	40	68	41	125	181	0·37	0·64
9	275	240	17	12	119	66	118	64	198	230	0·47	0·65
10	197	223	27	17	58	23	50	31	193	258	0·61	1·04
11	257	179	13	8	135	51	105	49	266	286	0·58	0·92
12	235	154	27	3	127	52	96	52	177	229	0·41	0·72
13	234	193	11	14	75	40	74	47	176	214	0·46	0·70
14	190	148	13	23	65	18	62	26	222	192	0·63	n.c.
15	215	188	5	7	47	24	60	39	217	238	0·63	0·85
16	225	175	9	14	96	28	90	32	113	153	0·27	0·61
17	203	n.r.	28	n.r.	113	n.r.	82	n.r.	235	n.r.	0·58	n.r.
18	223	192	4	8	66	47	56	53	127	205	0·38	0·64
19	234	151	14	2	90	40	75	31	191	195	0·49	0·79
20	221	163	8	5	100	84	70	58	289	290	0·78	0·86
21	115	n.r.*	32	n.r.*	40	n.r.*	38	n.r.*	110	n.r.*	0·40	n.r.
22	150	116	20	18	54	24	37	28	119	175	0·44	0·75
23	180	181	11	11	72	37	89	49	133	151	0·31	0·49
24	291	193	30	17	127	59	109	62	171	231	0·37	0·66
25	231	199	14	12	120	24	90	40	132	130	0·31	0·46
Mean	221	187	18	12	84	41	73	43	176	208	0·47	0·72
±s.d.	±33	±29	±8	±7	±29	±18	±21	±12	±50	±49	±0·14	±0·17
	$P<0{\cdot}00001$		$P<0{\cdot}01$		$P<0{\cdot}000001$		$P<0{\cdot}000001$		$P<0{\cdot}0001$		$P<0{\cdot}000001$	

AA, aortic valve area (cm^2); AF, systolic aortic flow ($ml\ s^{-1}$); LVEDP, left ventricular end-diastolic pressure (mmHg); LVPSP, left ventricular peak-systolic pressure (mmHg); MSG, mean systolic aortic gradient (mmHg); PSG, peak systolic aortic gradient (mmHg).

n.c., not computed because of significant aortic valve insufficiency after valvuloplasty; n.r., not recorded at the end of the last balloon inflation; n.r.*, not recorded because of patient's demise immediately after the first balloon inflation.

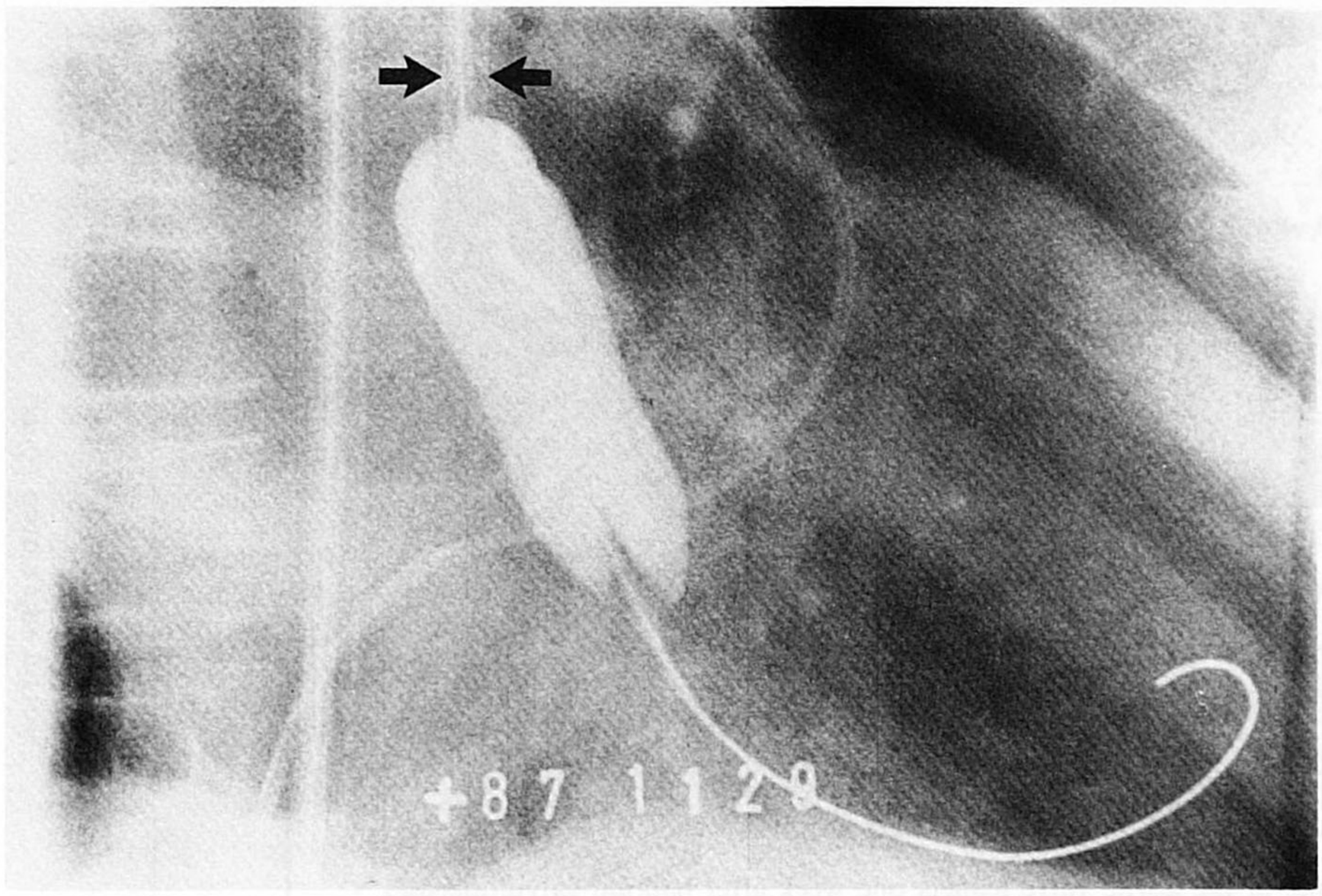

Figure 1 A 3 × 12, 40-mm long trefoil balloon (Schneider Medintag, Zürich) is inflated across the calcific stenotic aortic valve. The presence of the sheath (arrows) immediately above the balloon, prevents possible to-and-fro motion during inflation, allowing the use of shorter balloons.

STATISTICAL ANALYSIS

Student's *t*-test for paired data was used whenever appropriate.

Results

IMMEDIATE HAEMODYNAMIC RESULTS AND IN-HOSPITAL COMPLICATIONS

Haemodynamic data before and after aortic balloon dilatation are reported in Table 1. In Case 14, the aortic valve area could not be computed after the procedure because of the development of a grade III aortic insufficiency. In this patient, an increase in aortic regurgitation was suspected on the basis of a sudden reduction in diastolic aortic pressure shortly after the second inflation with a 19-mm balloon. The patient underwent surgery one day later, when he was in a stable haemodynamic condition. The congenitally biscuspid valve consisted of extensive areas of hyaline degeneration and calcification. Histology showed a disruption of the calcified mass within one of the leaflets. No macroscopic changes could be observed in the other, more heavily calcified, leaflet. It is noteworthy that the area between the calcium fragments showed recently formed scar tissue, predominantly consisting of young fibroblasts (Fig. 2).

In Case 17, a minimal increase in aortic valve area (from 0·58 to 0·64 cm^2) was observed after two inflations with a 19-mm circular balloon. During the second inflation with a 3 × 12 mm balloon, both the catheter and the guide-wire were pushed out of the left ventricule. No attempt at recrossing the valve was undertaken because of marked blood loss at the puncture site. In Case 21, a moribund patient in cardiogenic shock with severe obstructive coronary artery disease, marked left ventricular dysfunction (left ventricular ejection fraction 14%) and renal insufficiency, a further, progressive decrease of systemic arterial pressure began immediately after the first 20-s inflation with a 3 × 12 mm trefoil balloon. Despite increased inotropic support (dopamine and dobutamine infusion) arterial pressure could not be raised, resulting in the patient's demise. Comparison between pre- and post-valvuloplasty values in the remaining patients revealed highly significant changes in left ventricular peak-systolic and end-diastolic pressures ($P < 0{\cdot}00001$ and $P < 0{\cdot}01$, respectively), mean and peak systolic aortic gradients ($P < 0{\cdot}000001$), mean systolic aortic flow ($P < 0{\cdot}0001$), and aortic valve area ($P < 0{\cdot}000001$) (Figs 3 and 4). However, when the individual absolute values after valvuloplasty are considered, the presence of a mean systolic

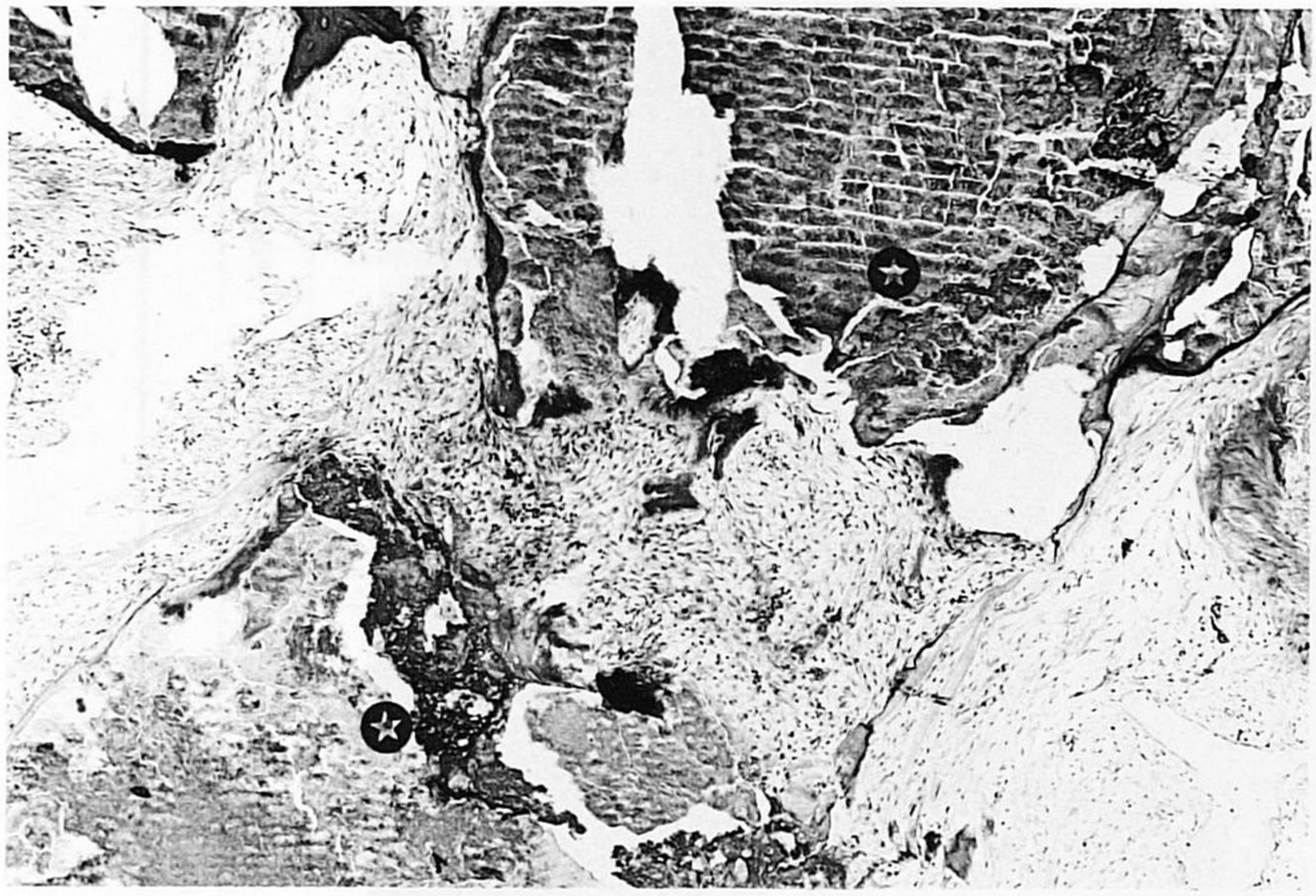

Figure 2 Histology of one of the aortic valve leaflets one week after balloon valvuloplasty. A disruption between the calcium fragments (indicated by stars) has already been filled in with young scar tissue, consisting mostly of fibroblasts (haematoxylin–eosin stain).

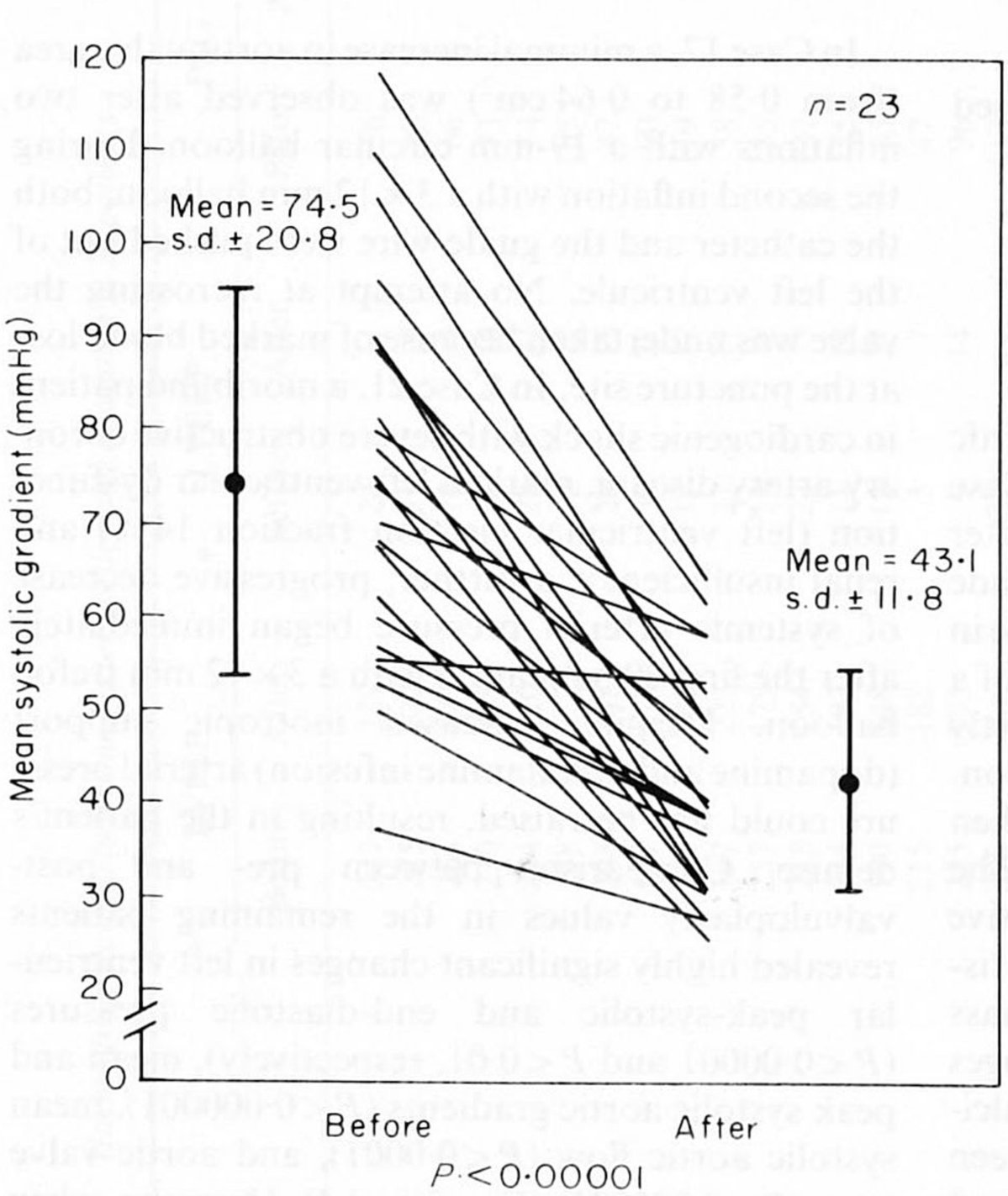

Figure 3 Mean systolic aortic gradient before and after percutaneous balloon aortic valvuloplasty.

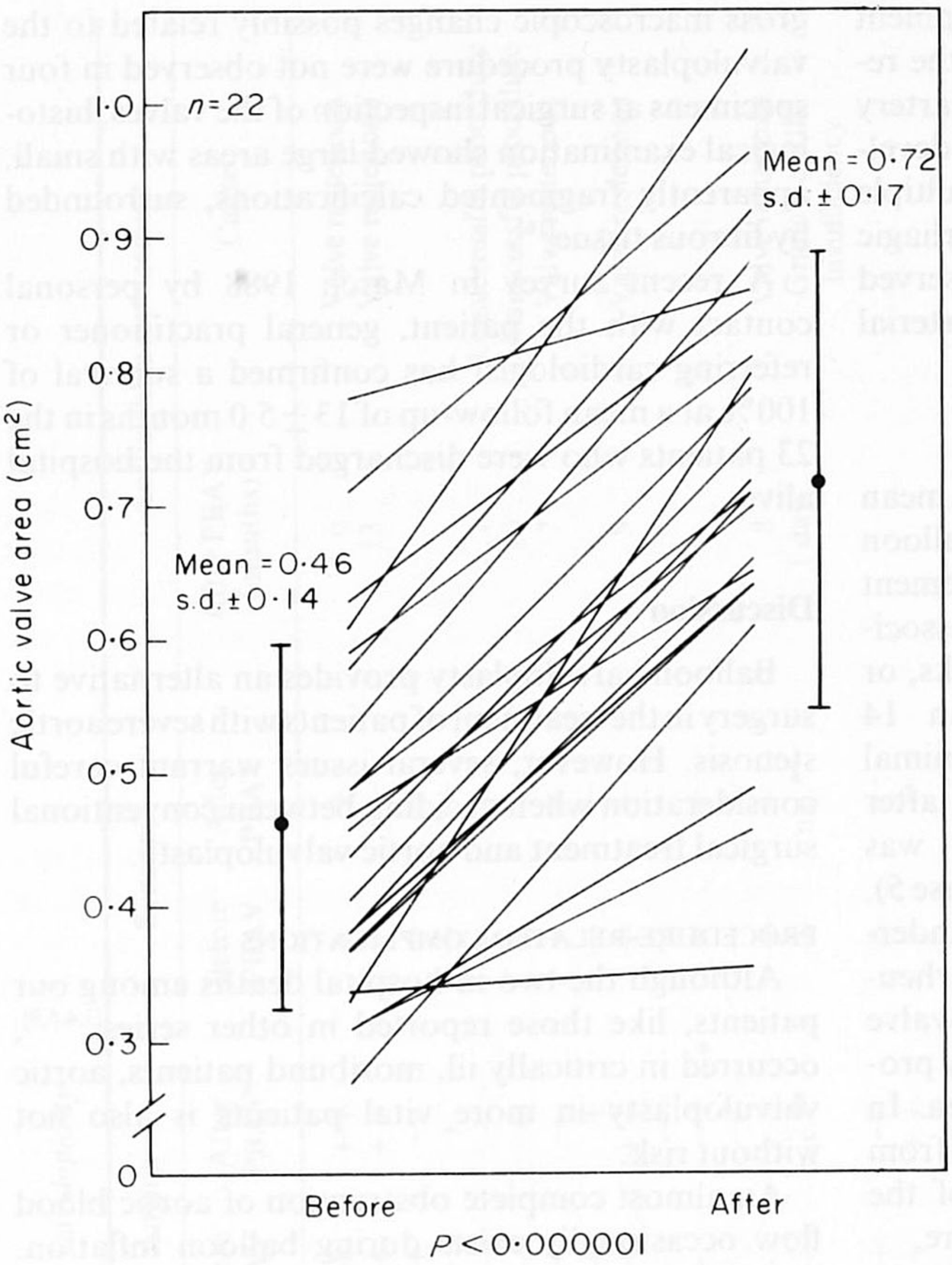

Figure 4 Aortic valve area before and after percutaneous balloon aortic valvuloplasty.

gradient $\geqslant 40$ mmHg in 12 patients (48%), and an aortic valve area $<0{\cdot}7\,cm^2$ in 9 patients (36%), should be emphasized.

A complete left haemianopia developed in patients 3 and 15 during the procedure. In the former patient, minimal residual defects in the field of vision were still present at follow-up, while a complete and prompt recovery was experienced by the latter patient. In patient 10 a left haemiplegia developed at the end of the aortic valvuloplasty, with persistent impairment of the mobility of the left arm at discharge from the hospital.

In patient 22, a 77-year-old man admitted to our Intensive Care Unit in cardiogenic shock, mentally confused, anuric, and with a left ventricular ejection fraction of 16%, the valve area was increased from 0·44 to $0{\cdot}75\,cm^2$ as a result of valvuloplasty. Although temporary clinical benefit was observed in the first days after the procedure, all attempts at reducing the inotropic intravenous support were unsuccessful and the patient died in cardiogenic shock 10 days after the procedure. Thus, the overall in-hospital mortality was 8%. At autopsy, no cardiac or vascular damage possibly related to the procedure could be observed.

Marked increase in aortic valve insufficiency, requiring elective aortic valve replacement, was observed in patient 14. A slight increase in aortic valve regurgitation occurred in 6 patients (24%), while disappearance of a trace of aortic insufficiency was observed in two patients (8%). Two patients (8%, cases 12 and 22) had to be promptly resuscitated because of ventricular fibrillation, which occurred during balloon inflation in one patient.

Patient 25 developed complete atrioventricular block immediately after insertion of the guide-wire into the left ventricle, which required implantation of a permanent pacemaker.

Three patients required surgical intervention at

the puncture site, two because of the development of a femoral pseudoaneurysm, and one for the removal of a balloon remnant from the femoral artery after balloon rupture. Four patients (16%) developed a large groin haematoma, requiring multiple blood transfusions in two cases. No haemorrhagic complications at the puncture site were observed in the latter eight procedures, when an arterial valvuloplasty introducer was used.

FOLLOW-UP

Table 2 illustrates the clinical follow-up at a mean interval of $13{\cdot}0 \pm 5{\cdot}0$ months after aortic balloon valvuloplasty. A major symptomatic improvement (reduction of at least one New York Heart Association Class, disappearance of syncopal attacks, or increase in angina threshold) persisted in 14 patients (56%). In three cases (12%) with minimal haemodynamic improvement immediately after aortic valvuloplasty, symptomatic status was unchanged. The youngest of these patients (Case 5), a 65-year-old woman who had previously undergone mitral valve replacement because of rheumatic mitral valve stenosis, required an aortic-valve replacement one month after the valvuloplasty procedure because of persistent, severe dyspnoea. In patient 10 the physical impairment resulting from the left haemiplegia precluded evaluation of the functional result of the valvuloplasty procedure.

Five patients with at least one year of follow-up (cases 1, 2, 7, 9 and 13) achieved a satisfactory immediate haemodynamic result (increase in aortic valve area from $0{\cdot}55 \pm 0{\cdot}10$ to $0{\cdot}80 \pm 0{\cdot}13$ cm^2), and experienced a significant functional improvement following aortic valvuloplasty. However, at a mean follow-up of $8{\cdot}6 \pm 2{\cdot}7$ months (range 6–13 months), deterioration of the effort tolerance and recurrence of angina and/or syncopal attacks required surgical aortic valve replacement. In these patients aortic valve replacement was decided against prior to the valvuloplasty on the basis of patient preference in one, and in the other four due to the presence of concomitant disease(s) and/or old age (mean age 72 years) so that they were initially considered unsuitable or high-risk candidates for surgery. Echo-Doppler examination showed a late increase in the peak aortic flow velocity relative to the values observed immediately after the valvuloplasty procedure in all of these patients. In patient 9 serial transoesophageal echocardiograms showed that the moderate increase in valve mobility, particularly of the non-coronary cusp, could no longer be observed 5 months after the procedure. Although gross macroscopic changes possibly related to the valvuloplasty procedure were not observed in four specimens at surgical inspection of the valves, histological examination showed large areas with small, apparently fragmented calcifications, surrounded by fibrous tissue[16].

A recent survey in March 1988 by personal contact with the patient, general practitioner or referring cardiologist has confirmed a survival of 100% at a mean follow-up of $13 \pm 5{\cdot}0$ months in the 23 patients who were discharged from the hospital alive.

Discussion

Balloon valvuloplasty provides an alternative to surgery in the treatment of patients with severe aortic stenosis. However, several issues warrant careful consideration when deciding between conventional surgical treatment and aortic valvuloplasty.

PROCEDURE-RELATED COMPLICATIONS

Although the two in-hospital deaths among our patients, like those reported in other series[3,4,6,8], occurred in critically ill, moribund patients, aortic valvuloplasty in more vital patients is also not without risk.

An almost complete obstruction of aortic blood flow occasionally exists during balloon inflation. However, potentially ominous effects of a prolonged impairment of both the cerebral and coronary circulations can be easily avoided with short-lasting balloon inflations.

Our clinical observations and the results of previous experimental studies[17] suggest that careful monitoring of electrocardiographic changes and the development of chest pain are necessary during long balloon inflations, because direct apposition of vegetative calcifications with protrusion into the sinuses of Valsalva can significantly impair coronary blood flow in spite of a preserved systemic arterial pressure.

Major injury to peripheral vessels occurred in 28% of our patients, and similar rates have been reported by most authors[5,8,11]. A large arterial sheath, designed to facilitate introduction and removal of balloon catheters of different sizes and to minimize injury to femoral and iliac vessels, was applied only in our latter patients. The clinical benefit of the long-sheath technique, applied in the last eight cases, was readily apparent and has been confirmed in more recent cases, since not a single vascular complication in peripheral vessels was

Table 2 Clinical follow-up data 4–23 months after percutaneous balloon aortic valvuloplasty

Case No.	Follow-up time (months)	NYHA class		Angina		Syncope		Aortic valve replacement	
		Before PBAV	After PBAV	Before PBAV	After PBAV	Before PBAV	After PBAV	After PBAV (months)	Cause
1	23	III	III	++	++	+	+	6	Valve restenosis
2	22	II	II	++	++	—	—	13	Valve restenosis
3	22	II	II	++	—	—	—		
4	18	III	II	+++	+	—	—		
5	17	III	III	—	—	—	—	1	Unsuccessful procedure
6	17	III	III	—	—	—	—	10	Unsuccessful procedure
7	15	III	III	—	—	+	+	7	Valve restenosis
8	15	IV	II	++	+	—	—		
9	15	III	III	++	++	—	—	9	Valve restenosis
10	14	III	n.e.*	—	—	—	—		
11	13	III	II	—	—	+	—		
12	12	IV	II	—	—	—	—		
13	12	III	II	+	—	—	—	8	Valve restenosis
14	12	III	n.e.**	++	n.e.**	—	n.e.**	1 day	Grade 3 aortic insufficiency
15	12	III	II	++	—	—	—		
16	11	III	II	++	++	+	—		
17	11	II	II	—	—	—	—		
18	11	III	II	++	—	—	—		
19	10	III	II	—	—	+	—		
20	9	III	III	—	—	—	—		
21	9	IV	†	+++	†	+	†		
22	9	IV	†	—	†	—	†		
23	8	IV	II	—	—	+	—		
24	4	III	II	++	—	—	—		
25	4	IV	II	—	—	—	—		
Mean ±s.d.	13·0 5·0								

NYHA, New York Heart Association; PBAV, percutaneous balloon aortic valvuloplasty.
Angina: —, absent; +, angina on strenuous effort; ++, angina on moderate effort; +++, angina on minimal effort or at rest.
Syncope: —, absent; +, present.
n.e*., not evaluable because of limited physical activity (left haemiplegia); n.e.**, not evaluable because of aortic-valve replacement the day after the procedure (grade 3 aortic-valve insufficiency).
†, In-hospital patient death.

observed in this series of patients, now a total of 13[18]. Nevertheless, a further decrease in the diameter of both introducers and valvuloplasty catheters is desirable.

Perforation of the left ventricle has been observed during the first procedures performed by Letac and Cribier[19]. To prevent this from occurring the same authors have proposed the use of a straight exchange guide-wire with the tip manually fashioned into a large J. The currently available J-shaped, floppy tip valvuloplasty guide-wires and pig-tail balloon catheters have decreased the procedural risks of mechanically induced myocardial trauma and of major ventricular arrhythmias. Furthermore, the continuous assessment of left ventricular and supravalvular aortic pressures is feasible with the recently introduced dilatation catheters, although these pressures can also be recorded through the inner lumen of the introducer when the long-sheath technique is employed.

Detachment of valvular calcium deposits with subsequent embolization may lead to catastrophic neurologic sequelae. However, valvuloplasty related embolic phenomena are infrequently observed, probably because of the subendocardial distribution of the calcium particles and the lack of direct contact between the balloon and the calcific tissue, which is predominantly concentrated within large nodular calcifications on the aortic side of the leaflets[20]. Valvular calcium deposits are not the only possible source of emboli during valvuloplasty. In fact, the exposed surface produced by the fracture of nodular calcifications is potentially thrombogenic since it is irregular, anfractuous, and denuded of endothelium. Furthermore, dislodgement of atheromatous debris from the frequently narrowed femoral and iliac vessels as a result of introduction of the valvuloplasty catheter is another potential cause of systemic embolization; however, this can probably be circumvented by more widespread application of the so-called long-sheath technique.

A few cases of severe aortic insufficiency have been reported after aortic valvuloplasty (two patients, 2·8%, in Cribier's reported experience[8]). Interestingly, the most likely cause of the aortic insufficiency in one of our patients was probably the asymmetrical distribution of calcific degeneration, resulting in a non-uniform dilatory effect of the balloon on the valvular cusps and consequently more damage to the more pliable, less calcified leaflet. We have previously stressed[21] the current absence of an imaging technique which allows instantaneous assessment of induced morphological changes of the individual valve leaflets. Such an imaging system would also allow a more rational selection of the proper size and shape of balloon catheter, in order to avoid excessive leaflet and/or aortic ring damage.

IMMEDIATE AND LATE EFFICACY

The available immediate and long-term results of aortic valvuloplasty suggest that only a palliative effect can be achieved with this technique. Table 3 summarizes the mean reduction in transvalvular gradients and the increase in aortic-valve area obtained as a result of aortic valvuloplasty in the larger clinical series reported so far[3–6,9,11,16,19,22]. In the present series of patients, an aortic-valve area of $\geqslant 1\ cm^2$ was achieved in 1 patient, and a mean aortic transvalvular gradient of <40 mmHg in 10 patients. However, the clinical improvement experienced by most patients indicates that even a limited haemodynamic improvement can provide a major increase in the patient's effort tolerance. Furthermore, the measured transvalvular gradients and computed valve areas post dilatation are generally above the limits conventionally regarded as an indication for surgical valve replacement therapy. In the initially reported series, enlargement of the stenotic-valve area was obtained by using circular balloons ranging from 10 to 14 mm in diameter[1]. In the two years since then, the size of the balloons used has increased progressively. Currently, several groups are employing circular balloons ranging from 19 to 25 mm[6,8,9,11], bifoil or trefoil balloons of comparable size[3,23], or double-balloon techniques[22]. The additional haemodynamic benefit of these balloons seems limited and the potential danger of an oversized balloon relative to the aortic ring should be considered, particularly when the lack of elasticity of the annulus in degenerative aortic-valve disease is borne in mind (S. King, pers. comm.).

Immediate improvement does not imply lasting clinical benefit. Reversible deformation of the aortic-valve orifice with prompt return to its original size[12], a scaring reaction around calcium particles fragmented during dilatation[16], and progression of the underlying degenerative valvular disease[24] have all been claimed as possible pathogenetic mechanisms of early and late valve restenosis. In the present series 5 out of 13 patients with a successful procedure followed for at least one year, had symptomatic late valve restenosis (38%).

Table 3 Reported haemodynamic results following aortic balloon valvuloplasty in adult patients

Author	No. patients	Mean age (years)	Transvalvular aortic gradient (mmHg)				Aortic valve area (cm^2)	
			Peak systolic		Mean systolic			
			Before PBAV	After PBAV	Before PBAV	After PBAV	Before PBAV	After PBAV
Slama *et al.*[3]	29	79	—	—	—	—	0·51	0·73
Jackson *et al.*[4]	8	74	86	53	—	—	—	—
Isner *et al.*[5]	9	78	68	35	57	30	0·42	0·81
McKay *et al.*[6]	32	79	77	39	60	36	0·60	0·90
Drobinski *et al.*[9]	34	74	81	40	—	—	0·38	0·75
Berland *et al.*[11]	502	75	78	37	—	—	0·46	0·80
Letac *et al.*[19]	170	74	72	30	—	—	0·52	0·92
Dorros *et al.*[22]	14	75	73	29	59	29	0·59	0·99
Present series	25	75	84	41	73	43	0·47	0·72

PBAV, percutaneous balloon aortic valvuloplasty.

This 'diluted' analysis of the results, does not confirm the previously reported observation[11,19] that only patients with minimal or mild initial haemodynamic improvement are at risk of recurrence of symptoms.

CLINICAL COMMENT

Aortic balloon valvuloplasty appears indicated, and provides a new therapeutic approach, in a symptomatic patient with an unacceptably high surgical risk or who refused surgical valve replacement. Retrospective studies concerning the most suitable candidate for aortic balloon dilatation, i.e. patients with severe, symptomatic aortic stenosis who did not undergo aortic valve replacement, confirmed the very poor survival (25% at three years) when on medical treatment only[25,26]. On the other hand, if candidates suitable for surgery are also considered, the risk–benefit ratio of each of these two treatment modalities must be compared. The perioperative mortality rate of an aortic-valve replacement in elderly patients with severe aortic stenosis ranges from 3[27] to 18%[28], and even higher rates are reported for patients older than 75 years of age[29], in the case of emergency surgery, or patients who are in a poor general condition[30]. However, comparison of in-hospital mortality rates following aortic-valve replacement versus percutaneous valvuloplasty may be somewhat misleading, given the strict selection criteria for surgery relative to the poor general condition of many valvuloplasty patients (cardiogenic shock, renal or respiratory insufficiency, malignancy, etc.). One of the consequences of this selection is an unpredictable immediate result of the valvuloplasty procedure, with an incidence of non-responding or poorly responding patients (aortic valve area $<0{\cdot}7\,cm^2$) ranging from 18·7[6] to 38·2%[11]. A rational selection of patients most amenable to balloon dilatation can not be performed as long as we do not have an imaging technique which allows detailed visualization of aortic-valve morphology.

Thorough knowledge of the result of valvuloplasty at long-term follow-up is mandatory, particularly of younger patients who are still engaged in active physical activites. In fact, our reported medium-term aortic-valve restenosis rate (5/25 = 20%) suggests that a cautious attitude should be taken as long as a persistent clinical benefit is yet to be confirmed. For this reason we have stressed that, at present, aortic valvuloplasty should be regarded as a palliative treatment. However, surgical valve replacement is not a curative procedure either. Mechanical valves require anticoagulation for life in order to minimize the existent risk of embolic complications. Biological valves have lower thrombogenicity, but may require a subsequent reoperation because of late valve degeneration. Nevertheless, the superior, and long-lasting haemodynamic results after aortic-valve replacement are evident when the effects of balloon valvuloplasty (Table 3) are compared with measured gradients and areas of the currently available prosthetic valves[29]. These observations[31] may explain why improvements in left ventricular ejection fraction after aortic balloon valvuloplasty[8,32] do not correspond with reported results after aortic-valve replacement[33]. To summarize, we believe that balloon valvuloplasty can be an adjunct to surgery for the following reasons:(1) percutaneous aortic valvuloplasty can be used to stabilize the haemodynamics in patients with severe heart failure or cardiogenic shock, in order to reduce the subsequent surgical mortality rate; or (2) the significant improvement obtained in patients with an extensively calcified aortic valve by a simple 'blind' procedure such as aortic valvuloplasty, may stimulate the development of alternative techniques of treatment, such as open-heart surgical valvuloplasty.

Conclusion

Percutaneous aortic valvuloplasty is an effective, palliative treatment in most inoperable patients or elderly patients at high surgical risk. At present[13,14], however, the inherent complication rate and its limited immediate and long-term efficacy preclude a more widespread application in young adults and elderly patients in good general health.

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Comment by B. Letac (Rouen, France)

This paper is another example confirming that percutaneous aortic balloon valvuloplasty in adult aortic stenosis is performed in more and more centres. When interpreting these results from Rotterdam, one has to take into consideration that this procedure is quite recent, and that continuous technical improvements are in progress. Therefore, at the time of appearance of this paper, due to the inevitable delay for publication, the results are less good than those obtained in more recently treated patients. The aortic-valve area obtained in this series was only a mean of 0·72 cm^2, whilst, at the present time, is normally around 1 cm^2.

However, all in all, the results presented by Serruys *et al.* are encouraging, in particular because this is a study with a 9-month follow-up, in which a marked functional improvement was observed in half the patients. These results are rewarding in themselves because the patients treated by this procedure were either not surgical candidates or very high-risk candidates.

It is a little surprising to see that in this series of 25 patients there were 3 cases of cerebrovascular accident which occurred during the procedure. This 12% CVA rate is very high as compared to other series, published or soon to be published. However, this may be only coincidental, related to a bad series law.