# CARDIOLOGY UPDATE

## Reviews for Physicians

1990 Edition

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# PREFACE

*Cardiology Update, 1990 Edition* continues a series dedicated to current topics of clinical importance in cardiology. These chapters cover a broad spectrum of timely topics that impact upon the important day-to-day problems that the practicing internist and cardiologist face. Appropriate to the titles of these volumes, it is our purpose to update important areas of cardiology and to ensure that the latest information in the field is presented. Among the newer developments discussed in the area of interventional cardiology are the current status of aortic balloon valvuloplasty in the adult patient, the roles of coronary artery bypass surgery and angioplasty in patients with multivessel coronary disease, and the problems facing the surgeon following unsuccessful angioplasty. In addition, one of our chapters is devoted to cardiac angioscopy and how it has helped our understanding of coronary and peripheral vascular disease. Thrombolysis is an area that has exploded on the cardiology scene in recent years, and in this volume there is a discussion of the newer thrombolytic agents. Complex ventricular arrhythmias following recovery from myocardial infarction portend decreased likelihood of survival; however, suppression of these ventricular arrhythmias with antiarrhythmic drugs has been singularly unsuccessful. The startling results of the newest of these trials, the Cardiac Arrhythmia Supression Trial (CAST), is reviewed. The intriguing issue of silent myocardial is-

chemia is also reviewed in this volume by one of the pioneers in this field. Finally, the last three chapters address three separate topics of interest; they are the diagnosis and management of syncope, calcium nutrition and metabolism as it relates to blood pressure control, and the receptor-G protein-adenylate cyclase complex in heart failure.

The Editorial Board and I are indebted to the outstanding authorities in the field who have contributed to this volume. I trust that the readers, whether clinicians or academicians, will be stimulated by the interesting, diverse subjects in this volume.

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# CONTENTS

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# CURRENT STATUS OF AORTIC BALLOON VALVULOPLASTY TREATMENT OF ADULT PATIENTS

## RESOLVED ISSUES, CONTROVERSIAL ISSUES, FUTURE DIRECTIONS

Charles R. McKay, MD

Over the last four years, considerable experience has been acquired regarding the performance and results of aortic balloon valvuloplasty. Published reports from many investigators have resolved certain issues regarding the safety, efficacy, and preferred methods of performing the technique. In addition, controversial issues regarding aortic valvuloplasty are being resolved by recent clinical studies and registry reports. With these experiences it is now possible to look to future directions in technical improvements, in clinical applications, and follow-up results of aortic balloon valvuloplasty. We are developing a clearer understanding of the clinical role and utility of the technique in patient care. This review will discuss those issues regarding aortic valvuloplasty that are resolved based on current data, then those that remain controversial; suggest future directions for clinical investigation; and discuss the possible role of aortic balloon valvuloplasty in patient care.

The practice of aortic balloon valvuloplasty has undergone an extraordinary evolution over its short 4-year history. It was first applied in adult patients by Cribier and Letac in 1985.[1] The immediate hemodynamic improvements were associated with dramatic symptomatic improvements in their elderly and very ill patients. These encouraging early results were quickly confirmed by other groups[2-4] and led to enthusiastic editorial responses.[5,6] The practice of aortic balloon valvulo-

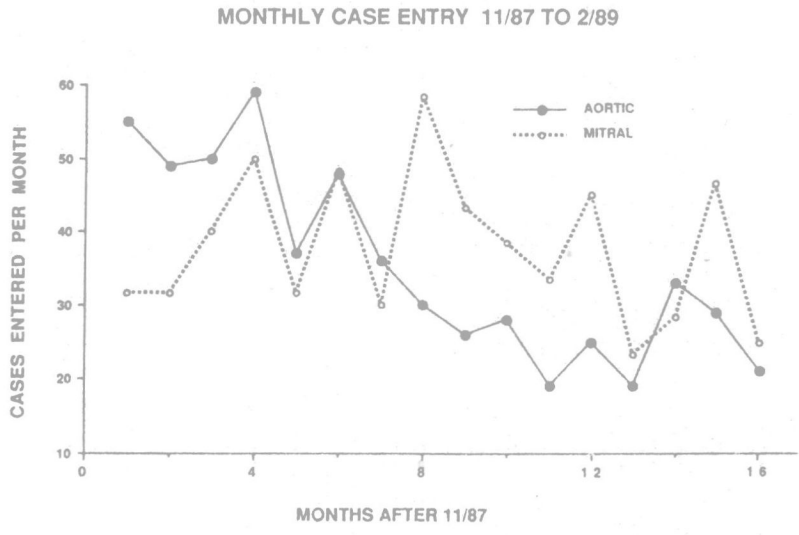


**FIGURE 1.** Monthly case entry into the NHLBI Balloon Valvuloplasty Registry. Although mitral case entry is relatively stable, the aortic case entry rate shows a steady decline over the first 16 months of the registry.

plasty quickly proliferated through many academic and community hospital centers, despite its status as an investigational technique. Recently, follow-up results[7-9] have demonstrated significant mortality and a high incidence of recurrent symptoms have led to a more restricted use of the technique in some centers. As an illustration, the case acquisition rates for the NHLBI Balloon Valvuloplasty Registry are shown in Figure 1. The addition of new mitral cases to the registry show that there is an erratic but persistent acquisition rate of new cases. In contrast, the acquisition rate of new aortic cases shows a steady decline over the initial 16-month registry period despite a potentially larger appropriate patient population. Although further studies will be needed to clarify the usefulness of this technique, certain issues regarding aortic valvuloplasty have been resolved.

## RESOLVED ISSUES

Resolved issues include the standard technique and possible variations in the technique for performing the procedure, the immediate hemodynamic results, and the associated procedure-related risks. In addition, data regarding the mechanisms of dilatation and restenosis and clinical results at midterm follow-up have now been published.

The optimum technique used to perform aortic balloon valvuloplasty has evolved from that originally reported by Cribier (Figure 2).

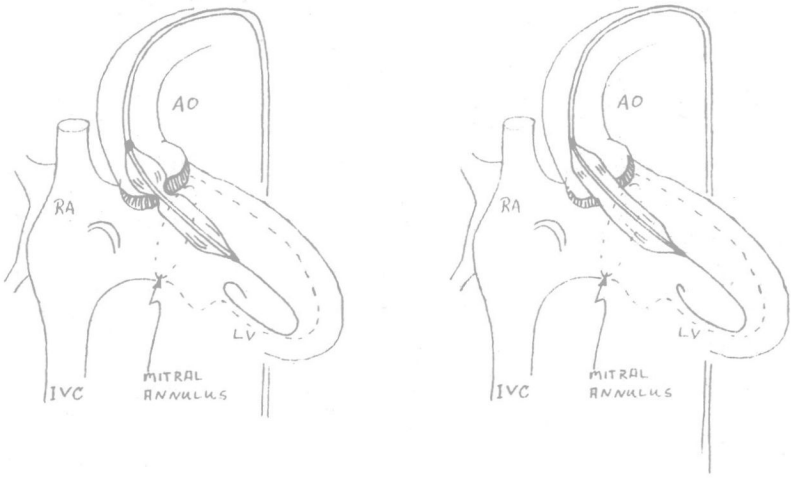


**FIGURE 2.** Retrograde technique of aortic balloon valvuloplasty. Line drawing in RAO position shows a single balloon catheter across the stenotic aortic valve. The distal catheter tip is in the left ventricle and is maintained in place over a long J-exchange guidewire.

A catheter is placed retrograde from a femoral artery over a guidewire across the stenotic aortic valve with the distal end of the balloon in the left ventricle. Careful positioning of the catheter across the aortic valve is followed by full inflation and rapid deflation of the balloon. The most commonly used balloon is 20 mm in diameter and 5.5 cm long. The inflation times vary from 10 to 60 seconds, as determined by a patient's hemodynamic response. Table 1 summarizes the immediate hemodynamic results from several groups. Analysis of these data demonstrate

**TABLE 1.** Immediate Results of Aortic Balloon Valvuloplasty

| Lead author (Ref. no.) | No. of patients | Mean age (yr) | Gradient AO-LV (mm Hg) | | AO valve area ($cm^2$) | | Comments |
|---|---|---|---|---|---|---|---|
| | | | Pre | Post | Pre | Post | |
| Cribier (10) | 92 | 75 | 75 | 30 | 0.5 | 0.9 | 15- to 20-mm balloon |
| McKay (11) | 32 | 79 | 77 | 39 | 0.6 | 0.9 | |
| Block (12) | 25 | 79 | 63 | 35 | 0.4 | 0.7 | Retrograde cases |
| Schneider (13) | 14 | 77 | 55 | 37 | 0.6 | 0.8 | |
| Isner (14) | 16 | 74 | 56 | 42 | 0.5 | 0.6 | Single-balloon cases |
| McKay (15) | 22 | 67 | 56 | 33 | 0.6 | 0.9 | Single-balloon cases |
| Brady (16) | 26 | 86 | 59 | 31 | 0.5 | 0.7 | All patients >80 yr |

*Abbreviations:* AO-LV = mean transaortic valve gradient aorta to left ventricle; AO valve area = aortic valve area by the Gorlin formula.

ABLE 2. Immediate Major Risks of Aortic Balloon Valvuloplasty

| Lead author (Ref. no.) | No. of patients | Death | CVA or emboli | Perforation and tamponade | Vascular complications | | Other |
|---|---|---|---|---|---|---|---|
| | | | | | Total | To OR | |
| Cribier (10) | 92 | 3 | 0 | — | 3 | 1 | 2 AR increased |
| McKay (11) | 32 | 3 | 0 | 1 | — | — | EF ↑ 0.40 to 0.46 |
| Block (12) | 25 | 1 | 3 | 0 | 6 | 0 | One transfusion |
| Schneider (13) | 14 | 0 | 0 | 0 | 2 | 0 | Two transfusions |
| Isner (14) | 16 | 1 | 0 | 1 | 2 | 1 | — |
| McKay (15) | 22 | 1 | 0 | 0 | 3 | 2 | Two transfusions |
| Brady (16) | 26 | 3 | 1 | 0 | 3 | 2 | Four AV block, one AR |

*Abbreviations:* Death = in-hospital mortality; CVA = cerebrovascular accident; perforation = left ventricular guidewire or catheter perforation; to OR = number requiring surgical correction; AR = new increased aortic regurgitation; EF = left ventricular ejection fraction; AV = aortic valve.

that aortic valvuloplasty results in an immediate 25% to 60% decrease in the transaortic valve gradient and average increases in aortic valve areas of 0.1 to 0.4 $cm^2$. Some reports also demonstrated decreases in left ventricular diastolic pressure, with minimal changes in the cardiac output and improvement in symptomatic clinical status in most patients. In these reports the increase in aortic valve area is usually 0.2 to 0.4 $cm^2$ and only occasionally (15% to 37% of cases) is the final area increased to greater than 1 $cm^2$. The major risks of balloon valvuloplasty reported by these groups (Table 2) include a 3% to 12% in-hospital mortality rate, cardiac perforation with tamponade, systemic embolization, and damage to the aortic cusps or annulus. In addition, there was approximately a 10% incidence of vascular trauma, about half requiring operative correction. Although early in the use of valvuloplasty, transfusion was common, recently it is rare to transfuse these patients. There were also small associated risks of arrhythmias, aortic valve block, and bacteremia.

These reports are consistent in demonstrating the ability of aortic balloon valvuloplasty to acutely decrease the aortic valve gradient and improve the aortic valve function. Midterm follow-up of larger groups of patients (Table 3) has demonstrated a 3% to 9% 30-day mortality, an incidence of recurrent symptoms from 13% to 57%, hemodynamic deterioration even without increased symptoms in over half of the patients, and a 6- to 12-month mortality rate of 5% to 42%. In some reports these adverse events are collectively viewed as *clinical restenosis*, because few of these elderly patients actually have repeat catheterization. More important, however, are the consistent findings in subsets of patients of increased aortic valve gradients in both asymptomatic and symptomatic patients, reevaluated by doppler or catheterization.[7,8,18,22] To put these results in perspective, it should be noted that young symptomatic patients with critical aortic stenosis face an 8% annual mortality[25] and el-

derly patients face a 45% to 57% 12-month mortality without surgery.[26,27] With surgery they may face up to 29% early mortality and 20% late mortality.[28] Recent reports have nevertheless advanced more pessimistic views of the potential of aortic balloon valvuloplasty to achieve persistent improvement in aortic valve function in unselected patients and have concentrated on identifying clinical predictors of restenosis. As might be expected, recurrent symptoms and poor survival are more commonly seen in patients with associated systemic diseases, poor left ventricular function,[24] diffuse coronary artery disease, and aortic valve areas after dilatation of only 0.7 $cm^2$ or less.[23]

Complementary careful morphologic studies of stenotic aortic valves at autopsy and at the time of aortic valve replacement offer explanations for these immediate and midterm follow-up results (Table 4). Early studies[2,4] emphasized splitting and fracturing of calcific nodules and stretching of valve cusps as mechanisms for improving aortic valve function. In these studies, the false commissures of bicuspid or unicuspid valves were not split, but the fused commissures in the few rheumatic valves studied were split and resulted in improved aortic cusp mobility. These results were confirmed by Waller et al,[29] who studied the mechanism of aortic valve stenosis in autopsy specimens using videotaped records of the valves and valvuloplasty balloon dilatation procedures. With this technique, the stresses and stretching mechanisms could be directly observed as the balloon inflated across the valve and could be reanalyzed on videotape in light of the resultant morphologic changes produced by the valvuloplasty procedure. Obvious fractures of large calcific nodules, small fractures in calcified cusps, and splitting of fused commissures were observed (Figure 3). *In many of the valves, fusion of these commissures was not a prominent morphologic mechanism causing aortic valve stenosis.*

Review of videotaped records during balloon expansion showed that as the balloon expanded, the calcific nodules were displaced, and directly stretched and distorted the aortic cusps and aortic wall. These studies showed that a primary mechanism of improved valve mobility in the nonrheumatic valves was stretching of the aortic valve cusps and annulus.[29] Furthermore, observing these specimens immediately after valvuloplasty demonstrated that the elastic tissues soon returned to their original shape and size unless there was dehiscence and damage to the aortic wall tissues. Therefore, we have postulated that a significant amount of *restenosis* after aortic valvuloplasty occurs shortly after the procedure and is due to elastic recoil of the aortic wall.

Further study of over 50 such specimens has confirmed these findings and has shown that the etiology of the stenosis is a major determinant of the mechanism by which valvuloplasty relieves stenosis and the mechanism of restenosis.[30] In rheumatic valves where commissural fusion is a prominent mechanism of aortic orifice restriction, splitting of the commissures results in improved cusp mobility. However, where commissural fusion is not present, namely, in senile calcific aortic stenosis and in middle-aged and elderly patients with calcified bicuspid aortic valves, fracture of nodules and expansion of the aortic wall are

**TABLE 3.** Follow-Up After Aortic Balloon Valvuloplasty

| Lead author (Ref. no.) | Total no. of patients* | F/U (mo) | AVG (mm Hg) | | | AVA ($cm^2$) | | | 30-day mortality (% of total) | No. patients with recurrent symptoms (%) | No. patients with AVR | Repeat BV | No. patients dead at end of F/U | Comments |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Pre | Post | F/U | Pre | Post | F/U | | | | | | |
| Cribier (17) | 204 | 12 | 71 | 21 | — | 0.5 | 1.0 | — | 19(9) | 61(30) | — | — | 40(20) | 24% restenosis at cath. |
| Desnoyers (7) | 42 | 10 | 70 | 37 | 38 | 0.5 | 0.8 | 0.5 | 3(7) | 16(38) | 3 | 1 | 4(10) | AVG in 52% of patients on echo |
| Nishimura (8) | 55 | 6 | 48 | 33 | 46 | 0.5 | 0.9 | 0.7 | 3(5) | | 9 | 4 | 8(5) | 78% of patients symptoms pre BV symptoms at 30 d |
| Brady (16) | 26 | 6 | 59 | 31 | — | 0.5 | 0.7 | — | 2(8) | 2(8) | 1 | | 5(19) | All patients >80 yr; cost of BV (11 d and $30K) on 8/26 with complications (16 d and $44K) |
| Bashore (18) | 44 | 6 | — | — | — | 0.7 | 1.0 | 0.7 | — | 25(57) | — | — | — | Systematic recath.; symptoms underestimate restenosis |

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