BALLOON MITRAL VALVULOPLASTY AT GROOTE SCHUUR HOSPITAL

RESULTS, COMPLICATIONS AND SHORT-TERM FOLLOW-UP

JOHN BERNARD LAWRENSON

Research dissertation presented for the approval of Senate in fulfilment of part of the requirements for the degree of Master of Medicine.

Cape Town 1994

To Natalie and Michael

DECLARATION

I, John Bernard Lawrenson, hereby declare that the work on which this dissertation is based is my original work (except where acknowledgements indicate otherwise) and that neither the whole work nor any part of it has been, is being, or is to be submitted for another degree in this or any other University.

I empower the University to reproduce for the purpose of research either the whole or any portion of the contents in any manner whatsoever.

Signed by candidate

Signature removed

JOHN LAWRENSON

20 December 1994

ACKNOWLEDGEMENTS

I would like to thank my supervisor, Prof Patrick Commerford for guiding the dissertation to completion.

I also wish to thank Drs Basil Levetan and Sean Latouf for helping to construct a computerised database of patients undergoing mitral valvuloplasty at Groote Schuur Hospital.

In addition I wish to acknowledge the role of Professor Commerford and Drs Adrian Horak and Belinda Green in performing the valvuloplasty procedures. These procedures could not have been performed without the assistance of the nursing and radiography staff of the C25 Cardiac Catheterisation Laboratory at Groote Schuur Hospital.

SUMMARY

Balloon dilatation of the stenosed mitral valve, in an attempt to relieve symptoms, was developed to replace the surgical procedure of closed mitral valvotomy. This procedure, whereby a balloon tipped catheter is introduced from the femoral vein and directed across the mitral valve after an atrial septal puncture, was developed in 1982. The procedure was first performed at Groote Schuur Hospital in 1988. Two types of dilating balloon (Inoue and Bifoil types) have been used.

The aim of this retrospective study was to analyze the results of balloon mitral valvuloplasty procedures performed from 1988 until November 1992. In addition a detailed analysis was made of all complications of the procedure.

118 patients (mean age 30.7 years) underwent 124 attempted procedures. 93% of attempts were successfully completed and an optimal result was achieved in 76% of patients. Mitral valve area increased from 0.9cm^2 to 2.0 cm^2 . Equivalent results have been achieved with both balloon types.

Death occurred in 1.6% of patients. 2.4% of patients had severe mitral regurgitation as a complication. 4% of procedures resulted in cardiac chamber perforation.

The experience at Groote Schuur has been similar to other centres treating young patients with rheumatic mitral stenosis.

TABLE OF CONTENTS

CHAPTER 8

CHAPTER 9

CHAPTER 1

INTRODUCTION

A significant aspect of recent cardiological research has been the development of techniques which can eliminate the need for a thoracotomy and general anaesthesia (as well as cardio - pulmonary bypass) and achieve comparable results to standard surgical procedures. Despite the absence of a controlled study comparing medical and surgical therapy, the surgical treatment of patients with significant mitral stenosis is felt to be superior to medical therapy and to offer patients relief from symptoms at least if not a survival advantage. Balloon dilatation of the stenosed mitral valve has been developed to replace surgery. Short-term results suggest that balloon valvotomy is a good alternative to surgical valvotomy. It has not yet been established whether this will be true in the long-term.

Balloon mitral valvotomy has been performed at Groote Schuur hospital since 1988. Prior to presenting the results of balloon valvotomy at Groote Schuur Hospital, I will attempt to review the published literature relating to the technique in order to place the local results in perspective.

MITRAL STENOSIS - ETIOLOGY AND NATURAL HISTORY

Mitral stenosis in the adult is usually the result of rheumatic fever and the mitral valve is most commonly involved by the rheumatic process.[1]

Only 50% of patients with mitral stenosis will have a history of rheumatic fever despite having typical pathological features consistent with a rheumatic process when pathological material is examined.[2] In the classical review of the subject of mitral stenosis Paul Wood found that progression to a stage of symptoms equivalent to Class IV of the New York Heart Association (NYHA) grading takes 7 to 10 years from onset of symptoms.[3] The average age of onset of Wood's patients was 12 years of age. Symptoms developed at an average age of 31 years. In a series of 1000 patients started by Duckett Jones in 1928 the average age of onset of rheumatic fever was 8 years.[4] (This average is in a sense artificial in that patients older than 20 years were left out of the series).[4]

In the series of Bland and Jones of the 117 patients who had clinical mitral stenosis at 20 years of follow-up, only 40 (34%) had evidence of mitral stenosis at 10 years of follow-up. The time taken for the development of symptomatic mitral stenosis following an attack of rheumatic fever in other areas of the world, particularly the so called 'Third World', is felt to be shorter.[5]

The progression of stenosis may relate to repeated attacks of rheumatic fever, or an ongoing inflammatory reaction,[6] or to the effect of turbulence causing repetitive minor trauma to the narrowed mitral orifice with progression of stenosis being as a result of fibrosis.[7] There does not appear to be hard evidence for the last claim other than by comparison to the natural history of bicuspid aortic stenosis. The progression of disease clearly varies in patients with varying levels of symptomatology. This is illustrated in Rowe's series and in a paper from Selzer's group.[8,9] 40% of patients with clinical mitral stenosis but no symptoms on presentation survived 20 years. Half of these patients remained asymptomatic.[8] This slow

progression of mitral stenosis in patients who have minor symptoms can be seen as a counter to those who favour early dilatation of the clinically stenosed mitral valve by whatever means. The variable progression to the development of symptomatic mitral stenosis is reflected in the marked differences in the mean age of populations currently undergoing balloon valvotomy in different parts of the world. Chen et al reported a series from mainland China with a mean age of 33.8 +/- 7.6 years.[10] A typical United States based study has a mean age of 53 +/- 15 years.[11]

In a review in Circulation in 1972 Selzer and Cohn summarized '(t)he pathologic evolution of mitral stenosis'.[8] They described the progression of rheumatic involvement of the valve from the nodules consisting of fibroblasts and macrophages which are found along the opposing edges of the valve and which characterise acute valvulitis to the thickening and fibrosis of the valve which results in alteration of the underlying structure of the valve. These structures then are also subject to calcification. The valve cusps become fused together as do the subvalve structures. Rusted, Scheifley and Edwards, in a pathological study, listed three main types of mitral stenosis depending on the parts of the valve structure involved.[12] They described a commissural type in which the fusion of commissures predominates, a cuspal type in which the leaflets are stiffened and inflexible and a chordal type. In the latter type the chordae become fused. They define these as pure forms and feel that combinations of the pure forms occur.

Sellors and co-workers developed a grading system of stenosis based on pathological findings at operation.[13] The Sellors grading is reflected in the contemporary echocardiographic scoring system. In their system Sellors et al defined three types of valvar abnormality: (a) the mobile

cusps without subvalve changes - diaphragmatic type; (b) thickened cusps with mild to moderate subvalvar abnormality; and (c) rigid cusps with severe subvalvar change - the funnel type. They recognised that the patients with mobile valves had the best results at surgery.

The clinical findings noted in patients with mitral stenosis are well documented[1] and I will not deal with them specifically. The patient with NYHA class IV symptoms usually has a valve area of 1 cm^2 or less. (The normal mitral valve area is between 4 and 6cm^2).[1] At this stage the pressure in the left atrium is approximately 25mm Hg while the left ventricular end diastolic pressure is normal.[14] The cardiac index at this time may have fallen to 2.5 $l.min^{-1}.m^{-2}$ from 3 $l.min^{-1}.m^{-2}$.[14] The increased left atrial pressure is associated with an increase in pulmonary vein pressure and an increase in pulmonary capillary pressure. Pulmonary oedema is said to occur when plasma oncotic pressure is exceeded by pulmonary capillary pressure (25mm Hg).[14] Reactive changes in the pulmonary vascular bed are thought to develop at a left atrial pressure beyond 25mm Hg. Reactive changes in pulmonary pressure do not occur in all patients. Theoretical explanations consider pulmonary vasoconstriction to be responsible for this change. No author has been able to explain why pulmonary hypertension is present only in a percentage of patients with severe mitral stenosis.[15] The elevation in pulmonary pressures and consequent elevation of right ventricular pressures are responsible for the ultimate development of signs consistent with right sided failure. Patients with mitral stenosis and pulmonary hypertension are likely to be NYHA class IV limited.[15]

The pressure gradient between left atrium and left ventricle (the transmitral gradient) increases with any increase in flow across the valve - therefore an increase in heart rate will increase the flow across the mitral valve and left atrial pressure will increase. (Transvalvular

gradient is a function of the square of the transvalvular flow rate).[16] The symptoms of patients with mitral stenosis are thus worsened if they develop a tachycardia or if they develop atrial fibrillation with a rapid ventricular response. Furthermore loss of atrial contraction associated with the development of atrial fibrillation *may* decrease cardiac output by 20%.[17] (The rapid ventricular rate associated with uncontrolled atrial fibrillation probably has a far more important role in the production of symptoms).

There is no documented natural history of untreated mitral stenosis. Kirklin refers to the documented series of patients from the 1950's and 1960's as 'the spectrum of mitral stenosis in *surgically untreated* patients receiving the medical treatment that was available in the mid-portion of the twentieth century'.[18] The survival of such patients is seen to relate to the clinical stage at which they were first seen and to the presence or absence of atrial fibrillation.

Rowe and others documented a 84% survival over 10 years in patients without symptoms when first seen.[8] A large series of patients documented by Olesen[19] showed that patients with NYHA Class IV equivalent symptoms as a result of mitral stenosis had a 0% 10 year survival rate (15% 5 year survival), compared to a 33% 10 year survival rate (62% at 5 years) in patients in NYHA functional class III. (Rowe's group with NYHA class III symptoms had a 15% 10 year survival).[8] The group as a whole had a mean survival of between 6 and 7 years from first consultation. Olesen's large natural history series although widely quoted suffers from its vintage in that 14% of the patients were felt to have significant mitral regurgitation. (It even includes 12 patients who underwent mitral valvotomy - who were felt not to materially alter the series). In addition no patients were asymptomatic. Atrial

fibrillation was associated with a 0% survival at 20 years versus 29% in patients in sinus rhythm.[19] Modern statistical techniques may have shown that the apparently worse outcome in patients with atrial fibrillation may not be true. Rowe's series showed little difference in outcome between patients with atrial fibrillation and those without.[8] Rowe's group as a whole had a higher percentage of survivors at 10 years (61% versus 35%) because of the inclusion of the asymptomatic patients. At 20 years 79% of Rowe's patients were dead as opposed to 83% of Olesen's.

Patients with medically treated stenosis eventually develop irreversible pulmonary hypertension and right heart failure with cardiac cachexia being the final stage of the disease. Atrial fibrillation, secondary to raised atrial pressure and subsequent atrial dilatation is initially paroxysmal and then becomes permanent in 40 to 50% of patients.[20] Atrial fibrillation and associated atrial enlargement form the 'breeding ground' for potential emboli. These systemic emboli are often fatal.[15]

THE SURGICAL TREATMENT OF MITRAL STENOSIS

Just as medical therapy has probably altered the natural history of patients with mitral stenosis from that of patients in the earlier part of the century where death closely followed the onset of symptoms, so surgical therapy has altered the natural history to allow the development of significant aortic and tricuspid valve disease.[18]

As a result of refinements developed during the late 1940's the technique of splitting the fused mitral commissures of a beating heart entered the armamentarium of the surgeon. In 1923

E.C. Cutler and S.A. Levine had reported on mitral commissurotomy performed from the ventricular apex. H.S. Souttar reported on the opening of a stenotic mitral valve from the left atrium in 1925. Apart from Souttar's single patient, the initial results were poor.[18]

Baker, Brock and Campbell reported 6 survivors out of 8 in 1950. Their technique involved splitting the fused commissures with a specially developed knife or by simply using a finger.[21]

After these initial reports mitral valve surgery was attempted in many centres. The initial experience of mitral valvotomy at Groote Schuur Hospital was reported in 1955 when Schrire and co-workers detailed their experience with 75 patients.[22]

The Tubb's dilator was introduced in the late 1950's and allowed better control of splitting of the commissures with a dilator introduced from the apex of the ventricle.[23] The development of cardiopulmonary bypass techniques in the early 1960's allowed the surgeon to split the commissures under direct vision. In addition, if the valve was deemed to be unsuitable for simple commissurotomy it could be replaced with a mechanical prosthesis.[18] (Mechanical prostheses began to be developed at this time). The chances of a clot being dislodged from the atrium and resulting in an embolus are also decreased.

Apart fom embolisation the major complication of the technique is the production of significant mitral regurgitation which may lead to the need for valve replacement. This resulted in open techniques becoming more popular.[18]

The published series of closed mitral valvotomy are substantially larger than the series of

open valvotomy patients. Three series involving survival data on more than five hundred patients have been documented. The largest series is that of John et al from India.[24] They documented the course of 3724 patients with a mean age of approximately 27 years. The early post operative mortality in this series was 3.8%. The authors were able to obtain follow-up data on 70% of the patients. (This was an outstanding effort considering that their patients came from all over the Indian subcontinent!) There was an actuarial survival of approximately 85% at 24 years after operation. 78.3% had survived without the need for reoperation. The incidence of post operative mitral regurgitation was low - 0.3% had severe regurgitation. Interestingly, in this series the patients in NYHA Class IV had a 4.2% mortality which is relatively low.

In a series of patients from South Africa, Commerford and co-authors reported their experience in 680 patients.[25] Actuarial survival was 78% at 12 years post operation. 47% had not required reoperation. Operative mortality was just below 3%. Like the series from India the patients were younger than a comparable group from the USA (mean age 33 years). In this series operative mortality for patients in NYHA Class II was 0% while it was approximately 12% for those patients in Class IV.

The major study of closed mitral valvotomy from the United States is that of Ellis et al who reported prolonged follow-up in 1000 patients.[26] This study documents an earlier time period than John's and Commerford's since the patients began to be collected from 1949 - shortly after the introduction of the technique. Operative mortality in this series of patients was 3.3% in their group III patients and 24% in their group IV patients. (Groupings are approximately equivalent to New York Heart Association class). This series states its incidence of

reoperation and mortality in terms of the 15 year follow-up for all patients. The incidence of reoperation for patients in group III is 33%. Mortality data are difficult to compare since the 15 year mortality figures do not include those patients who had another operation.

A smaller but more recently documented study from the Mayo clinic details the long term survival of patients who underwent closed mitral valvotomy in the early 1960's. 57% of patients were alive without a second operation at 10 years post surgery. (79% of the total group of 207 patients were alive at 10 years and 55% at 20 years post surgery). At 10 years post operation 90% were free of any embolic complication.[27] In this retrospective report the authors divide the patients into two groups on the basis of left atrial thrombus and valve calcification. (These are two features which they feel currently tend to preclude the performance of balloon valvotomy). Survival in the group without either characteristic was significantly better at 10 years post operation (84% versus 70.3%).

Feigenbaum and others documented the haemodynamic changes after closed commissurotomy in 35 patients prior to a closed procedure and repeated the investigations 6 to 36 months post procedure.[28] Valve area increased from 1.3 $\pm$ 0.3 to 2.6 $\pm$ 1.0 cm^2. (Interestingly, the empiric constant used in calculation of valve area by the Gorlin formula was 31 instead of 37.7 used currently[29] and this would mean that valve areas were slightly overestimated). Left atrial pressure declined from 20 $\pm$ 8 to 12 $\pm$ 4mm Hg. Pulmonary artery pressure dropped from 31 $\pm$ 11 to 21 $\pm$ 7mm Hg.

A common theme to many reports is the attempt to identify factors present prior to surgery and at operation which may be used to predict survival without the need for repeat surgery

as well as overall long-term mortality. More recent series have used current methods of multivariate analysis to define such factors. As mentioned above preoperative NYHA class predicts outcome. Some series report demographic factors such as older age, female gender and Black race as being predictive of a worse outcome.[30,31] In other series such as that of Commerford these factors do not predict an adverse outcome. (This is presumably because of fundamental differences in the make-up of the population undergoing surgery).[25]

Commerford et al reported that the 'suitability' of the valve as determined by the surgeon at operation predicted the results of the procedure. For example, patients with 'good' valves had a survival of just over 50% versus a cumulative survival of slightly more than 20% in patients with 'bad' valves. Clinical assessment of mobility could also be used to predict a successful result. In the series of Ellis et al valvar calcification (in a sense partially equivalent to a 'bad' valve) at operation contributed to a poorer outcome with time.[26]

The presence of pulmonary hypertension preoperatively is possibly a predictor of a poor outcome. Hickey et al (see below) concluded that a raised pulmonary vascular resistance preoperatively was associated with a decreased long-term survival.[30] In a series from Denmark, Ravkilde and Hansen demonstrated that a mean capillary wedge pressure greater than 25mm Hg rather than pulmonary pressure was an independent risk factor for poor survival extending out to 34 years.[31] Hickey determined that mitral regurgitation immediately after the procedure was also a risk factor for a poor long-term outlook.[30] Repeat valvotomy, while associated with a slightly higher operative mortality is not associated with a poorer outcome.[25]

It has been claimed that open mitral valvotomy is better than the closed technique. Apart from the 'visibility' and ability to avoid embolus propagation the technique is said to provide better results in terms of valve area.

The favourable light in which many surgeons view open valvotomy is probably as a result of its use during a period when there were few imaging modalities available to predict valvar morphology prior to surgery. For the reader of published series differences in statistical techniques used to assess survival and differences in selection of patients render easy comparison difficult. Furthermore, careful reading of papers is often required to determine how many patients underwent mitral valvotomy *alone* - in other words, without other procedures such as coronary bypass grafting).

Aaron and Lower summarized their perceived advantages of the open technique in an article in 1974.[32] According to these authors the technique offered the following:

1. The valve could be viewed in a dry field.
2. Clot in the left atrium can be seen and removed reducing embolisation.
3. Easy assessment of valve competence can be made and the valve can be replaced if necessary.
4. Clinical entities masquerading as mitral stenosis such as myxoma can be dealt with readily. *(Irrelevant in the 'echo' era.)*
5. Other valve lesions can be dealt with at the same sitting.
6. Trainee surgeons can easily be trained to perfect techniques in an easily viewed field.

The operative mortality (older authors use the hospital mortality while more recent authors use 30 day mortality) for open valvotomy ranges from 0 to 10.8 percent.[30,33,34,35,36,37,38,39]

In these series reporting results in numbers of patients ranging from 95 to 347 patients, it is difficult to determine how the operation was selected. In one series of patients with what appears to have been simple mitral stenosis (reported by Laschinger et al) more than 50% underwent valve replacement.[39] Long-term survival analysis using actuarial techniques has been performed and while most authors report good long-term results in terms of mortality data on freedom from reoperation is not always stated. Nakano and co-authors report a 14 year actuarial survival rate of 94.6 $\pm$ 0.02% with 83.8 $\pm$ 0.05% freedom from operation at 14 years.[38] Laschinger reported an actuarial survival of 100% at 9 years post operation.[39] Gross reported a survival of 92% at 10 years.[37] In Housman's series the actuarial survival at 10 years was 97 $\pm$ 2%. However, the chance of not requiring a reoperation was 38 $\pm$ 16% at 10 years.[36] The reasons for the differences in survival without reoperation between these two groups are not easily established. Housman's group dates from 1960 to 1975 and Nakano's dates from 1972 to 1985. Housman's group contains more patients in class III (NYHA).

In a refreshing paper (because of its lucid descriptions of patient selection, operative technique and subsequent events) Cohn and co-authors described their experience with open mitral valvotomy.[40] In this series of 120 patients with a mean age of 49 years, the operative mortality was 0% and the actuarial probability of survival at 10 years was 95 $\pm$ 2%. Freedom from reoperation at 10 years was 84 $\pm$ 5 %.

A number of series have documented the haemodynamic changes after open valvotomy. In Nakano's series in patients with pliable valves (Sellors type 1) the valve area increased from $0.72 \pm 0.08cm^2$ to $2.52 \pm 0.19cm^2$.[38] In a series of younger patients from Iran, open mitral valvotomy was noted to result in a decrease in pulmonary wedge pressure from 28.8 ± 6.3 to 13.8 ± 5.2mm Hg, whereas closed mitral valvotomy resulted in a decrease from 28.6 ± 6.2 to 17.8 ± 7.1mm Hg. The numbers of patients are probably too small to offer an overall conclusion as to the superiority of one technique over the other.[5]

Analyses of open valvotomy series to obtain predictive outcome related variables have been reported. (These series are similar to the series discussed above concerning the closed procedure).

In a series from New Zealand, Smith et al reported that Maori race, preoperative symptomatology, preoperative mitral regurgitation, atrial fibrillation and pulmonary vascular resistance predicted a worse outcome. Valvular calcification and subvalvar deformity were operative predictors of a poorer outcome.[41]

Hickey et al state that the superiority of open versus closed techniques represents 'conventional wisdom'. They state that this superiority has not been tested in a randomised controlled trial. By applying multivariate techniques to their own results (retrospectively) they concluded that after adjustments were made for preoperative risk factors 'all outcomes were the same after open and closed commissurotomy.'[30] They reviewed all procedures performed between 1967 and 1988 at the University of Alabama. Median follow-up of the patients was 11.4 years. Overall 10 year actuarial survival was 87%. After adjustment for risk factors

there was felt to be no difference between the techniques.

In addition, Hickey et al criticise comparisons of closed mitral valvotomy to open mitral valvotomy for not having taken into account that at least some of the closed procedures were not performed with the aid of a Tubb's dilator.[30]

It is worthwhile noting that reoperation post valvotomy is not always as a result of restenosis - failure of the first operation to improve the valve area and mitral regurgitation induced by the first operation are further reasons for repeat surgery.[15,42] Higgs noted in a series from 1970 (post closed valvotomy) that only 11% of patients with recurrent symptoms actually had restenosed. Early catheterisation had been performed in a series of 163 patients - 45 subsequently returned with symptoms and were restudied. 36% of these patients had *persistent* mitral stenosis with little change in valve area between their valve area at the time of symptoms and that immediately post commissurotomy.[42] In the large series of closed cases from India restenosis occurred at between 4.2 per 1000 patients at 5 years of follow-up and 11.4 per 1000 at 15 years of follow-up.[24] (The method of diagnosis of restenosis is not stated).

Dalen feels that no series of catheterisations post open mitral valvotomy has been performed and that the true incidence of restenosis has not been determined.[15]

Comparisons between open and closed valvotomy do not only involve considerations of mortality and reoperation but also consider differences in intraoperative and post operative emboli. The incidence of embolisation both preoperatively and post operatively depends on

the length of observation of the patient.[41] Furthermore, the occurrence of intraoperative emboli and the effects of these events are often ignored.[41] It is difficult to compare the incidence of intraoperative and post operative embolisation (perioperative) in patients undergoing closed or open operations. In Ellis and Harken's series the incidence was 6%.[26] In Housman's series (of open valvotomy) the incidence of early emboli was 2%.[36] In the series from India of closed valvotomy the overall incidence of emboli in the early post operative period was approximately 1.2%.[24] (This is calculated from the figures expressed in the paper which divide the patients according to sinus rhythm and atrial fibrillation).

Long-term incidence of embolisation is also documented - in Laschinger's series (post open valvotomy) the yearly incidence of post operative embolisation was 0.66%.[39] John et al reported an incidence of embolism of 0.4% after closed valvotomy.[24] The interpretation of these data and their application to modern practice is difficult given that the practice of anticoagulation of patients in atrial fibrillation prior to surgery and after surgery varied. It is hard to imagine that post operative anticoagulation as practised in the United States was feasible on the Indian subcontinent.

In Hickey's series the freedom from first post valvotomy thrombotic event was 90% at 10 years and 82% at 20 years. This is a combined value for both types of procedure. Previous embolism (pre-valvotomy) is a risk for embolism post operatively with freedom from a postoperative embolus in these patients of 61% at 10 years and 38% at 20 years.[30] Cohn's series showed a 91 $\pm$ 3% freedom from emboli at 10 years post operation. Annual rates of thromboembolism expressed as percentage per patient year vary from 0.7 to 1.8% in patients who have had an open mitral valvotomy.[40]

Because of the difficulties in comparing reported data and validating claims of improvement in incidence of embolisation after surgery it is felt that the ability of an operation alone to influence the occurrence of post operative emboli is doubtful.[43] Smith et al reviewed the problem of embolisation and came to a similar conclusion.[41] Partial protection from embolism also seems doubtful.[41]

The greatest weakness of all comparisons of closed and open valvotomy is that series are seldom presented with open valvotomy results on an 'intention to treat' basis. It is therefore difficult to distinguish which mitral valve replacements occurred because of a failed open valvotomy and which replacements occurred when the surgeon declared that the native valve was beyond repair on first inspection.

Valve replacement has not assumed the position of primary treatment for uncomplicated mitral stenosis because even 30 years after development valve replacement remains an imperfect technique with a significant operative mortality. Despite relieving the pathology it creates a 'neodisease' with the potential for embolisation, thrombosis and anticoagulant related bleeding.[44]

The risk of warfarin-related mortality is said to be 0.2 per 100 patient years (2.2 for morbidity) and the risk of a fatal thrombosis is 0.2 per 100 patient years (1 to 2 for morbidity).[1]

Bioprosthetic valves, which do not require the use of anticoagulants do not offer any benefit especially in a younger population as they are prone to degeneration.[44]

No randomised prospective series comparing elective mitral valve replacement to commissurotomy has been reported. In Laschinger's group of patients with mitral stenosis reported in 1982 who probably had 'too many' mitral valve replacements, the operative mortality was 11.2%.[39] Patients underwent Starr-Edwards prosthetic valve implantation. The group undergoing open mitral valvotomy had a 0% mortality. Actuarial survival at 9 years post valve replacement was 83.9 $\pm$ 5.5% (excluding operative deaths) versus 100% in patients after commissurotomy.

The average operative mortality for valve replacement in the mitral position is said to be between 5 and 10%.[45] A recent series has documented an operative mortality of 7.5% in a group of male patients undergoing mitral valve replacement with either a Bjork-Shiley mechanical valve or a Hancock tissue valve.[46] In this group operative mortality was 6.6% in those patients not needing additional bypass grafting. Interestingly, the mortality in patients less than 60 years of age was 3%. Although the exact valvular pathology was not stated in the paper, it is safe to assume that the mitral valve lesions included severe calcification and significant regurgitation. No patient with ischaemic mitral regurgitation was included.

The 11 year actuarial probability of survival with a prosthetic heart valve in the same group of patients was 58%.[47] During the 1970's the operative mortality for valve replacement in patients with mitral stenosis at Groote Schuur Hospital was 7% with a three year survival of approximately 84%.[48]

Simple comparisons of survival data are difficult since the groups undergoing valve replacement represent a far more complex group as regards valve pathology, abnormal

ventricular function, coronary artery disease and associated illness than the patient with uncomplicated mitral stenosis.

A report from Eguaras et al in 1990 compared two groups of (non-randomised) patients with mitral stenosis who either underwent valve replacement or open mitral commissurotomy depending on the degree of valve calcification.[49] Although the groups of patients were relatively similar in terms of demographic description, there were slightly more patients with NYHA Class IV symptoms in the replacement group. The patient groups were relatively small - 60 patients with open repairs and 75 with valve replacements. The operative mortalities were not significantly different (4% in the replacement group and 0% in the repair group) and the actuarial survival of the groups to 10 years did not differ significantly. Intriguingly, there was no difference in the probability of freedom from thromboembolism. The major difference related to freedom from reoperation. The patients undergoing valve replacement had a significantly higher actuarial probability of needing repeat surgery than the patients undergoing valve repair (69 $\pm$ 8.74% vs 84 $\pm$ 15.4%). In the valve replacement group, tissue valve degeneration contributed to the higher reoperation rate.

REFERENCES FOR CHAPTER 1

1. Braunwald E. Valvular heart disease. In: Braunwald E, ed. Heart disease. A textbook of cardiovascular medicine. 4th ed. Philadelphia: WB Saunders, 1992:1007-1077.

2. Gorlin R. Natural history, medical therapy and indications for surgery in mitral valve disease. In: Ionescu MI, Cohn LH, eds. Mitral valve disease: diagnosis and treatment. London: Butterworths, 1985:105-123.

3. Wood P. An appreciation of mitral stenosis. Part I. Clinical features. Br Med J 1954;1:1051-1063.

4 Bland EF, Jones TD. Rheumatic fever and rheumatic heart disease: A twenty year report on 1000 patients followed since childhood. Circulation 1951;4:836-843.

5. Aryanpur I, Shakibi J, Yazdanyar A, Mehranpur M, Paydar M, Azar H, Motlagh FA, Tarbiat S, Siassi B. Closed versus open mitral commissurotomy in children with rheumatic mitral stenosis. J Thorac Cardiovasc Surg 1978;19:654-658.

6. Chopra P, Tandon H D, Raizada V, Gopinath N, Butler C, Williams RC. Comparative studies of mitral valves in rheumatic heart disease. Arch Intern Med 1983;143:661-666.

7. Selzer A, Cohn K. Natural history of mitral stenosis: a review. Circulation 1972;45:878-890.

8. Rowe JC, Bland EF, Sprague HB, White PD. The course of mitral stenosis without surgery: Ten- and twenty-year perspectives. Ann Intern Med 1960;52:741-749.

9. Dubin A, March HW, Cohn K, Selzer A. Longitudinal hemodynamic and clinical study of mitral stenosis. Circulation 1971;44:381-389.

10. Chen CR, Hu SW, Chen JY, Zhou YL, Mei J, Cheng TO. Percutaneous mitral valvuloplasty with a single rubber-nylon balloon (Inoue balloon): Long-term results in 71 patients. Am Heart J 1990;120:561-568.

11. Herrmann HC, Kleaveland JP, Hill JA, Cowley MJ, Margolis JR, Nocero MA, Zalewski A, Pepine CJ. The M-Heart percutaneous balloon mitral valvuloplasty registry: initial results and early follow-up. J Am Coll Cardiol 1990;15:1221-1226.

12. Rusted IE, Scheifley CH, Edwards JE. Studies of the mitral valve: II. Certain anatomic features of the mitral valve and associated structures in mitral stenosis. Circulation 1956;14:398-406.

13. Sellors TH, Bedford DE, Somerville W. Valvotomy in the treatment of mitral stenosis. Br Med J 1953;2:1059-1067.

14. Grossman W. Profiles in valvular heart disease. In: Grossman W, Baim DS, eds. Cardiac catheterisation, angiography and intervention. 4th ed. Philadelphia: Lea and Febiger, 1991:557-581.

15. Dalen JE. Mitral stenosis. In: Dalen JE, Alpert JS, eds. Valvular heart disease. 2nd ed. Boston: Little and Brown, 1987:49-110.

16. Braunwald E, Turi Z. Pathophysiology of mitral valve disease. In: Ionescu MI, Cohn LH, eds. Mitral valve disease: diagnosis and treatment. London: Butterworths, 1985:3-10.

17. Thompson ME, Shaver JA, Leon DF. Effect of tachycardia on atrial transport in mitral stenosis. Am Heart J 1977;94:297-306.

18. Kirklin JW, Barratt-Boyes BG. Mitral valve disease with or without tricuspid valve disease. In: Cardiac Surgery. New York: Churchill Livingstone, 1986:323-372.

19. Olesen KH. The natural history of 271 patients with mitral stenosis under medical treatment. Br Heart J 1962;24:349-357.

20. Rackley CE, Edwards JE, Karp RB. Mitral valve disease. In: Hurst JW, Schlant RC, Sonnenblick EH, Kass Wenger N, eds. The Heart. 7th ed. New York: McGraw-Hill, 1990:820-851.

21. Baker C, Brock RC, Campbell M. Valvotomy for mitral stenosis. Report of six successful cases. Br Med J 1950;1:1285-1293.

22. Schrire V, Vogelpoel L, Phillips W, Nellen M. Experience with mitral valvotomy at Groote Schuur Hospital, Cape Town. S Afr Med J 1955;29:1108-1114.

23. Austen WG, Wooler GH. Surgical treatment of mitral stenosis by the transventricular approach with a mechanical dilator. New Engl J Med 1960;263:661-665.

24. John S, Bashi VV, Muralidharan S, Ravikumar E, Rajarajeswari T, Krishnaswami S, Sukumar IP, Sundar Rao PSS. Closed mitral valvotomy: early results and long-term follow-up of 3724 consecutive patients. Circulation 1983;68:891-896.

25. Commerford PJ, Hastie TW, Beck W. Closed mitral valvotomy: actuarial analysis of results in 654 patients over 12 years and analysis of preoperative predictors of long-term survival. Ann Thorac Surg 1982;33:473-479.

26. Ellis LB, Singh JB, Morales DD, Harken DE. Fifteen- to twenty-year study of one thousand patients undergoing closed mitral valvotomy. Circulation 1973;48:357-364.

27. Rihal CS, Schaff HV, Frye RL, Bailey KR, Hammes LN, Holmes DR. Long-term follow-up of patients undergoing closed transventricular mitral commissurotomy: a useful surrogate for percutaneous balloon mitral valvuloplasty? J Am Coll Cardiol 1992;20:781-786.

28. Feigenbaum H, Linback RE, Nasser WK. Hemodynamic studies before and after instrumental mitral commissurotomy. A reappraisal of the pathophysiology of mitral stenosis and the efficacy of mitral valvotomy. Circulation 1968;38:261-276.

29. Carabello BA, Grossman W. Calculation of stenotic valve orifice area. In: Grossman W, Baim DS, eds. Cardiac catheterisation, angiography and intervention. 4th ed. Philadelphia: Lea and Febiger, 1991:152-165.

30. Hickey MS, Blackstone EH, Kirklin JW, Dean LS. Outcome possibilities and life history after surgical mitral commissurotomy: Implications for balloon commissurotomy. J Am Coll Cardiol 1991;17:29-42.

31. Ravkilde JL, Hansen PS. Late results following closed mitral valvotomy in isolated mitral valve stenosis: Analysis of thirty-five years of follow-up in 240 patients using Cox regression. Thorac Cardiovasc Surg 1991;39:133-139.

32. Aaron BL, Lower RR. Advantages of open mitral commissurotomy using a triple-orifice technique. Ann Thorac Surg 1974;19:654-658.

33. Roe BB, Edmunds H, Fishman NH, Hutchinson JC. Open mitral valvotomy. Ann Thorac Surg 1971;12:483-491.

34. Halseth WL, Elliot DP, Walker EL, Smith EA. Open mitral commissurotomy. A modern re-evaluation. J Thorac Cardiovasc Surg 1980;80:842-848.

35. Finnegan JO, Gray DC, MacVaugh H, Joyner C, Johnson J. The open approach to mitral commissurotomy. J Thorac Cardiovasc Surg 1974;67:75-82.

36. Housman LB, Bonchek L, Lambert L, Grunkemeier G, Starr A. Prognosis of patients after open mitral commissurotomy. Actuarial analysis of late results in 100 patients. J Thorac Cardiovasc Surg 1977;73:742-745.

37. Gross RI, Cunningham JN, Snively SL, Catinella FP, Nathan IM, Adams PX, Spencer FC. Long-term results of open radical mitral commissurotomy: Ten year follow-up study of 202 patients. Am J Cardiol 1981;47:821-825.

38. Nakano S, Kawashima Y, Hirose H, Matsuda H, Shirakura R, Sato S, Taniguchi K, Kawamoto T, Sakaki S, Ohyama C. Reconsiderations of indications for open mitral commissurotomy based on pathologic features of the stenosed mitral valve. A fourteen-year follow-up study in 347 consecutive patients. J Thorac Cardiovasc Surg 1987;94:336-342.

39. Laschinger JC, Cunningham JN, Baumann FG, Isom OW, Catinella FP, Mendelsohn A, Adams PX, Spencer FC. Early open radical commissurotomy: surgical treatment of choice for mitral stenosis. Ann Thorac Surg 1982;34:287-298.

40. Cohn LH, Allred EN, Cohn LA, Disesa VJ, Shemin RJ, Collins JJ. Long-term results of open mitral valve reconstruction for mitral stenosis. Am J Cardiol 1985;55:731-734.

41. Smith WM, Neutze JM, Barratt-Boyes BG, Lowe JB. Open mitral valvotomy. Effect of preoperative factors on result. J Thorac Cardiovasc Surg 1981;82:738-751.

42. Higgs LM, Glancy DL, O'Brien KP, Epstein SE, Morrow AG. Mitral restenosis: an uncommon cause of recurrent symptoms following mitral commissurotomy. Am J Cardiol 1970;26:34-37.

43. De Vivie ER, Helberg K. Closed transventricular mitral commissurotomy. In: Ionescu MI, Cohn LH (eds). Mitral valve disease. Diagnosis and treatment. London: Butterworths, 1985:139-151.

44. Grunkemeier GL, Rahimtoola SH. Artificial heart valves. Ann Rev Med 1990;41:251-263.

45. Gray RJ, Helfant RH. Timing of surgery for valvular heart disease. Cardiovasc Clinics 1993;23:209-231.

46. Sethi GK, Miller DC, Souchek J, Oprian C, Henderson WG, ul Hassan Z, Folland E, Khuri S, Scott SM, Burchfiel C, Hammermeister KE. Clinical, hemodynamic and angiographic predictors of operative mortality in patients undergoing single valve replacement. J Thorac Cardiovasc Surg 1987;93:884-897.

47. Hammermeister KE, Sethi GK, Henderson WG, Oprian C, Kim T, Rhahimtoola S - for the Veterans affairs cooperative study on valvular heart disease. A comparison of outcomes in men 11 years after heart-valve replacement with a mechanical valve or bioprosthesis. N Engl J Med 1993;328:1289-1296.

48. Dalby AJ, Firth BG, Forman R. Preoperative factors affecting the outcome of isolated mitral valve replacement: a 10 year review. Am J Cardiol 1981;47:826-834.

49. Eguaras MG, Luque I, Montero A, García MA, Calleja F, Román M, Concha M. A comparison of repair and replacement for mitral stenosis with partially calcified valve. J Thorac Cardiovasc Surg 1990;100:161-166.

CHAPTER 2

THE HISTORY OF BALLOON VALVULOPLASTY

The conversion of techniques employed by cardiothoracic surgeons to their less invasive equivalents (which could be performed in the catheterisation laboratory) commenced during the 1960's. Cheng has described William Rashkind's development of balloon atrial septostomy as the birth of therapeutic balloon dilatation.[1] He considers the development of balloon angioplasty by Andreas Greuntzig to be another milestone in the history of balloon valvuloplasty since the technology relating to the development of flexible angioplasty catheters was applied when devices suitable to dilate stenotic heart valves were developed.

Mitral stenosis (in a 33 year old man) was first treated with transvenous balloon dilatation on the 3rd of June 1982 by Kanji Inoue in Japan.[2] The device used was developed by Inoue himself and was used in 6 patients. (The device was introduced via a saphenous vein cutdown until changes in design facilitated the percutaneous introduction of the device). Ironically this balloon catheter had been developed for experimental atrial septostomy in dogs in 1980 as a potentially more effective device than the Rashkind balloon since it had the ability to create a larger orifice. This would have the theoretical advantage of avoiding the need for repeat septostomy or surgical septectomy in patients with transposition of the great arteries.[3] The device has been used in humans for this purpose as well.[1]

Lock et al reported the use of an alternative balloon (originally designed for peripheral vessel angioplasty) for valvuloplasty in 1985.[4] This device was used in a group of 8 young patients

ranging from 9 to 23 years of age who came from India and the Azores.

Al Zaibag and co-workers described balloon dilatation with two transvenous catheters in 1986.[5] The reason for using two balloons appears to have been the concern that the larger diameter single balloon had a greater deflated profile and needed a larger hole in the atrial septum. Two smaller balloon catheters could be passed via two transeptal punctures. The resultant combined diameter of the two holes in the atrial septum would be less than the orifice needed for the single balloon.[5] In addition they were concerned that a single balloon *totally* occluded the mitral orifice. Furthermore, it became evident that the dilatation of the valve with the single balloons (then commercially available) was inferior to the double balloon technique.[6] The first groups of patients who had their mitral stenosis treated were generally younger people with non-calcified rheumatic mitral stenosis.

Palacios[7] and McKay[8] in separate reports in 1986 described balloon dilation of calcified mitral stenosis with good immediate effect - McKay's patient was 75 years old. (The patients were unable or reluctant to undergo surgery).

In an attempt to avoid the perceived dangers of balloon septostomy of the atrial septum, Babic and co-workers developed a retrograde approach whereby the dilating catheter was drawn back across the mitral orifice via the femoral artery - this technique required both femoral arteries and both femoral veins to be punctured as well as a transseptal puncture. The long guide wire (which stretched from the right femoral vein across the atrial septum, through the mitral valve and out of the left femoral artery via the aortic valve) was said to stabilize the dilating balloon and allow for inflation of the balloon away from the submitral apparatus.[9]

This technique has been further modified to allow retrograde placement of the balloon without a transeptal puncture.[10]

Other balloon devices originally developed for pulmonary valve dilatation were also utilised in mitral valvotomy - the 'bifoil' and 'trefoil' devices offered the potential advantage of two or three smaller balloons with a large total area which could be introduced on a single catheter.[11]

A combination of the trefoil balloon with the 'conventional' balloon (such as those used by Lock and Palacios) to provide better stability has been described.[12]

At present the Inoue and double-balloon techniques appear to be the most commonly used techniques for balloon mitral valvuloplasty and series utilising these techniques account for the bulk of the published literature.

REFERENCES FOR CHAPTER 2

1. Cheng TO. A history of percutaneous balloon valvuloplasty. In: Cheng TO, ed. Percutaneous balloon valvuloplasty. New York: Igaku-Shoin, 1992:1-11.

2. Inoue K, Owaki T, Nakamura T, Kitamura F, Miyamoto N. Clinical application of transvenous mitral commissurotomy by a new balloon catheter. J Thorac Cardiovasc Surg 1984;87:394-402.

3. Inoue K, Kitamura F, Chikusa H, and Miyamoto N. Atrial septostomy by a new balloon catheter. Jpn Circ J 1981;45:730-738.

4. Lock JE, Khalilullah M, Shrivastava S, Bahl V, Keane JF. Percutaneous catheter commissurotomy in rheumatic mitral stenosis. N Engl J Med 1985;313:1515-1518.

5. Al Zaibag M, Ribero PA, Al Kasab S, Al Fagih MR. Percutaneous double-balloon mitral valvotomy for rheumatic mitral stenosis. Lancet 1986;1:757-761.

6. Abdullah M, Halim M, Rajendran V, Sawyer W and Al Zaibag M. Comparison between single (Inoue) and double balloon mitral valvuloplasty: Immediate and short-term results. Am Heart J 1992;123:1581-1588.

7. Palacios IF, Lock JE, Keane JF, Block PC. Percutaneous transvenous balloon valvotomy in a patient with severe calcific mitral stenosis. J Am Coll Cardiol 1986;7:1416-1419.

8. McKay RG, Lock JE, Keane JF, Safian RD, Aroesty JM, Grossman W. Percutaneous mitral valvuloplasty in an adult patient with calcific rheumatic mitral stenosis. J Am Coll Cardiol 1986;7:1410-1415.

9. Babic UU, Pejcic P, Djurisic Z, Vucinic M, Grujicic S M. Percutaneous transarterial balloon valvuloplasty for mitral valve stenosis. Am J Cardiol 1986;57:1101-1104.

10. Orme E, Wray RB, Mason JW. Balloon mitral valvuloplasty via retrograde left atrial catheterisation. Am Heart J 1989;117:680-683.

11. Patel J, Vythilingum S, Mitha AS. Balloon dilatation of the mitral valve by a single bifoil (2 X 19 mm) or trefoil (3 X 15 mm) catheter. Br Heart J 1990;64:342-346.

12. Vahanian A, Michel PL, Cormier B, Ghanem G, Vitoux B, Maroni JP, Cazaux P, Acar J. Immediate and mid-term results of percutaneous mitral commissurotomy. Eur Heart J 1991;12:Suppl B:84-89.

CHAPTER 3

METHODS OF BALLOON MITRAL VALVULOPLASTY

At least four terms - valvotomy, valvulotomy, commissurotomy and valvuloplasty - are used to describe the treatment of mitral stenosis with a balloon catheter. The relationship of the new technique to the surgical method it replaces has resulted in the suggestion that valvulotomy or commissurotomy are the more appropriate terms.[1,2] The Greek for the verb to mould or form gives rise to the suffix '-plasty' whereas the Greek for the verb to cut gives rise to the suffix '-tomy'.[2] Valvulotomy and valvotomy are essentially synonymous since 'valvular' and valvar are equivalent terms, the former being a North American term and the latter being a British term.[3]

Cheng argues that the term valvuloplasty is the appropriate term since mere cutting of the fused commissures is not all that is responsible for the eventual increase in valve area. Furthermore, he argues that valvuloplasty is a better generic term since the 'curing' of other stenotic valves is not always related to the splitting of commissures. (He refers in particular to the tearing of leaflets in pulmonary stenosis).[3]

I will use the term 'valvuloplasty' because it appears to be the most frequently used term in the published literature.

As alluded to in the previous chapter, the demonstration that the stenosed mitral valve could be treated with balloon techniques lead to attempts being made in many centres to achieve

similar results. The techniques used related largely to the availability of commercially produced balloon devices.

At Groote Schuur hospital two techniques have been used. The Bifoil and Trefoil balloons manufactured by Schneider and utilised for pulmonary valve dilatation were used before the Inoue balloon became commercially available.

The performance of balloon valvuloplasty in the laboratory at Groote Schuur is not preceeded by administration of prophylactic antibiotics. (Palacios and Block recommend the use of a short course of dicloxacillin started before the procedure).[4] Because balloon valvuloplasty is increasingly being performed in patients who are only NYHA Class II disabled, it is the laboratory's practice to omit diuretics for two days prior to the procedure since resting transmitral gradients in a patient with significant mitral stenosis may be negligible when the patient is well diuresed. In such patients the assessment of the haemodynamic effects of the balloon dilatation may otherwise be very difficult.

The procedure is performed in the fasting state. A single dose of an anxiolytic drug (lorazepam or other equivalent drug dependent on the practice of that institution) is administered on the morning of the procedure. Apart from liberal use of local anaesthetic, additional analgesia is provided with titrated doses of intravenous morphine. Balloon valvotomy is labour intensive usually requiring two operators as well as two to three members of the ancillary staff.

While a surgical team is not kept on standby during the performance of mitral balloon

valvuloplasty, the procedure is scheduled at a time when such facilities could be accessed with minimal delay should a severe complication ensue. It is suggested that balloon valvuloplasty should not be performed at institutions where a surgical facility is not available.[5]

THE BIFOIL AND TREFOIL TECHNIQUE

Suitable patients undergo the procedure following an echocardiographic study and a left and right sided cardiac diagnostic catheterisation. In older patients, coronary angiography is also advised. An aortic root angiogram may be of use in those patients with significant additional aortic valve disease as well as mitral stenosis. (Should the patients require emergency surgery, additional information concerning aortic regurgitation and coronary artery disease will be of use to the surgeon).

Initially these haemodynamic studies were performed prior to the performance of balloon valvuloplasty - however, with increasing experience, the haemodynamic study and the balloon dilatation are performed at the same 'sitting'. Turi advises that the former practice be continued to allow the haemodynamic and renal effects of any contrast agent to abate.[1] He does comment that because of the accuracy of echocardiography in assessing gradients the haemodynamic information obtained during valvuloplasty 'in young patients without other confounding issues' is sufficient.[1]

Left ventricular angiography will indicate the degree of mitral regurgitation present and allows the operator to judge the position of the mitral valve plane.

The initial therapeutic step, which the technique shares with the Inoue technique, consists of the performance of a transeptal puncture with a standard Brockenborough needle. An 8F Mullins sheath is advanced into the left atrium. A paediatric balloon catheter is then used to dilate the puncture site in the atrial septum. A large bore Swan-Ganz balloon tipped catheter is advanced into the left atrium and subsequently into the ventricle.

A Cook 0.038 inch guidewire is then advanced via the channel of the balloon catheter and allowed to lie with its tip in the left ventricular apex.

The Bifoil catheter consists of two 19mm (when inflated) diameter balloons on a single shaft and the Trefoil catheter consists of three 15mm balloons on a single shaft.

A large bore sheath (16.5F) is passed into the right femoral vein and the balloon catheter passed through this device along the guidewire, crossing the atrial septum. The balloon device is positioned across the mitral orifice and inflated with diluted radiographic contrast. The final diameter of the inflated balloons was related (prior to the procedure) to the diameter of the mitral annulus on two dimensional echocardiography.

A number of inflations are employed until the 'waisting' of the balloon (as seen on fluoroscopic screening when the inflated balloon device was positioned within the stenotic valve) disappears. The device is then removed and haemodynamic measurements repeated to allow calculation of the final mitral valve area.[6]

The double balloon technique is similar to the above technique, except that two guidewires

are passed into the left ventricle - in earlier procedures this necessitated two septal punctures. Specially designed catheters facilitate the passage of two guidewires through a single puncture. Two dilating balloons are passed sequentially along the guidewires and positioned across the mitral valve. Various combinations of balloon size (typically two 20mm diameter balloons) are used, according to patient size.[7] In patients with severe stenosis, a single balloon may be used to perform the initial dilatation. Other centres have used a combination of a more rigid catheter such as the Bifoil with a Mansfield type balloon in order to stabilize the dilating apparatus.[8]

THE INOUE TECHNIQUE

Concern over the length of the shaft of the Schneider catheter beyond the end of the dilating balloons (and the potential of this stem to tear the ventricle during balloon inflation) led to the replacement of the Schneider technique with the Inoue Balloon catheter technique when this became commercially available in South Africa.

The Inoue balloon differs from all other balloons utilised in interventional cardiology in that the rubber balloon is designed such that the tip of the balloon inflates first and then is followed by the inflation of the rest of the balloon when increasing volumes of dilute contrast are added to the balloon. This allows the balloon to be placed by 'flow-direction' - in other words, the partially inflated balloon can be floated across the stenosed mitral valve without a guidewire having to be placed into the ventricle - decreasing the risk of ventricular perforation.

The Inoue balloon is, in a sense, a balloon within a balloon. Two layers of latex rubber tubing are separated by a layer of nylon micromesh which reinforces the structure. The balloon is able to be inflated in three stages as a result of rubber bands wound tightly around the mesh in the centre of the balloon and more loosely around the proximal mesh. The balloon can thus be inflated in the following sequence: tip first, then proximal section and then the complete balloon.[9] The balloon is mounted on a 12F polyvinyl chloride catheter with two coaxial lumina.[5]

The procedure starts with a transeptal puncture with a conventional Brockenborough needle. The site of puncture varies from the traditional site (the fossa ovalis) used when a transeptal puncture is performed during diagnostic catheterisation. To compensate for the increase in the size of the left atrium and bulging of the fossa ovalis the puncture in the septum is lower than the conventional technique.

In order to puncture the atrium in the correct place, Inoue and co-workers have suggested puncturing the atrial septum about one vertebral body above the inferior border of the left atrium. (The atrium can usually be visualised with fluoroscopy, but Inoue et al suggest that the levophase of a right atrial contrast injection may provide a useful map of the left atrium).[5]

The atrial puncture is usually made with the Brockenborough direction indicator at 6 o'clock rather than the traditional 4 o'clock position. In addition the pigtail catheter above the aortic valve and the Swan-Ganz catheter in the main pulmonary artery (which are not removed after the haemodynamic study from the left femoral vessels) are used as landmarks to prevent accidental aortic puncture. It has been suggested that transoesophageal echocardiographic

techniques may be of use during the transeptal puncture since the atria, atrial septum and needle can be identified without difficulty.[10] The positioning of the balloon may also be facilitated as may the detection of mitral regurgitation.

With successful left atrial puncture, the Mullins or Brockenborough catheter is advanced into the left atrium and the puncturing needle withdrawn. The simultaneous recording of left atrial and left ventricular pressures is then performed to confirm the transmitral gradient. (Cardiac output is usually measured by thermodilution and the Fick principle at the time of the haemodynamic study).

Following successful placement of the catheter, heparin is administered intravenously. Generally a 'rule of thumb' bolus is used (10 000 units for larger patients and 7 500 for smaller individuals). (Inoue suggests that a dose of 150u/kg is used in a patient who is not maintained on Warfarin).

A special 0.025 inch stainless steel guidewire which has a coiled floppy tip is passed into the left atrium. The floppy tip should be seen to coil within the atrium - confirming that the guidewire has not entered a pulmonary vein. The Mullins or equivalent catheter is then removed.

The Inoue technique dispenses with the need for a special dilating balloon for atrial septostomy dilatation by using a rigid 14F polythene dilator. This dilator is used to dilate both the subcutaneous tissues and the right femoral vein as well as the interatrial septum. The dilatation of the femoral vein allows the balloon to be passed transvenously without the need

for a venous sheath. The dilator is then passed over the stainless steel guidewire and advanced to dilate the interatrial septum.

The maximum diameter for the balloon catheter is assessed prior to the procedure by using the empirically derived relationship developed by Hung et al.[11] Balloon reference size is obtained by measuring patient height (in centimetres) and rounding off to the nearest 10, dividing by 10, and adding 10 to that result. Balloon catheters are available in a range of sizes (according to the maximum diameter of the fully inflated balloon).

The central lumen of the catheter allows passage of a thin 18g metal tube which fits snugly into the device. The metal hub is then pushed forward and locked into the hub of the catheter. This stretches the distal end of the balloon increasing its length from 45mm to 60mm. Inoue describes the elongated balloon as 'slenderized'.[5] The thin distal balloon can then be advanced into the femoral vein and across the atrial septum.

The elongated balloon is advanced over the guidewire and brought to lie with the distal end in the left atrium and a 0.038 inch high torque J-tipped spring wire stylet is introduced into the catheter. This catheter allows the tip of the balloon to be steered accurately while in the atrium. The guidewire is fairly stiff resulting in an exactly equivalent movement of the catheter with each movement of the guidewire at the catheter hub.

The metal tube is withdrawn incrementally allowing the balloon catheter to follow the curve of the coiled atrial guidewire with the balloon coming to lie entirely within the left atrium. The balloon is finally advanced along the guidewire to lie in the vicinity of the mitral orifice

before the coiled guidewire is removed. The *gradual* withdrawal of the metal tube, which fully elongates the balloon, is important since the aggressive introduction of the entire elongated balloon into the left atrium may result in the bending of the coiled guidewire by the balloon tip against the roof of the left atrium. This bending of the guidewire may make its removal impossible.[5]

The spring wire stylet is now used to aim the tip of the balloon at the mitral orifice. The tip of the balloon is then inflated with a few millilitres of contrast. A combination of counterclockwise motion of the guidewire and gentle retraction of the whole catheter usually results in the partially inflated balloon starting to enter the mitral orifice. (Occasionally, the mitral valve may be so severely stenosed that the distal balloon tip cannot be inflated at all). A final combination of movements executed simultaneously will result in the balloon passing into the left ventricle. These movements are the withdrawal of the stylet some 5cm (while maintaining full counterclockwise rotation) and the advancing of the balloon some 5cm. This combination of movements (described by one operator at the Groote Schuur laboratory as 'twisting the neck of a chicken') is usually successful. If the shape of the atrium is such that the tip of the balloon does not point along the axis of the mitral orifice when the manoeuvre is started but is perpendicular to it, another manoeuvre known as the loop method is used. The stylet is withdrawn slightly from the tip and the entire catheter is rotated 360° clockwise resulting in the end of the catheter looping in the atrium and directing the tip of the balloon into the mitral orifice.

Variations of the technique are utilised to facilitate balloon insertion when the transseptal puncture is made too high or too low or when the atrium is very large.[5]

With the balloon tip within the left ventricle, the operator is now in a position to assess whether the balloon is clear of the subvalvar structures or not. Usually free movement of the balloon within the ventricle is easily demonstrated. The inflated tip is then pulled back to lodge in the mitral orifice and a second operator will then continue the inflation of the balloon. If the balloon is in the correct position a waist will be noted at the centre of the inflating balloon. If commissural splitting is achieved, this waist is seen to 'pop' with full inflation of the balloon.

In a manner analogous to the way in which closed mitral valvotomy surgeons suggested that the Tubbs dilator should be opened to its full diameter in increments, Inoue has suggested that a series of balloon inflations should be performed starting at a smaller final diameter. This is said to lower the incidence of balloon induced mitral regurgitation.[5,12]

The balloon is withdrawn into the left atrium and the central channel of the catheter (from which the stylet is now removed) facilitates repeated haemodynamic measurements.

In the Groote Schuur catheterisation laboratory another observer (usually a catheterisation laboratory fellow) will listen for the presence of mitral regurgitation with a stethoscope following each inflation of the balloon. It has become the practice to stop the dilatation procedure if mitral regurgitation is detected by auscultation (even if an adequate reduction in gradient has not been felt to have been achieved).

The removal of the balloon across the atrial septum is facilitated by reinsertion of the coiled guidewire to lie within the atrium and repeat 'slenderizing' of the balloon. The entire

elongated balloon is then removed from the femoral vein following the performance of a left ventricular angiogram and the administration of protamine to reverse the effects of the heparin administered following the transseptal puncture.

Following haemostasis in the femoral vessels the patient is returned to the ward. Initially these patients were observed in the cardiac intensive care unit for 24 hours. However, with increasing operator experience, these patients are felt not to need intensive post-procedure monitoring unless a significant complication has occurred during the procedure or the procedure has been unusually difficult and prolonged.

REFERENCES FOR CHAPTER 3

1. Turi ZG. Valvuloplasty. Cardiovasc Clin 1993;23:293-326.

2. Smucker M.L. Percutaneous mitral balloon valvulotomy or balloon valvuloplasty? It's not just semantics anymore. Circulation 1990;82:643-645.

3. Cheng TO. A history of percutaneous balloon valvuloplasty. In: Cheng TO, ed. Percutaneous balloon valvuloplasty. New York: Igaku-Shoin, 1992:1-11.

4. Palacios IF, Block PC. Acquired mitral stenosis: double balloon catheter technique. In: Cheng TO, ed. Percutaneous balloon valvuloplasty. New York: Igaku-Shoin 1992:221-236.

5. Inoue K, Hung JS, Chen CR, Cheng TO. Mitral Stenosis: Inoue balloon catheter technique. In: Cheng TO, ed. Percutaneous balloon valvuloplasty. New York: Igaku-Shoin, 1992:237-279.

6. Patel J, Vythilingum S, Mitha AS. Balloon dilatation of the mitral valve by a single bifoil (2 x 19mm) or trefoil (3 x 15mm) catheter. Br Heart J 1990;64:342-346.

7. Palacios IF, Block PC, Wilkins GT, Weyman AE. Follow-up of patients undergoing percutaneous mitral balloon valvotomy. Analysis of factors determining restenosis. Circulation 1989;79:573-579.

8. Vahanian A, Michel PL, Cormier B, Ghanem G, Vitoux B, Maroni JP, Cazaux P, Acar J. Immediate and mid-term results of percutaneous mitral commissurotomy. Eur Heart J 1991;12:Suppl B:84-89.

9. Inoue K, Owaki T, Nakamura T, Kitamura F, Miyamoto N. Clinical application of transvenous mitral commissurotomy by a new balloon catheter. J Thorac Cardiovasc Surg 1984;87:394-402.

10. Vilacosta I, Iturralde E, San Román JA, Gómez-Recio M, Romero C, Jiménez J, Martínez-Elbal L. Transoesophageal echocardiographic monitoring of percutaneous mitral balloon valvulotomy. Am J Cardiol 1992;70:1040-1044.

11. Hung JS, Chern MS, Wu JJ, Fu M, Yeh KH, Wu YC, Cherng WJ, Chua S, Lee CB. Short and long-term results of catheter balloon percutaneous transvenous mitral commissurotomy. Am J Cardiol 1991;67:854-862.

12. Austen WG, Wooler GH. Surgical treatment of mitral stenosis by the transventricular approach with a mechanical dilator. N Engl J Med 1960;263:661-665.

CHAPTER 4

THE SELECTION OF PATIENTS FOR VALVULOPLASTY

Since the selection criteria for surgical therapy in patients with mitral stenosis were already well established,[1,2] it is not surprising that the original patients selected for balloon valvuloplasty should have been chosen according to the same criteria. Inoue's original patients were felt to have tight mitral stenosis with changes on echocardiogram equivalent to the better grades of the pathological grading system of Sellors et al.[3] (In the grading of Sellors et al, grade 1 is equivalent to the 'mobile' valve).[4]

Al Zaibag's patients were also carefully selected to include only young adults with pliable valves and no evidence of atrial clot, subvalve disease or mitral calcification.[5] In addition patients with mitral regurgitation were excluded. As mentioned in an earlier section, the initial North American experience was obtained in patients who were unable to have surgery.[6]

With increasing experience, balloon dilating procedures have been used in patients with more complex valvular pathology or in patients who were considered high risk for valve replacement because of additional cardiac or extracardiac pathology.[7,8]

The patient with clinically determined tight pliable mitral stenosis, who has no more than trivial mitral regurgitation and does not have marked valvular calcification on X-ray, should therefore be considered for balloon valvuloplasty. Significant mitral regurgitation is a contra-indication to balloon valvuloplasty. This practice is derived from the surgical results in

patients with mitral stenosis and regurgitation who underwent open mitral valvotomy and were shown to fare less well than patients with pure mitral stenosis. (Such patients are generally excluded from such surgery).[9] Balloon valvuloplasty has the potential to increase the degree of regurgitation.[10] Patients with additional moderate aortic regurgitation are not excluded from mitral valvotomy.[11]

Block has listed contra-indications to balloon valvuloplasty.[12] The contra-indications apply chiefly to those patients in whom transseptal puncture would be difficult or potentially hazardous. The patient with a left atrial thrombus, marked dilatation of the aortic root, giant right atrium, rotational anomaly of the heart or great vessels, or thoraco-lumbar scoliosis would therefore not be suitable. He added recent thromboembolic event, 2+ or more mitral regurgitation, left ventricular thrombus and associated surgical coronary or other valve disease to this list.

It would also seem logical to add the conventional contra-indication to diagnostic angiography to the list, since the dilatation procedure involves puncturing of vessels and the use of contrast agents. Most of these contra-indications are relative - correct assessment of risk and potential benefit and careful planning of the procedure in the patient with a history of allergy to contrast, renal failure or associated illness may allow the procedure to proceed without event.[13]

Initially patients with a history of embolic events or documented atrial thrombi were excluded from valvuloplasty. Hung and co-workers reported resolution of left atrial thrombus in two patients (examined with transthoracic echocardiography) treated with warfarin who

subsequently underwent balloon valvuloplasty.[14] Further work from the same group (using transoesophageal techniques) led to them suggesting a policy of 4 to 6 weeks of anticoagulation with warfarin prior to valvuloplasty.[15] Hung feels that this policy is safe even in patients in whom the atrial thrombus has not been shown to resolve since it has been demonstrated that *when the Inoue technique is used* thrombus within the left atrial appendage can be avoided.[16]

Thrombus in the vicinity of the intra-atrial septum or the mitral valve is still felt to be an absolute contra-indication to balloon valvuloplasty.[16] The symptomatic patient with a left atrial thrombus who cannot wait for definitive therapy should also undergo surgical valvotomy.

Transoesophageal echocardiography is far more sensitive in detection of atrial clot and it has been suggested that patients undergoing balloon valvuloplasty should all be screened with this technique.[17] Turi points out, however, that many procedures have been performed without preprocedure transoesophageal echocardiography and there has been a very low incidence of embolism. The detection of left atrial clot is still felt by some authors to be an absolute contra-indication to balloon valvuloplasty.[18]

Performance of valvuloplasty may be complicated by an increased risk of bleeding as a result of anticoagulation in patients with atrial fibrillation. At Groote Schuur hospital the patient's International Normalised Ratio is allowed to fall below 2 (by omitting warfarin) before the technique is performed. Other authors (such as Hung) continue standard anticoagulation and simply administer less heparin at the time of procedure.[16]

As alluded to in an earlier section, initial procedures were confined to a younger age group with 'straightforward' mitral stenosis. Age, per se, is not a contra-indication to balloon valvuloplasty. The older patient is, however, more likely to have significant valve calcification and a higher rate of unsuccessful procedures and complications related to the procedure.[19]

Previous surgical commissurotomy (either open or closed) is not a contra-indication to balloon valvuloplasty and most major series (independent of technique) contain a number of patients who have undergone surgical commissurotomy.[20] Results of the procedure in this group are equivalent to those in patients having valvuloplasty as an initial event.

Disease of other valves is not necessarily a contra-indication to balloon valvuloplasty of the mitral valve. The dilatation of a stenosed aortic valve at the procedure has been reported.[21]

THE ROLE OF ECHOCARDIOGRAPHY IN PATIENT SELECTION

It is a moot point whether echocardiographic assessment has any value other than excluding atrial clot and establishing baseline pathology in the patients with tight, pliable mitral stenosis who form the majority of patients undergoing valvuloplasty in the developing world. Good clinical results were achieved with closed valvotomy without the benefit of echocardiography. The proponents of complex echocardiographic scoring techniques which allow prediction of good echocardiographic results should retain perspective. The best echocardiographic result still only represents palliation. Furthermore a less impressive echocardiographic result is not necessarily failed therapy but less effective palliation.

Two-dimensional echocardiography allows visualisation of the mitral valve and associated structures in the majority of patients.[22,23] With two-dimensional echocardiography it is possible to measure the mitral valve area by planimetry. This planimetric valve area usually corresponds to the valve area derived from catheterisation data with the Gorlin formula.[24] The echocardiographic examination also allows assessment of other valvular abnormalities.

Doppler derived measurements of pressure (derived from measurements of velocity) correlate well with values determined by invasive study.[25] Measurement of pressure half-time (the time taken for the initial transmitral gradient to fall to half) is useful in patients in whom adequate cross sectional views of the valve cannot be obtained to assess valve area by planimetry.[26]

Measurement of the mitral annulus diameter during an echocardiographic examination may be needed to predict final balloon size (and hence the initial balloon size) when valvuloplasty is performed with techniques not utilising the Inoue balloon device.[27] The authors were able to determine that a ratio of 1 when comparing final balloon diameter (in their case the sum of 2 balloons) to the annulus diameter resulted in the best results as regards final balloon diameter. A ratio of greater than 1.1 resulted in the occurrence of significant mitral regurgitation.

The detection of mitral regurgitation with conventional Doppler pulsed-wave, continuous-wave or colour flow techniques is easily performed. However the quantification of regurgitation is difficult and may not compare to angiography (which could itself be considered to be a *tarnished* 'gold standard') except when regurgitation is trivial or severe.[28] Recent modifications of method (proximal flow convergence) may permit colour flow Doppler to

become equivalent to angiography in the assessment of regurgitation.[29]

In patients in whom adequate transthoracic information cannot be obtained or in whom the presence of intra-atrial clot is suspected and cannot be excluded, transoesophageal echocardiography may be of use. An elegant comparison of transoesophageal and transthoracic techniques in patients with mitral stenosis undergoing valvuloplasty has been performed by Rittoo and colleagues.[30] As would be expected because of the better quality of transoesophageal imaging, the views of the mitral valve were more detailed than those obtained with precordial imaging. Nevertheless, adequate information could be obtained transthoracically. The assessment of the subvalvular structures was *easier* with transthoracic views because of difficulty with obtaining transgastric views of the heart. The difficulty is apparently related to the size of the left atrium in patients with mitral stenosis. Transoesophageal techniques are superior for assessing left atrial clot and left-to-right shunts at an atrial level.

The echocardiographic grading system of Abascal, Wilkins and co-authors from Boston has become widely used. Although initially reported for a series of only 22 patients, subsequent work has 'validated' the system [18,31]

Four major categories are used to describe the structure of the mitral valve and a grading system (which is subjective) is used which attaches to each category a maximum score of four points. In the work of Abascal a score greater than 12 was associated with poor short- and intermediate-term results and a score less than 8 is associated with the best results. The scoring system is reproduced below.

Table 1 **Grading of mitral valve characteristics from the echocardiographic examination** *(Modified after Wilkins GT et al[31])*

Grade	Mobility	Subvalvar thickening	Thickening	Calcification
1	Highly mobile valve with only leaflet tips restricted	Minimal thickening just below the mitral leaflets	Leaflets near normal in thickness	A single area of increased echo brightness
2	Leaflet mid and base portions have normal mobility	Thickening of chordal structures extending up to one third of the chordal length	Mid-leaflets normal, considerable shortening of margins	Scattered ares of brightness confined to leaflet margins
3	Valve continues to move forward in diastole mainly from the base	Thickening extending to the distal third of the chords	Thickening extending through the entire leaflet	Brightness extending to the midportion of the leaflets
4	No or minimal forward movement of the leaflets in diastole	Extensive thickening and shortening of all chordal structures extending down to the papillary structures	Considerable thickening of all leaflet tissue	Extensive brightness throughout much of the leaflet tissue

The scoring system is usually easily used by echocardiographers although the complete assessment of the subvalvular apparatus requires unorthodox views.[31] Although the scoring system is useful to predict a 'good' or 'bad' outcome, it has limitations which both the originators of the system and its critics have pointed out. The score does not correlate well with the absolute increase in valve area.[32] In addition, the four components are arbitrarily given equal weighting. Subsequent multivariate analysis shows that valve thickness alone was the best predictor of increase in valve area.

Reid and co-workers used a quantitative system of measurement of mobility and thickness in their analysis and showed that the quantative assessment of mobility was superior to subjective scoring in predicting final valve area after double balloon valvuloplasty.[33] As with Abascal and co-workers, they conclude that echocardiographic assessment of subvalvar disease and calcification is complicated and difficult to quantify. Leaflet thickening, calcification and subvalvar disease may contribute to mitral valve leaflet mobility.

Chen and colleagues modified the Boston group's scoring system by assigning the maximum score for subvalvar disease only if severe disease was observed in all subvalvar structures and if there was absence of separation of the chordae arising from the anteromedial and posterolateral papillary muscles - grade 2 subvalvar change becoming equivalent to grade 3 of the Boston grading. Subsequent analysis resulted in subvalve apparatus disease being identified as an independent risk factor for a suboptimal result. (Total score did *not* predict a poor result). In their group of patients which were younger than Wilkins' group a high score was not necessarily related to subvalve disease.[27] It is felt that the underestimation of disease in the submitral apparatus may contribute to post dilatation mitral regurgitation.[34]

The use of scoring as the sole means of 'triaging' candidates for valvuloplasty is not recommended - Abascal and co-workers point out that 'a high echocardiographic echo score does not preclude the possibility of a good result.'[32] Score may, however, be a predictor of recurrent stenosis.[35]

Otto et al have reviewed the use of echocardiography in those patients entered into the National Heart, Lung and Blood Institute registry of valvuloplasty techniques in the United States.[36] Unlike the 'method' papers which have evaluated individual echocardiographic techniques, the use of these techniques in the registry may represent the use of echocardiography in the real world. All echocardiographically determined measures of area and gradient correlated poorly with their haemodynamic equivalents. The 'non-simultaneous' performance of techniques, observer bias and variations in technical expertise, as well as changes in transvalvular flow contribute to this poor correlation. Nevertheless, after valvuloplasty, the magnitude of change is similar whatever technique is used. In this group of patients the difference between methods was less than $0.5cm^2$ in 96% of patients.[36]

REFERENCES FOR CHAPTER 4

1. Ellis LB, Singh JB, Morales DD, Harken DE. Fifteen- to twenty-year study of one thousand patients undergoing closed mitral valvotomy. Circulation 1973;48:357-364.

2. Kirklin JW, Barratt-Boyes BG. Mitral valve disease with or without tricuspid valve disease. In: Cardiac Surgery. New York: Churchill Livingstone, 1986:323-372.

3. Inoue K, Owaki T, Nakamura T, Kitamura F, Miyamoto N. Clinical application of transvenous mitral commissurotomy by a new balloon catheter. J Thorac Cardiovasc Surg 1984;87:394-402.

4. Sellors TH, Bedford DE, Somerville W. Valvotomy in the treatment of mitral stenosis. Br Med J 1953;2:1059-1067.

5. Al Zaibag M, Ribero PA, Al Kasab S, Al Fagih MR. Percutaneous double-balloon mitral valvotomy for rheumatic mitral stenosis. Lancet 1986;1:757-761.

6. Palacios IF, Lock JE, Keane JF, Block PC. Percutaneous transvenous balloon valvotomy in a patient with severe calcific mitral stenosis. J Am Coll Cardiol 1986;7:1416-1419.

7. Lefèvre T, Bonan R, Serra A, Crépeau J, Dyrda I, Petitclerc R, Leclerc Y, Vanderperren O, Waters D. Percutaneous mitral valvuloplasty in surgical high risk patients. J Am Coll Cardiol 1991:17:348-354.

8. Wisenbaugh T. Complex balloon mitral valvuloplasty. J Heart Valve Dis 1993;2:218-222.

9. Smith WM, Neutze JM, Barratt-Boyes BG, Lowe JB. Open mitral valvotomy. Effect of preoperative factors on result. J Thorac Cardiovasc Surg 1981;82:738-751.

10. Hung JS, Chern MS, Wu JJ, Fu M, Yeh KH, Wu YC, Cherng WJ, Chua S, Lee CB. Short and long-term results of catheter balloon percutaneous transvenous mitral commissurotomy. Am J Cardiol 1991;67:854-862.

11. Chen CR, Cheng TO, Chen JY, Zhou YL, Mei J, Ma TZ. Percutaneous balloon mitral valvuloplasty for mitral stenosis with and without associated aortic regurgitation. Am Heart J 1993;125:128-137.

12. Block PC. Percutaneous balloon valvuloplasty. In: Hurst JW, Schlant RC, Rackley CE, Sonnenblick EH, Kass Wenger N, eds. The Heart. 7th ed. New York: McGraw-Hill, 1990:2162-2176.

13. Grossman W. Cardiac catheterization. Historical perspective and present practice. In: Grossman W, Baim DS, eds. Cardiac catheterization, angiography and intervention. 4th ed. Philadelphia: Lea and Febiger, 1991:3-14.

14. Hung JS, Lin FC, Chiang CW. Successful percutaneous transvenous catheter balloon mitral commissurotomy after warfarin therapy and resolution of left atrial thrombus. Am J Cardiol 1989;64:107-109.

15. Tsai LM, Hung JS, Chen JH, Lin LJ, Fu M. Resolution of left atrial appendage thrombus in mitral stenosis after warfarin therapy. Am Heart J 1991;121:1232-1234.

16. Hung JS. Mitral stenosis with left atrial thrombi: Inoue balloon catheter technique. In: Cheng TO, ed. Percutaneous balloon valvuloplasty. New York: Igaku-Shoin, 1992:280-293.

17. Turi ZG. Valvuloplasty. Cardiovasc Clin 1993;23:293-326.

18. Holmes DR, Nishimura RA. Balloon valvuloplasty. Adv Intern Med 1992;37:363-389.

19. Le Feuvre C, Bonan R, Lachurie ML, Leclerc Y, Peticlerc R, Dyrdha I, Crépeau J. Balloon mitral commissurotomy in patients aged ≥ 70 years. Am J Cardiol 1993;71:233-236.

20. Davidson CJ, Bashore TM, Mickel M, Davis K. Balloon mitral commissurotomy after previous surgical commissurotomy. Circulation 1992;86:91-99.

21. Babic UU, Grujicic S, Popovic Z, Djurisic Z, Pejcic P, Vucinic M. Percutaneous transarterial balloon dilatation of the mitral valve: five year experience. Br Heart J 1992;67:185-189.

22. Tajik AJ, Seward JB, Hagler DJ, Mair DD, Lie JT. Two-dimensional real-time ultrasonic imaging of the heart and great vessels. Technique, image orientation, structure identification, and validation. Mayo Clin Proc 1978;53:271-303.

23. Come PC, Riley MF. M Mode and cross-sectional echocardiographic recognition of fibrosis and calcification of mitral valve chordae and left ventricular papillary muscles. Am J Cardiol 1982;49:461-466.

24. Smith MD, Handshoe R, Handshoe S, Kwan OL, DeMaria AN. Comparative accuracy of two-dimensional echocardiography and Doppler pressure half-time methods in assessing severity of mitral stenosis in patients with and without prior commissurotomy. Circulation 1986;73:100-107.

25. Stamm RB, Martin RP. Quantification of pressure gradients across stenotic valves by Doppler ultrasound. J Am Coll Cardiol 1983;2:707-718.

26. Chen C, Abascal VM. Echocardiographic evaluation. In: Cheng TO, ed. Percutaneous balloon valvuloplasty. New York: Igaku-Shoin, 1992:127-184.

27. Chen C, Wang X, Wang Y, Lan Y. Value of two-dimensional echocardiography in selecting patients and balloon sizes for percutaneous balloon mitral valvuloplasty. J Am Coll Cardiol 1989;14:1651-1658.

28. Spain MG, Smith MD, Grayburn PA, Harlamert EA, DeMaria A, O'Brien M, Ling Kwan O. Quantitative assessment of mitral regurgitation by Doppler color flow imaging: angiographic and hemodynamic correlations. J Am Coll Cardiol 1989;13:585-590.

29. Rivera JM, Vandervoort PM, Thoreau DH, Levine RA, Weyman AE, Thomas JD. Quantification of mitral regurgitation with the proximal flow convergence method: A clinical study. Am Heart J 1992;124:1289-1296.

30. Rittoo D, Sutherland GR, Currie P, Starkey IR, Shaw TRD. The comparative value of transesophageal echocardiography and transthoracic echocardiography before and after percutaneous mitral balloon valvotomy: a prospective study. Am Heart J 1993;125:1094-1105.

31. Wilkins GT, Weyman AE, Abascal VM, Block PC, Palacios IF. Percutaneous balloon dilatation of the mitral valve: an analysis of echocardiographic variables related to outcome and mechanism of dilatation. Br Heart J 1988;60:299-308.

32. Abascal VM, Wilkins GT, O'Shea JP, Choong CY, Palacios IF, Thomas JT, Rosas E, Newell JB, Block PC, Weyman AE. Prediction of successful outcome in 130 patients undergoing percutaneous balloon mitral valvotomy. Circulation 1990;82:448-456.

33. Reid CL, Chandraratna AN, Kawanishi DT, Kotlewski A, Rhahimtoola SH. Influence of mitral valve morphology on double-balloon catheter balloon valvuloplasty in patients with mitral stenosis. Analysis of factors predicting immediate and 3-month results. Circulation 1989;80:515-524.

34. Essop MR, Wisenbaugh T, Skoularigis J, Middlemost S, Sareli P. Mitral regurgitation following mitral balloon valvotomy. Differing mechanisms for severe versus mild-to-moderate lesions. Circulation 1991;84:1669-1679.

35. Palacios IF, Block PC, Wilkins GT, Weyman AE. Follow-up of patients undergoing percutaneous mitral balloon valvotomy. Circulation 1989;79:573-579.

36. Otto CM, Davis KB, Holmes DR, O'Neill W, Ferguson J, Bashore TM, Bonan R. Methodologic issues in clinical evaluation of stenosis severity in adults undergoing aortic or mitral balloon valvuloplasty. Am J Cardiol 1992;69:1607-1616.

CHAPTER 5

THE PATHOLOGICAL CONSEQUENCES OF BALLOON VALVULOPLASTY

The splitting of one or both fused commissures is the presumed mechanism whereby the performance of closed surgical valvotomy is able to relieve mitral stenosis.[1] The mechanism whereby balloon dilatation relieves mitral stenosis could have been predicted to be similar. Inoue demonstrated that commissural splitting occurred in a series of patients undergoing open mitral valvotomy where a balloon was used intra-operatively to dilate the valve.[2]

Kaplan and co-workers, in an in vitro study of excised stenotic valves (removed from patients undergoing valve replacement), demonstrated that commissural splitting occurred when a valvuloplasty balloon was inflated within the stenotic valve. Interestingly, calcification of the leaflets did not preclude splitting of the commissures. The most important caveat to their findings (noted by the authors) was that the in vitro valves did not include an intact submitral apparatus. Therefore the effect of balloon dilatation on the subvalvular apparatus could not be determined.[3]

Ribeiro and colleagues were able to demonstrate similar findings using a double-balloon technique. (Interestingly, they were able to show that double balloon techniques resulted in a larger resultant area than single balloons). Their findings were documented in excised valves from patients undergoing valve replacement. A portion of these valves (41%) had attached subvalvular chordae but splitting of these structures was not seen.[4] McKay and co-workers documented similar findings in postmortem dilatations of valves from patients who had

evidence of mitral stenosis at autopsy.[5]

Block's group provided similar results in two patients who had undergone valvuloplasty. One patient had died following an unrelated septicaemia and at autopsy commissural splitting was demonstrated. In their second patient, who had mitral valve replacement, they were able to demonstrate that balloon dilatation had not achieved the desired effect because of severe subvalvular disease. Although the commissures had been slightly split, the subvalvular fibrosis had not been affected.[6]

Reid and co-workers were able to demonstrate that splitting of the commissures occurred in successful balloon valvuloplasty by careful cross-sectional echocardiographic studies before and after the balloon procedure.[7] They argued that in their patients the documented increase in the intercommissural distance was not as a result of stretching of the valve leaflets but as a result of commissural tearing. They also documented that poorer results occurred in patients with calcification of the commissures - the commissures failed to split if both were calcified.

In all of the studies discussed above, tears in the leaflets had been produced in a small proportion of the valves. It was postulated that similar tearing in life would produce mitral regurgitation.[3,4,5] In another study Sadee and Becker demonstrated that significant subvalvular disease was associated with an increase risk of tearing. In these patients the tearing did not originate in the leaflets but in the fused chordal structures.[8] In life, Vahanian et al reported only paracommissural tears in the eight patients in their series who required mitral valve replacement following balloon valvuloplasty. All the patients had extensive valvular or subvalvular disease.[9] Similar findings have been noted with the Inoue technique.[10]

REFERENCES FOR CHAPTER 5

1. Austen WG, Wooler GH. Surgical treatment of mitral stenosis by the transventricular approach with a mechanical dilator. N Engl J Med 1960;263:661-665.

2. Inoue K, Owaki T, Nakamura T, Kitamura F, Miyamoto N. Clinical application of transvenous mitral commissurotomy by a new balloon catheter. J Thorac Cardiovasc Surg 1984;87:394-402.

3. Kaplan JD, Isner JM, Karas RH, Halaburka KR, Konstam MA, Hougen TJ, Cleveland RJ, Salem DN. In vitro analysis of mechanisms of balloon valvuloplasty of stenotic mitral valves. Am J Cardiol 1987;59:318-323.

4. Ribeiro PA, Al Zaibag M, Rafendran V, Ashmeg A, Al Kasab S, Al Faraidi Y, Halim M, Idris M, Al Fagih MR. Mechanism of mitral valve area increase by in vitro single and double balloon mitral valvotomy. Am J Cardiol 1988;62:264-269.

5. McKay RG, Loch JE, Safian RD, Come PC, Diver DJ, Baim DS, Berman AD, Warren SF, Mandell VE, Royal HD, Grossman W. Balloon dilatation of mitral stenosis in adult patients: postmortem and percutaneous mitral valvuloplasty studies. J Am Coll Cardiol 1987;9:723-731.

6. Block PC, Palacios IF, Jacobs ML, Fallon JT. Mechanism of percutaneous mitral valvotomy. Am J Cardiol 1987;59:178-179.

7. Reid CL, McKay CR, Chandraratna PAN, Kawanishi DT, Rahimtoola SH. Mechanisms of increase in mitral valve area and influence of anatomic features in double-balloon, catheter balloon valvuloplasty in adults with rheumatic mitral stenosis: a Doppler and two-dimensional echocardiographic study. Circulation 1987;76:628-636.

8. Sadee AS, Becker AE. In vitro balloon dilatation of mitral valve stenosis: the importance of subvalvular involvement as a cause of mitral valve insufficiency. Br Heart J 1991;65:277-279.

9. Vahanian A, Michel P, Cormier B, Vitoux B, Michel X, Slama M, Sarano LE, Trabelsi S, Ben Ismail M, Acar J. Results of percutaneous mitral commissurotomy in 200 patients. Am J Cardiol 1989;63:847-852.

10. Hernández R, Macaya C, Banuelos C, Alfonso F, Goicolea J, Inquez A, Fernandez-Ortiz A, Castillo J, Aragoncillo P, Aguado MG, Zarco P. Predictors, mechanisms and outcome of severe mitral regurgitation complicating percutaneous mitral valvotomy with the Inoue balloon. Am J Cardiol 1992;70:1169-1174.

CHAPTER 6

COMPLICATIONS

The 'average' balloon valvuloplasty consists of two phases, a confirmatory diagnostic catheterisation and the valvuloplasty procedure itself. Attribution of complications to the catheterisation rather than the valvuloplasty is probably not valid, since it can be argued that no invasive data are needed when echocardiographic data are available. Catheterisation was not always performed before closed mitral valvotomy in the pre-echocardiography era.[1]

The incidence of complications may differ markedly according to the population selected for the procedure.

The reports of misadventure can be divided into three groups:

1) Where complications are reported as part of a report of a centre's or group's experience;
2) Where complications are of interest or very unusual; and
3) In 'registry' type reports which focus on data collected prospectively relating specifically to complications and which represent the work of a number of centres and operators.

In a large collection of prospectively acquired data, the 'serious' complication rate was 12%.[2] 'Serious' included death, cardiac perforation, cardiac tamponade, the need for emergency surgery, embolic events (including cerebrovascular accidents), myocardial infarction,

pulmonary embolus, and acute severe mitral regurgitation.

The perception that certain events are important complications of a procedure may alter with time. The iatrogenic atrial septal defect is such an event - its incidence is widely reported, yet its presence is of little relevance (see below). Techniques of measurement may also differ making it difficult to compare groups; for example, methods of quantitation of mitral regurgitation and left to right shunting vary widely. Furthermore the publication of complication rates in older 'First World' populations may not have much relevance to the performance of valvuloplasty in a homogeneous group of patients in a developing country.

Death during the procedure or in the 30 days following the procedure (early mortality) ranges from 0% to 3% according to reports detailing events in more than 400 patients each.[2,3,4] The differences between groups as regards mortality can be attributed to selection criteria as well as the number of operators involved. Inoue and Hung reported the results of the procedure in 527 patients who had valvuloplasty procedures performed by 8 operators.[3] Vahanian's series of 600 patients underwent the procedure at *one* hospital.[4] In contrast, the largest series reported from the United States details the experience in 24 sites with at least as many operators.

In all of these reports, initial experience was gained with carefully screened patients who would have been considered ideal candidates for surgical commissurotomy. With increasing experience, less 'favourable' patients were selected. All series include small numbers of patients who were at high risk for surgery as well as balloon valvuloplasty. The incidence of death (and all complications) is noted to decrease with increasing operator experience (the

'learning curve') and with increasing patient numbers at individual centres.[2,5] In the NHLBI registry, centres who performed valvuloplasty procedures on less than 25 patients had 6 times the mortality of centres involved with more than 100 patients.[2]

The reasons for early mortality are diverse. Ventricular perforation, either as a result of the balloon device perforating the ventricle or secondary to the transseptal puncture is the most common reason for procedural death in the NHLBI series. Other problems such as coronary emboli, death during mitral valve replacement after the creation of severe mitral regurgitation and persistent pulmonary hypertension also resulted in deaths.[2,4] A high echo score (greater than or equal to 13) was shown to be a predictor of mortality when the data was subjected to multivariate analysis.[2]

In the NHLBI registry, half the early deaths were related to the procedure. The other deaths were predominantly non cardiac and are probably the result of the procedure being attempted in high risk candidates. The rate of death in patients of greater than 70 years of age was 14% versus 2% in younger patients. This trend (higher mortality in older patients) has been confirmed by other authors. Le Feuvre et al showed that their patients older than 70 years (only 28 patients - 10% of their experience) had a mortality of 12% versus the 0.8% mortality of the rest of the group. Complications in general were common in the older group (27% vs 9%).[6]

It is tempting to ascribe mortality to a particular technique or balloon type. Although the large series of 527 patients reported by Inoue and Hung was free from mortality, two other series reporting events in more than 200 patients each have recorded 0.5% mortality when the Inoue

balloon was used.[7,8] Both deaths occurred in patients in extremis prior to the procedure however.

Perforation of a cardiac chamber is reported to occur in 4% of procedures.[2] In the registry the perforation was a result of balloon or guidewire perforation and not as a result of attempted transseptal puncture. In the North American Inoue registry, the transseptal puncture resulted in cardiac perforation in 1.5% of procedures.[7]

The use of non-transseptal (ie retrograde) techniques is associated with less risk of cardiac perforation, as would be expected. In a series of 150 patients reported from Greece, the incidence of cardiac perforation was 0%.[9] The larger series of Babic et al, in which a transseptal puncture was created to stabilise a retrogradely introduced balloon device, had a 2% incidence of cardiac perforation.[10]

Cardiac perforation is not always associated with tamponade and may be treated expectantly. In the NHLBI registry all perforations resulted in tamponade.[2] In the North American Inoue study one of three perforations did not result in cardiac tamponade. In the series of Vahanian and Inoue, asymptomatic perforation is not documented as a separate entity. Proponents of the Inoue balloon claim that the absence of a left ventricular guidewire decreases the incidence of ventricular perforation.[3]

Bassand et al's series documented a higher incidence of tamponade in the double balloon group as opposed to the Inoue group (3/161 vs 0/71).[11] In the series of Abdullah et al, again comparing these two balloon types, the incidence of tamponade was identical (1/60 vs 1/60).[12]

In the randomised trial from Park and colleagues no instances of tamponade were reported in either group.[13]

The potential to cause severe mitral regurgitation is common to all types of technique. The mechanisms whereby severe regurgitation is caused have been alluded to in a previous section. An increase in mitral regurgitation (measured either by left ventricular angiography or echocardiography) by at least one grade (using conventional subjective grading techniques) occurs in up to 51% of patients undergoing balloon valvuloplasty.[14] Severe mitral regurgitation defined variously as an increase of greater than 2 or 3 grades, however, occurs in far fewer patients (6.6% in Hernandez et al group, 1.9% in Inoue/Hung's group, 3.8% of Vahanian's group). Between 50 and 100% of these patients will require mitral valve replacement.[3,4,14] Most series report a small number of patients (usually less than 5%) whose degree of mitral regurgitation is considered to be *less* immediately post procedure.[8,12]

The majority of patients with lesser degrees of mitral regurgitation tend to remain unchanged with time.[15,16] In the reports of Ribeiro et al and Pan et al, approximately 10% of patients with some degree of mitral regurgitation post procedure (not necessarily new) experienced further change during the year after valvuloplasty. 2 to 5% developed grade 3+ or worse mitral regurgitation.

Minor degrees of mitral regurgitation are well tolerated.[3] The propensity for a particular technique to cause more regurgitation has been debated. Hernandez and co-workers have argued that it is easier to cause mitral regurgitation with the Inoue balloon. The balloon is more readily placed across a deformed valve and could more easily result in subvalvular

apparatus damage. (In a severely deformed valve it may be impossible to obtain a stable position with the double balloon apparatus - hence inflation may not even be feasible).[14]

In the comparison of techniques performed by Abdullah and co-workers there was no difference in the techniques as regards mitral regurgitation.[12] Undefinable variables such as experience and careful patient selection may have played a role. The randomised trial of Park et al reported similar results between balloon techniques as regards mild and severe regurgitation, although the authors deviated from the suggested step-wise approach and used a single inflation at optimum diameter.[13] (The effective balloon dilating area to body surface area ratio of 4 cm^2/m^2 as suggested for the double balloon technique was used).

In three large series embolic events related to the procedure occurred in 0.6% to 3.3%.[2,3,4] These emboli were predominantly cerebral. In the NHLBI registry patients with a left atrial thrombus or previous embolic event were at higher risk of embolic events during the procedure. Increasing use of transoesophageal echocardiography and techniques of anticoagulation have the potential to decrease the incidence of such events. Thrombus or air may in addition occlude a coronary artery resulting in myocardial infarction.[2]

A plethora of publications have detailed the incidence of atrial septal defect post procedure. In all transatrial techniques a small hole should be present in the atrial septum post procedure. The detection of left to right shunting through the defect depends on the technique of measurement and differing perceptions of 'significance' (ie what represents a significant left to right shunt). Up to 14% of patients will have a left to right shunt defect immediately post procedure if a Qp/Qs ratio of >1.3 is used.[4] Very few of these shunts are clinically

significant.[4] On the other hand colour flow imaging with transoesophageal techniques may demonstrate a left to right shunt in up to 95% of patients examined post procedure.[17]

When Qp/Qs is derived from the difference in thermodilution cardiac output when the iatrogenic septal defect is occluded and when it is patent, the incidence of haemodynamically detected shunting approaches colour flow techniques.[18] The use of the Fick principle immediately post valvuloplasty has also been questioned, since many patient variables are changing at that time and oxygen consumption measurement may not be accurate.[19]

The size of the atrial septal defect is related to the catheter techniques utilized and the number of punctures made in the atrial septum. Fields and co-workers demonstrated (in vitro) that two punctures and simultaneous withdrawal rather than sequential withdrawal of catheters resulted in the creation of larger defects.[20]

The majority of atrial septal defects tend to close with time.[21] The persistence of a septal defect may relate to its initial size (and hence to the technique used) and to inadequate treatment of the stenosed mitral valve.[22]

Arterial complications necessitating surgical repair of the femoral artery are obviously more common in transarterial techniques. In the series of Babic and co-workers the reported incidence was 2%.[10] In contrast, in the NHLBI registry the need for vascular surgery was 0.4%.[2]

Most studies report other complications such as the need for blood transfusion secondary to

bleeding from the femoral vein, prolonged hypotension and transient arrhythmias occurring in less than 10% of patients.[2,4,23] Other rare complications have been documented (NHLBI) usually on a single case per series basis.

The table below summarises the incidence of major complications in three series containing more than 400 patients each.

	Vahanian	Inoue/Hung	NHLBI
Attempts	n = 600	n = 527	n = 738
Tech success	n = 581	n = 515	n = 680
Mortality	0.5%	0%	3%
Severe MR	3.8%	1.9%	3%
Tamponade	0.8%	1.5%	4%
Emboli	3.3%	0.6%	3%

REFERENCES FOR CHAPTER 6

1. Commerford PJ, Hastie TW, Beck W. Closed mitral valvotomy: actuarial analysis of results in 654 patients over 12 years and analysis of preoperative predictors of long-term survival. Ann Thorac Surg 1982;33:473-479.

2. Complications and mortality of percutaneous balloon mitral commissurotomy: a report from the National Heart, Lung, and Blood Institute Balloon Valvuloplasty Registry. Circulation 1992;85:2014-2024.

3. Inoue K, Hung JS. Percutaneous transvenous mitral commissurotomy (PTMC): The Far East experience. In: Topol EJ, ed. Textbook of interventional cardiology. Philadelphia: WB Saunders, 1990:887-899.

4. Vahanian A, Michel PL, Cormier B, Ghanem G, Vitoux B, Maroni JP, Cazaux P and Acar J. Immediate and mid-term results of percutaneous mitral commissurotomy. Eur Heart J 1991;12:Suppl B:84-89.

5. Tuzcu EM, Block PC, Palacios IF. Comparison of early versus late experience with percutaneous mitral balloon valvuloplasty. J Am Coll Cardiol 1991;17:1121-1124.

6. Le Feuvre C, Bonan R, Lachurie ML, Leclerc Y, Petitclerc R, Dyrda I, Crépeau J. Balloon mitral commissurotomy in patients aged ≥ 70 years. Am J Cardiol 1993;71:233-236.

7. Hermann HC, Ramaswamy K, Isner JM, Feldman TE, Carroll JD, Pichard AD, Bashore TM, Dorros G, Massumi GA, Sundram P, Tobis JM, Feldman RC, Ramee S. Factors influencing immediate results, complications, and short-term follow-up status after Inoue balloon mitral valvotomy. A North American multicenter study. Am Heart J 1992;124:160-166.

8. Hung JS, Chern MS, Wu JJ, Fu M, Yeh KH, Wu JC, Cherng WJ, Chua S, Lee CB. Short- and long-term results of catheter balloon percutaneous mitral commissurotomy. Am J Cardiol 1991;67:854-862.

9. Stefanadis C, Stratos C, Vlachopoulos C, Kallikazaros I, Triposkiadis F, Androvlakis A, Trikas A, Toutouzas P. Effectiveness and complications of retrograde non-transeptal balloon mitral valvuloplasty. J Am Coll Cardiol 1993;21:429A.

10. Babic UU, Grujicic S, Popovic Z, Djurisic Z, Pejcic P, Vucinic M. Percutaneous transarterial balloon dilatation of the mitral valve: five year experience. Br Heart J 1992;67:185-189.

11. Bassand JP, Schiele F, Bernard Y, Anguenot T, Payet M, Ba SA, Daspet JP, Maurat JP. The double-balloon and Inoue techniques in percutaneous mitral valvuloplasty: Comparative results in a series of 232 cases. J Am Coll Cardiol 1991;18:982-989.

12. Abdullah M, Halim M, Rajendram V, Sawyer W, Al Zaibag M. Comparison between single (Inoue) and double balloon mitral valvuloplasty: Immediate and short-term results. Am Heart J 1992;23:1581-1588.

13. Park SJ, Kim JJ, Park SW, Song JK, Doo YC, Lee SJK. Immediate and one-year results of percutaneous mitral balloon valvuloplasty using Inoue and double-balloon techniques. Am J Cardiol 1993;71:938-943.

14. Hernandez R, Macaya C, Banuelos C, Alfonso F, Goicolea J, Inquez A, Fernandez-Ortiz A, Castillo J, Aragoncillo P, Aguado MG, Zarco P. Predictors, mechanisms and outcome of severe mitral regurgitation complicating percutaneous mitral valvotomy with the Inoue balloon. Am J Cardiol 1992;70:1169-1174.

15. Pan JP, Lin SL, Go JU, Hsu TL, Chen CY, Wang SP, Chiang BN, Chang MS. Frequency and severity of mitral regurgitation one year after balloon mitral valvuloplasty. Am J Cardiol 1991;67:264-268.

16. Ribeiro, PA, Fawzy ME, Mimish L, Awad M, Dunn BE, Arafah MR, Duran CGM. Mitral restenosis and mitral regurgitation 1 year after Inoue mitral balloon valvotomy in a population of patients with pliable mitral valve stenosis. Am Heart J 1993;126:136-140.

17. Rittoo D, Sutherland GR, Currie P, Starkey IR, Shaw TRD. The comparative value of transoesophageal and transthoracic echocardiography before and after percutaneous mitral balloon valvotomy: a prospective study. Am Heart J 1993;125:1094-1105.

18. Manga P, Singh S, Brandis S, Friedman B. Mitral valve area calculations immediately after percutaneous balloon mitral valvuloplasty: effect of the atrial septal defect. J Am Coll Cardiol 1993;21:1568-1573.

19. Block PC, Palacios IF, Block EH, Tuzcu EM, Griffin B. Late (two-year) follow-up after percutaneous balloon mitral valvotomy. Am J Cardiol 1992;69:537-541.

20. Fields CD, Slovenkai GA, Ioner JM. Atrial septal defect resulting from mitral balloon valvuloplasty relation of defect morphology to transeptal balloon catheter delivery. Am Heart J 1990;119:568-576.

21. Nigri A, Alessandri N, Martuscelli E, Mangieri E, Berni A, Comclo F. Clinical significance of small left-to-right shunts after percutaneous mitral valvuloplasty. Am Heart J 1993;125:783-786.

22. Crawford M. Iatrogenic Lutembacher's syndrome revisited. Circulation 1990;81:1422-1424.

23. Herrmann HC, Kleaveland JP, Hill JA, Cowley MJ, Margolis JR, Nocero MA, Zalewski A, Pepine CJ. The M-Heart percutaneous balloon mitral valvuloplasty registry: initial results and early follow-up. J Am Coll Cardiol 1990;15:1221-1226.

CHAPTER 7

RESULTS OF BALLOON VALVULOPLASTY

The measured assessment of the success of balloon valvuloplasty (a good 'technical result') is arbitrary and was based on initial experience and on the assumption that a valve area greater than 1.5cm^2 represented moderate mitral stenosis. A change in mitral valve area of 25% was also added to measure improvement in valve area when valvuloplasty was performed in patients with larger valve areas.[1] Other authors have increased the area change to 50% and added the absence of significant complications as a caveat.[2] Restenosis in the follow up period is also arbitrarily defined either as valve area less than 1.5cm^2 and less than 50% improved, compared to the valve area prior to the valvotomy (using the Gorlin formula).[2] Others define restenosis as 'loss of 50% of the initial gain with valve area < 1.5cm^2 on follow up'.[3] Although the wording of the definitions is different, theoretically they are equivalent.

The need (and the ability) to measure mitral valve area non-invasively has highlighted discrepancies between non-invasive and invasively determined data. A significant weakness in publications comparing methods of measurement is the inappropriate use of the correlation coefficient or 'r' value, which should be used to test an association between two variables and not used to compare methods of measurement.[4] The comparison of methods is also complicated by non-simultaneous measurement. Furthermore, methods are often compared in small numbers of patients.

Prior to valvuloplasty, the valve areas calculated by planimetry, Doppler derived pressure half-time methods and the Gorlin formula are equivalent. Immediately following valvuloplasty, both Doppler derived methods and the use of the Gorlin formula to determine valve area may be inaccurate. Chen et al found that simultaneously measured Doppler valve areas were larger than the values obtained with the Gorlin formula immediately post valvuloplasty. The Doppler derived figure in their patients by 24 hours post procedure, was equivalent to the data obtained by repeat catheterisation.[5] Manga and co-workers found that the Doppler derived values were lower than the invasively derived values until the hole in the atrial septum was occluded with a balloon to remove any left to right shunting which would result in the cardiac output measurement being inaccurate.[6] (The pressure half-time measurements by Doppler were performed during the 24 hour period post valvuloplasty). In the work of Otto et al, detailing echocardiographic measurements in the patients in the NHLBI registry, valve areas post valvuloplasty were consistently lower than haemodynamically derived data. These studies were performed a mean of 38 hours after the procedure.[7]

Thomas et al pointed out that the Doppler derived values are potentially inaccurate, since analysis of the half-time equation should take changes in net chamber compliance and net transmitral gradient into account. These values change in opposite directions in the valvuloplasty setting and 'cancel each other out' as regards their effect on half time measurement.[8] Manga points out that the work of Thomas et al which involved in vitro analysis of the derivation of the simple 220/half-time formula did not take the potential role of the septal defect into account. The modifying influences on the half-time formula have presumably resolved by 24 hours at which time it is suggested that the Doppler derived values

are obtained.[5]

Planimetry of the valve using 2-dimensional echo has also been used in the assessment of valvuloplasty.[9] The technique has limitations in that adequate images of the valve are not obtained in every patient.[10]

In the presence of aortic regurgitation, the half-time measurement of mitral valve area can be inaccurate.[5] Nakatani et al offered an alternative method, using the continuity equation, to estimate mitral valve area in this situation. The measurement was equivalent to the Gorlin measurement immediately post the procedure and declined in the first 24 hours post procedure. (It was not compared to half-time measurements).[11]

The accuracy of echocardiographic or Doppler techniques is relevant since these methods are used to repeat estimations of the valve area during follow-up. Centres attempting to describe the incidence of restenosis in the follow-up period may discover that as with some patients following surgical commissurotomy, 'restenosis' in follow-up may simply be equivalent to failure to enlarge the originally stenosed valve and not time-related loss of gain in valve area.[12]

The haemodynamic changes effected during valvuloplasty tend to be similar whatever technique is used. The table below (Table 1) compares haemodynamic changes achieved in a number of series (for clarity standard deviation data have been omitted). All the valve areas are determined using the Gorlin formula.

Table 1

Author	Type	Complete	Success	MVA pre	MVA post	Grad pre	Grad post
Hung	I	216	77%	1.0	2.0	13	5.7
Block	D	432	79%	0.9	2.0	15	5
Hermann	I	200	72%	1.0	1.8	14	6
Vahanian	M	581	87%	1.1	2.2	11	2.2
Inoue	I	515	NA	1.13	1.97	11.9	5.5
Babic	R	294	100%	1.1	2.3	18	6
NHLBI	85% D	576	76%	1.0	2.0	14	6

Note: R - retrograde technique, I - Inoue balloon, M - mixed series, D - double balloon, MVA - mitral valve area (cm^2), grad - transmitral gradient(mm Hg)

The changes in haemodynamic data between double-balloon and the Inoue techniques are equivalent. Both randomised[13] and comparative series from the same centre have been reported in the literature.[14,15]

Changes in other haemodynamic measures such as left atrial pressure measurements, and cardiac output measures are also well documented. A typical change in left atrial pressure is 24.2 $\pm$ 5.1 to 5.7 $\pm$ 2.6.[16] Cardiac output tends to increase. (In the series of Hung et al 4.4 $\pm$ 1.4 to 4.7 l/m $\pm$ 1.2).[16] These data are consistent between techniques.[14] In the small series of Manga et al where the septal defect is occluded, the change in cardiac output by thermodilution was less than that seen when the atrial septal defect was open.[6]

Pulmonary artery pressure measurements tend to decrease following valvuloplasty[3] but the magnitude of the change is much smaller than the change in the transmitral gradient in the same patient suggesting that either a degree of fixed pulmonary vascular disease is present or that these changes resolve more slowly. Patients with severely elevated pressure (> 50mm Hg systolic) showed decreases in both pulmonary artery pressure and resistance, but both values remained supranormal.[17]

Alfonso and colleagues studied a group of patients with severe pulmonary hypertension (defined by them as > 60mm Hg systolic pulmonary artery pressure) who underwent valvuloplasty. Pulmonary artery pressure dropped from 77 ± 15mm Hg to 60 ± 17mm Hg immediately post valvuloplasty. (These figures were equivalent to the doppler derived values in these patients (73± 18 to 54 ± 14)). Follow-up data (in 43 patients) at a mean of 14 ± 3 months showed that a pressure drop of at least 10mm Hg had occurred. (The small numbers limit the usefulness of the study, since 4 patients experienced an increase, and 13 experienced a decrease in pressure. In 18 patients tricuspid regurgitation could not be detected and hence pulmonary pressure could not be estimated). Dev and Shrivavasta obtained catheterisation data in a small series of patients at 1 week and 3 to 12 months after valvuloplasty and showed a drop post procedure and a further decline in vascular resistance at 1 week but no significant decline thereafter.[18]

In all series, clinical improvement is usually reported in terms of the change of the New York Heart Association functional class. The initial status of the patients may vary according to the population the patients come from, but the bulk of patients have class II or III symptoms. Occasionally class I patients undergo valvuloplasty, typically because of the expressed need

to fall pregnant.[19]

Generally the bulk of patients improve to class I or II. For example, in the series reported from Baragwanath hospital, in a population with a median age of 29 years, 65% of the patients were in class III prior to the procedure, 6% in class IV, 28% in class II, and 1% in class I. After the valvuloplasty, 94% were in class I or II with an improvement in functional class of one grade in 91%.[19]

In the NHLBI series, where the patients had a mean age of 54 years 9% of patients were in class I, 27% in class II, 51% in class III and 13% in class IV prior to valvuloplasty. 8% of all patients did not have completed procedures. 85% had improved one class by discharge with 62% of patients in class I.[20]

The improvements in symptoms post valvuloplasty have been measured objectively and patients show increased effort tolerance on treadmill testing three months post procedure.[16] Immediate improvement in exertional symptoms immediately after the procedure is however thought not to relate to immediate improvement in exercise capacity but to relief of excessive exercise ventilation. The changes in exercise capacity may take a few months to become evident and may relate to improved skeletal muscle functioning.[21]

The initial improvement tends to be maintained at follow-up. In the large French series of Vahanian et al[3] consisting of 600 patients with a mean age of 43 years, 23% were class II limited, 73% in class III and 4% in class IV. By 42 months of follow-up 72% of the patients were in class I or II.

In a North American series, in which the majority of patients were treated with the now outmoded single Mansfield balloon technique, 5% were in class II, 60% in class III, and 34% in class IV. Following the procedure, 91% of patients increased by at least one functional grade. The improvement was maintained so that at a mean follow-up of 36 months 96% of patients were still in class I or II.[22]

In the report of Chen et al the results of valvuloplasty in 85 patients who had been followed up from 43 to 79 months (mean 5 years) were analyzed.[23] No patients in that small group had died and 87% were in class I at extended follow up.[23]

The results of balloon valvuloplasty have also been stated in terms of survival data. Vahanian et al have provided actuarial survival data extending to 42 months post procedure. 87% of their patients were alive, and 81% were free of the need for re-operation.[3]

In a group of (350) patients from Spain who underwent valvuloplasty performed with a variety of methods, the 5 year actuarial event-free probability rate was 85% with a 94% probability of survival at that time.[24]

The North American series of Block and Palacios consisting of 320 patients, followed for a mean of 20 months, had a 92% 4 year probability of survival. 87% of the patients at 4 year follow-up had not required mitral valve replacement. 83% of the surviving patients were NYHA class I or II at 4 years.[25]

The recurrence of symptoms post valvuloplasty may relate to a technically inadequate

valvuloplasty (in this instance, a small increase in valve area which, although not reaching the arbitrary criteria for success, probably allowed amelioration of symptoms for at least a short interval), true restenosis of the valve and progression of mitral regurgitation present prior to the valvuloplasty or created during the procedure.

In the small series of Palacios et al, in which a subset of 100 patients underwent re-catheterisation at a mean follow-up of 13 months, the incidence of restenosis was 22%.[26]

Block and co-authors attempted to document restenosis in their patients at 2 years of follow-up. In the group re-catheterised, which may not have been representative of the whole series, 50% of the patients showed restenosis (defined as a decrease of greater than 50% of the area gain). Interestingly, their echocardiographic evaluation showed areas not significantly different from shortly after valvuloplasty, highlighting previously mentioned difficulties with the measurement of mitral area.[27]

In an echocardiographic study of 66 patients from Saudi Arabia with a younger mean age than the North American patients, Ribeiro and co-workers reported an incidence of restenosis of 9% after 1 year of follow-up. (Restenosis was defined as mitral valve area of less than 1.5cm^2 with a minimum loss of 0.4cm^2 of valve area gain). 6% of the patients were classified as having persistent residual restenosis (valve area $< 1.5cm^2$). Despite the echocardiographic evidence for restenosis, nearly all of the patients were still in functional class I or class II.[28]

Mitral regurgitation post valvuloplasty may be the reason for deterioration in functional class. Severe regurgitation post procedure may not lead to valve replacement with patients being

successfully treated with medical therapy in the majority of cases. In the series of Hernandez et al, one third of the patients with severe mitral regurgitation underwent early valve replacement. 90% of the remaining patients had their symptoms controlled medically at a mean of 11 months of follow-up.[29]

The larger series have been subjected to statistical analysis in order to obtain predictors of early and late outcome. The series from the Massachussets General Hospital highlighted a number of categories on univariate analysis which predicted a worse early outcome - including older age, NHYA class, previous surgical commissurotomy, atrial fibrillation, valvular calcification on fluoroscopy and existing mitral regurgitation.[25] The series of Hung and Inoue added large left atrial size and increased cardiothoracic ratio on chest X-ray as univariate predictors of a poor outcome.[30]

Multivariate analysis tends to identify valvar anatomy expressed either as the echocardiographic score or its components as the most important predictor of a good short-term outcome.[3,25,30] While a good score (8 or less) is associated with a higher probability of a good outcome, a higher score does not preclude a good outcome in terms of increase in valve area and the absolute score should probably not be used to predict the outcome of the procedure.[31]

The weighting of individual components of the echocardiographic analysis as a predictor of outcome has been discussed in an earlier section. Commissural morphology may have a more important role as a predictor of outcome than previously realised since the prediction that one or both fused commissures would split has been shown to be a better predictor of a good

outcome than the absolute score.[31]

Similar statistical inferences have been made to predict successful long term outcomes. Pan and co-workers identified atrial fibrillation, valve calcification or fibrosis and a previous commissurotomy as being associated with a higher risk for restenosis. This work is similar to the work of Palacios et al with echo score and atrial fibrillation predicting restenosis over a shorter follow-up period.[26] Advanced age, atrial fibrillation, mitral regurgitation prior to the procedure, increased pulmonary resistance and decreased ejection fraction were identified as being associated with the need for mitral valve surgery. Survival was predicted by younger age, larger valve area prior to valvuloplasty, sinus rhythm, pliable mitral valve and absence of mitral regurgitation as well as absence of calcification and decreased pulmonary resistance. On multivariate analysis atrial fibrillation and valve calcification at fluoroscopy were identified as predictors of poor long term success in a series reporting a 5 year follow-up.[24] On this basis a patient with sinus rhythm and no valvar calcification could be predicted to have a 96% probability of event free survival at 5 years post procedure. A patient with valve calcification and atrial fibrillation is likely to have a 60% probability of event free survival. In the series of Hung et al the long term outlook for the patients with valvular calcification and subvalvular disease was also worse than in the patients with pliable valves.[16]

In the series of Cohen et al a score less than 8, a low left ventricular end-diastolic pressure and a lower NYHA class predicted event-free survival to 5 years on multivariate analysis. A patient with three favourable variables would have an 84% probability of event-free survival at 5 years as opposed to 13% in a patient with no favourable variables.[22]

An alternative means of analysing results is to examine results in individual categories in patients suspected of being less likely to benefit or at higher risk than the patient with an optimal valve. The elderly patient, the patient with a previous commissurotomy and the pregnant patient are examples of such patients. The elderly have been discussed in a previous section. The patients with a previous surgical commissurotomy are as likely to benefit from balloon valvuloplasty if their valvular anatomy is suitable.[32] Pregnant patients can be safely treated with balloon valvuloplasty.[33]

Just as the comparison of the older closed surgical procedure with the more modern open procedure was made difficult by variables in patient selection and evaluation, the comparison of balloon valvuloplasty to its surgical predecessors is difficult. With limited follow-up data in the large series available, the outcome in 'good' patients undergoing surgical commissurotomy is possibly better than patients undergoing balloon valvuloplasty.[34]

Attempts to perform randomised studies comparing surgical commissurotomy to balloon valvuloplasty have been made. As would be expected the total number of patients involved is small. In a study from Durban, utilizing haemodynamic estimation of valve area, balloon valvuloplasty using a bifoil catheter resulted in a larger valve area than closed commissurotomy using a Tubb's dilator. A total of 45 patients with a mean echo score of 6 were entered into the study.[35]

Turi and co-workers randomised 40 patients in a similar study utilising the double balloon technique. In their patients the surgical and the balloon techniques produced equivalent results which were sustained at 8 months post procedure. A significant number of patients did not

return for follow-up lessening the impact of their results.[36] The same group has reported similar preliminary results at 3 years of follow-up in a group of patients treated with open surgical valvotomy or balloon valvuloplasty.[37]

The closed surgical technique and the balloon technique have been compared in a group of 200 patients also from India. By a mean of 22 months of follow-up results were equivalent as regards mortality, haemodynamic improvement and restenosis.[38]

In a study from Tunisia involving 30 patients in each group, open valvotomy was shown to be equivalent to balloon valvuloplasty and better than closed valvotomy at 4 years of follow-up.[39]

All these randomised studies have been performed in patients with relatively pliable valves. The widespread use of the balloon technique in these areas is probably limited by economic reasons alone, since the cost of the disposables in the balloon procedure exceeds the cost of a surgical procedure.[36]

The patient with the less pliable valve may be better dealt with surgically.[34] This group of patients has not been studied in a randomised trial.

Even if balloon valvuloplasty is not equivalent in terms of long term outcome to its surgical predecessors, this will probably not result in it being abandoned. The technique at worse offers a means to non-operatively relieve symptoms for a number of years and to defer the need for a general anaesthetic and a thoracotomy as well as decrease the need for medication.

In terms of 'the unnatural history of mitral stenosis' balloon valvuloplasty is another means of 'turning the clock back.'[40]

REFERENCES FOR CHAPTER 7

1. Abascal VM, Wilkins GT, O'Shea JP, Choong CY, Palacios IF, Thomas JD, Rosas E, Newell JB, Block PC, Weyman AE. Prediction of successful outcome in 130 patients undergoing percutaneous balloon mitral valvotomy. Circulation 1990;82:448-456.

2. Herrmann HC, Ramaswamy K, Isner JM, Feldman TE, Carroll JD, Pichard AD, Bashore TM, Dorros G, Massumi GA, Sundram P, Tobis JM, Feldman RC, Ramee S. Factors influencing immediate results, complications, and short-term follow-up status after Inoue balloon mitral valvotomy: A North American multicenter study. Am Heart J 1992;124:160-166.

3. Vahanian A, Michel PL, Cormier B, Ghanem G, Vitoux B, Maroni JP, Cazaux P, Acar J. Immediate and mid-term results of percutaneous mitral commissurotomy. Eur Heart J 1991;12:Suppl B:84-89.

4. Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. Lancet 1986;1:307-310.

5. Chen C, Wang Y, Guo B, Lin Y. Reliability of the Doppler pressure half-time method for assessing effects of percutaneous mitral balloon valvuloplasty. J Am Coll Cardiol 1989;13:1309-1313.

6. Manga P, Singh S, Brandis S, Friedman B. Mitral valve area calculations immediately after percutaneous balloon mitral valvuloplasty: effect of the atrial septal defect. J Am Coll Cardiol 1993;21:1568-1573.

7. Otto CM, Davis KB, Holmes DR, O'Neill W, Ferguson J, Bashore TM, Bonan R. Methodologic issues in clinical evaluation of stenosis severity in adults undergoing aortic or mitral balloon valvuloplasty. Am J Cardiol 1992;69:1607-1616.

8. Thomas JD, Wilkins GT, Choong CYP, Abascal VM, Palacios IF, Block PC, Weyman AE. Inaccuracy of mitral pressure half-time immediately after percutaneous mitral valvotomy. Dependence on transmitral gradient and left atrial and ventricular compliance. Circulation 1988;78:980-993.

9. Abascal VM, Wilkins GT, Choong CY, Thomas JD, Palacios IF, Block PC, Weyman AE. Echocardiographic evaluation of mitral valve structure and function in patients followed for at least 6 months after percutaneous balloon mitral valvuloplasty. J Am Coll Cardiol 1988;12:606-615.

10. Come PC, Riley MF, Diver D, Morgan JP, Safian RD, McKay RG. Noninvasive assessment of mitral stenosis before and after percutaneous balloon mitral valvuloplasty. Am J Cardiol 1988;61:817-825.

11. Nakatani S, Nagata S, Beppu S, Ishikura F, Tamai J, Yamagishi M, Ohmori F, Kimura K, Takamiya M, Miyatake K. Acute reduction of mitral valve area after percutaneous balloon mitral valvuloplasty: assessment with the Doppler continuity method. Am Heart J 1991;121:770-775.

12. Higgs LM, Glancy DL, O'Brien KP, Epstein SE, Morrow AG. Mitral restenosis: an uncommon cause of recurrent symptoms following mitral commissurotomy. Am J Cardiol 1970;26:34-37.

13. Park SJ, Kim JJ, Park SW, Song JK, Doo YC. Lee SJK. Immediate and one-year results of percutaneous mitral balloon valvuloplasty using Inoue and double-balloon techniques. Am J Cardiol 1993;71:938-943.

14. Abdullah M, Halim M, Rajendran V, Sawyer W, al Zaibag M. Comparison between single (Inoue) and double balloon mitral valvuloplasty: Immediate and short-term results. Am Heart J 1992;123:1581-1588.

15. Bassand JP, Schiele F, Bernard Y, Arguenot T, Payet M, Ba SA, Daspet JP, Maurat JP. The double balloon and Inoue techniques in percutaneous mitral valvuloplasty: Comparative results in series of 232 cases. J Am Coll Cardiol 1991;18:982-989.

16. Hung JS, Chern MS, Wu JJ, Fu M, Yeh KH, Wu YC, Cherng WJ, Chua S, Lee CB. Short and long-term results of catheter balloon percutaneous transvenous mitral commissurotomy. Am J Cardiol 1991;67:854-862.

17. Ribeiro PA, al Zaibag M, Abdullah M. Pulmonary artery pressure and pulmonary vascular resistance before and after mitral balloon valvotomy in 100 patients with severe mitral stenosis. Am Heart J 1993;125:1110-1114.

18. Dev V, Shrivavasta S. Time course of changes in pulmonary vascular resistance and the mechanism of regression of pulmonary arterial hypertension after balloon mitral valvuloplasty. Am J Cardiol 1991;67:439-442.

19. Röthlisberger C, Essop MR, Skudicky D, Skoularigis J, Wisenbaugh T, Sareli P. Results of percutaneous balloon mitral valvotomy in young adults. Am J Cardiol 1993;72:73-77.

20. The National Heart, Lung, and Blood Institute Balloon Valvuloplasty Registry Participants. Multicenter experience with balloon mitral commissurotomy: NHLBI Balloon Valvuloplasty Registry Report on immediate and 30-day follow-up results.

Circulation 1992;85:448-461.

21. Tanabe Y, Suzuki M, Takahashi M, Oshima M, Yamazaki Y, Yamaguchi T, Igarashi Y, Tamura Y, Yamazoe M, Shibata A. Acute effect of percutaneous transvenous mitral commissurotomy on ventilatory and hemodynamic responses to exercise. Pathophysiological basis for early symptomatic improvement. Circulation 1993;88:1770-1778.

22. Cohen DJ, Kuntz RE, Gordon SPF, Piana RN, Safian RD, McKay RG, Baim DS, Grossman W, Diver DJ. Predictors of long-term outcome after percutaneous balloon mitral valvuloplasty. N Engl J Med 1992;327:1327-1325.

23. Chen CR, Cheng TO, Chen JY, Zhou YL, Mei J, Ma TZ. Long-term results of percutaneous mitral valvuloplasty with the Inoue balloon catheter. Am J Cardiol 1992;70:1445-1448.

24. Pan M, Medina A, de Lezo JS, Hernández E, Romero M, Pavlovic D, Melián F, Franco M, Cabrera JA, Romo E, Ortega JR. Factors determining late success after mitral balloon valvotomy. Am J Cardiol 1993;71:1181-1185.

25. Palacios IF, Block PC. Acquired mitral stenosis: double balloon catheter technique. In: Cheng TO, ed. Percutaneous balloon valvuloplasty. New York: Igaku-Shoin, 1992:221-236.

26. Palacios IF, Block PG, Wilkins GT, Weyman AE. Follow-up of patients undergoing percutaneous mitral balloon valvotomy. Analysis of factors determining restenosis. Circulation 1989;79:573-579.

27. Block PC, Palacios IF, Bloch E, Tuzcu EM, Griffen B. Late (two-year) follow-up after percutaneous balloon mitral valvuloplasty. Am J Cardiol 1992;69:537-541.

28. Ribeiro PA, Fawzy ME, Mimish L, Awad M, Dunn BE, Arafah MR, Duran CGM. Mitral restenosis and mitral regurgitation 1 year after Inoue mitral balloon valvotomy in a population of patients with pliable mitral valve stenosis. Am Heart J 1993;126:136-140.

29. Hernandez R, Macaya C, Banuelos C, Alfonso F, Goicolea J, Inquez A, Fernandez-Ortiz A, Castillo J, Aragoncillo P, Aguado MG, Zarco P. Predictors, mechanisms and outcome of severe mitral regurgitation complicating percutaneous mitral valvotomy with the Inoue balloon. Am J Cardiol 1992;70:1169-1174.

30. Inoue K, Hung JS. Percutaneous transvenous mitral commissurotomy (PTMC): The Far East experience. In: Topol EJ, ed. Textbook of interventional cardiology. Philadelphia: WB Saunders, 1990:887-899.

31. Fatkin D, Roy P, Morgan JJ, Fenely MP. Percutaneous balloon mitral valvotomy with the Inoue single-balloon catheter: commissural morphology as a determinant of outcome. J Am Coll Cardiol 1993;21:390-397.

32. Davidson CJ, Bashore TM, Mickel M, Davis K. Balloon mitral commissurotomy after previous surgical commissurotomy. Circulation 1992;86:91-99.

33. Patel JJ, Mitha AS, Hassen F, Patel N, Naidu R, Chetty S, Pillay R. Percutaneous balloon mitral valvotomy in pregnant patients with tight mitral stenosis. Am Heart J 1993;125:1106-1109.

34. Kirklin JW. Percutaneous balloon versus surgical closed commissurotomy for mitral stenosis. Circulation 1991;83:1450-1451.

35. Patel JJ, Shama D, Mitha AS, Blyth D, Hassen F, le Roux BT, Chetty S. Balloon valvuloplasty versus closed commissurotomy for pliable mitral stenosis: a prospective haemodynamic study. J Am Coll Cardiol 1991;18:1318-1322.

36. Turi ZG, Reyes VP, Raju S, Raju R, Kumar DN, Rajagopal P, Sathyanarayana PV, Rao DP, Srinath K, Peters P, Connors B, Fromm B, Farkas P, Wynne J. Percutaneous balloon versus surgical closed commissurotomy for mitral stenosis. A prospective, randomized trial. Circulation 1991;83:1179-1185.

37. Turi ZG, Raju BS, Raju R, Reyes V, Fromm B, Singh S, Farkas P, Stephenson LW, Wynne J. Percutaneous balloon vs open surgical mitral commissurotomy: Three year follow-up of a randomised trial. Circulation 1993;88:I-339.

38. Arora R, Nair M, Kalra GS, Nigam M, Khalilullah M. Immediate and long-term results of balloon and surgical closed mitral valvotomy: A randomized comparative study. Am Heart J 1993;125:1091-1094.

39. Farhat MB, Ayari M, Betbout F, Maatouk F, Gamra H, Jarrar M, Esseghairi K, Cherif A. Percutaneous balloon versus surgical closed and open mitral commissurotomy. J Am Coll Cardiol 1993;21:428A.

40. Braunwald E. Valvular heart disease. In: Braunwald E, ed. Heart disease. A textbook of cardiovascular medicine. 4th ed. Philadelphia: WB Saunders, 1992:1007-1077.

CHAPTER 8

THE RESULTS OF PERCUTANEOUS BALLOON VALVULOPLASTY AT GROOTE SCHUUR HOSPITAL

SECTION A: METHODS, DEFINITIONS AND PROCEDURES

1) Methods

By retrospective review of cardiac clinic records, hospital folders, echocardiography reports and catheterisation reports, data relating to the clinical, echocardiographic and haemodynamic variables relating to each valvuloplasty procedure were extracted. Procedures performed from the 18th March 1988 (when balloon mitral valvuloplasty was first performed at Groote Schuur Hospital) until the end of November 1992, form the basis for the study.

Data were entered onto a commercially available database program (Paradox 3, Borland International). Statistical analyses were performed using Epistat Version 3.2 and Epi Info Version 5 software on an IBM compatible personal computer.

Clinical data utilised are the opinions of one of four consultant cardiologists involved in patient care during the period under review. Echocardiographic data utilised are the opinions of one of two consultant cardiologists. Echocardiographic images were not available for review. The commonly used echocardiographic scoring developed in Boston appears to have been used from August 1989 at Groote Schuur hospital.[1]

Haemodynamic data are those as listed in the printed catheterisation report for each procedure. These data were cross-checked with the haemodynamic data reported at the time of the joint departmental catheterisation review meeting held one to three days following the procedure. Recordings of haemodynamic data were checked only when data were incomplete or inconsistent.

Follow-up clinical data were obtained from the clinical records.

Procedures are assigned to a specific balloon type if reference is made to that type on the record sheet or in the clinical notes.

Assignment to a specific New York Heart Association Class is inferred from symptoms listed in the clinical record, unless the attending clinician has allocated the patient to a specific class.

Early mortality is defined as death during the first 30 days post valvuloplasty.

2) Statistical methods

Standard descriptive statistics are used to describe groups. Chi-square and Fisher's exact test are used for discrete variables where appropriate. The signed rank test was used to evaluate paired non-parametric continuous variables. T-tests were used to evaluate independent samples. (Data sets generally had sample sizes greater than 30, allowing the use of parametric statistical testing in samples with potentially non-parametric distributions).

3) Additional definitions

Valvuloplasty procedures were considered **'attempted'** if balloon valvuloplasty was the stated aim of the catheterisation procedure as described in the clinical notes, prior to the procedure. Valvuloplasty procedures were considered to have been **'completed'** if at least one balloon inflation across the mitral valve occurred.

Valvuloplasty procedures are considered **'successful'** if no significant complication (tamponade, death or torrential mitral regurgitation) occurred following balloon inflation.

'Technical success' refers to an increase in mitral valve area (as measured at catheterisation) of 50% with respect to original valve area, without the creation of more than 2+ mitral regurgitation. Data referring to an alternate definition of success, that is an increase in valve area to 1,5cm^2 or greater, will also be provided.

4) Attempted valvuloplasty procedures

Table 1 lists the number of procedures performed per year. An average of 2.2 attempted procedures were made per month over a 57 month period.

Table 1

YEAR	No. of Attempts
1988	18
1989	15
1990	33
1991	30
1992	28

124 procedures were attempted. Six patients had two attempts at balloon valvuloplasty. Four of these patients had a second procedure after the occurrence of a significant complication (usually tamponade) during the first procedure. Two patients had a second balloon valvuloplasty during the 57 month period.

Four procedures were not completed (see earlier definition). Two procedures were abandoned after minor tamponade during transseptal puncture . The procedures were then performed after a period of monitoring in the intensive care unit. During one procedure, the operators were unable to maintain the guidewire position in the left atrium and thus could not pass a balloon into the left atrium (attempted Bifoil catheter placement). During another procedure,

the balloon was inflated by error across the tricuspid valve when it was not recognised that the guidewire had slipped into the right atrium (attempted Inoue balloon placement). This procedure was repeated successfully at a later date.

Unsuccessful procedures occurred in nine of 124 attempts (7%). In some of these patients, symptoms may have been relieved after correction of an adverse event, but this adverse event precluded the accurate calculation of the post valvuloplasty valve area. In others the adverse event was either life threatening or its effect negated any improvement in effort tolerance.

54 procedures were attempted with Bifoil or Trifoil balloons. 70 attempts were made using the Inoue balloon system.

SECTION B: BASE - LINE CHARACTERISTICS

1) Base-line Characteristics

The base-line data of the 118 patients who underwent 124 attempted procedures are listed below (Table 2).

Table 2 **Baseline data.** (modified after Cohen et al[2])

Age	(mean)	30.7	(SD 10.2)
Age	(median)	30	
Males		19	(17%)
Females		99	(83%)
Rhythm -	Atrial Fibrillation	9	(8%)
-	Sinus	109	(92%)
Previous surgical commissurotomy -			
	Open	2	(2%)
	Closed	12	(10%)
Associated cardiac disease - clinical			
	Aortic regurgitation	17	(14%)
	Aortic stenosis/Aortic regurgitation	3	(2.5%)
	Tricuspid valve disease	1	(1%)
Pregnancy		8	(8 % of females)
NYHA class			
	II	24	(20%)
	III	79	(66%)
	IV	15	(13%)
Additional pathology			
	Previous CVA	2	
	SLE	1	
	Epilepsy	3	
	Diabetes	1	
	Thyrotoxicosis	1	
	TB	1	
	Recent caesarian section	1	
	Recent pulmonary embolus	1	
	Mental retardation	1	
	Fibroid uterus	1	

2) Haemodynamic data prior to valvuloplasty

Table 3 documents prevalvuloplasty haemodynamic data as measured at catheterisation. Data were available for 122 patients. (Two procedures were repeated the day after the first attempt).

Table 3 **Data obtained at catheterisation**

Mitral valve area (*n* = 122)	0.9 (0.2) range 0.3 - 1.9 median 0.9cm^2
Mean transmitral gradient (*n* = 122)	16.9 (6.6) mmHg
Mean cardiac output (*n* = 122)	4.1 (1.1) l/min
Mean Left atrial pressure (*n* = 122)	25.9 (7.1) mmHg
Mean pulmonary pressure (*n* = 121)	44.4 (17.1) mmHg

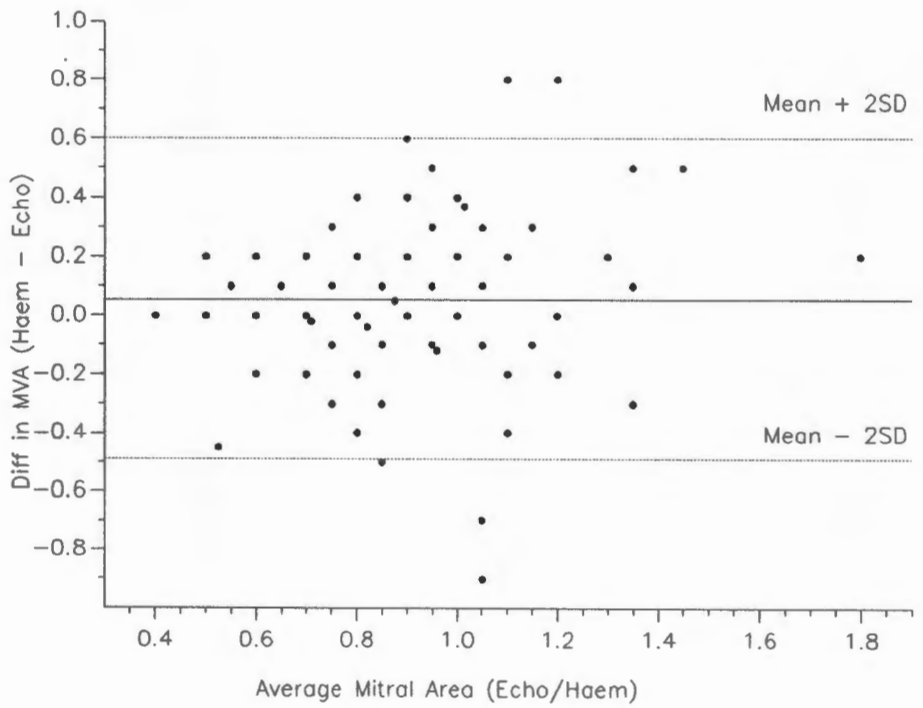
Note: Cardiac output data are the average of the thermodilution and Fick principle outputs.

Figures within brackets represent one standard deviation.

3) Echocardiographic data prior to valvuloplasty

Data relating to valve areas measured prior to the procedure were available in 112 patients. The mean valve area was 0.86(SD 0.24)cm^2. These areas were usually derived from the half-time pressure measurements. The means of the echocardiographically derived data are not significantly different from the data obtained at catheterisation, although data comparing the agreement between the techniques suggests that the measurements obtained in an individual patient may vary widely[3] (Figure 1).

Figure 1 Comparison of methods of measuring mitral valve area



Data reflecting echocardiographic score and the individual components thereof were available in 80 patients and are listed below (Table 4). The scores ranged form 3 to 9. The median score was 5.

Table 4 **Echo score data (median values)**

Mobility	1
Thickening	2
Calcification	0
Subvalvar disease	2
Total	5

Two subjective variables, good and suboptimal, have been assigned retrospectively to the conclusion reported as regards the 'suitability' of the mitral valve on echocardiography. 106 of 120 procedures were attempted in patients with 'good' valves (implying mobile, uncalcified valves). Twelve procedures were in patients with suboptimal valves. In two patients data relating to the echocardiographic appearance of the valves are not available.

In 22 patients tricuspid regurgitation was detected at echocardiography. This was generally classified as 'mild'. 19 patients had mild aortic regurgitation detected at echocardiography. In two patients the aortic regurgitation was moderate in degree. Three patients had mixed aortic valve disease. Four patients had 2+ mitral regurgitation (as determined by mapping with pulse width doppler or colour flow doppler) and 42 patients had 1+ mitral regurgitation.

SECTION C: THE OUTCOME OF BALLOON VALVULOPLASTY

1) The haemodynamic effects of balloon valvuloplasty

Table 5 below lists the effects of balloon valvuloplasty on selected haemodynamic variables. Apart from cardiac output data, significant statistical differences exist in the pre- and post-valvuloplasty populations. No attempt was made to compensate for any effect in change in cardiac output as a result of the creation of an atrial septal defect.

Table 5 **The haemodynamic effects of balloon valvuloplasty**

	Pre-procedure	Post procedure	P value
Mitral valve area	0.9(0.3)	2.0(0.6)	p<0,000001
Mean transmitral gradient	16.9(6.6)	5.3(2.6)	p<0,000001
Mean Left atrial pressure	25.9(7.1)	13.8(5.3)	p<0,000001
Mean Pulmonary pressure	44.4(17.1)	31.7(13.0)	p<0,000001
Cardiac output	4.1(1.1)	4.5(1.1)	p = NS

Of 120 procedures in which at least 1 inflation of the balloon across the mitral valve was achieved, 91 procedures resulted in the achievement of a mitral valve area of 1.5cm^2 or greater, without additional mitral regurgitation of more than 3 grades (76%). 79% of procedures (95) resulted in an increase in the valve area of 50% or more. In 102 procedures (85%) the final valve area had increased to 1.5cm^2 or greater or increased by 50% relative to its starting value or both.

Left to right shunting across an atrial septal defect was sought during the early Groote Schuur experience. Published evidence as to the minor importance of the iatrogenic atrial septal defect has resulted in these shunts not being sought during later procedures.

2) Echocardiographic assessment of the increase in valve area

The mitral valve area was measured echocardiographically in 46 patients. The measurement was made between 1 to 3 days after the procedure. The mean mitral valve area increased to 1.6cm^2(SD 0.42). this measurement is significantly different from the pre-valvuloplasty measurement. Comparison of these values to the haemodynamically calculated values would be futile since only half of the patients underwent post procedure echocardiography.

3) Mitral regurgitation following balloon valvuloplasty:

Angiographic assessment of mitral regurgitation was performed before and after each procedure, unless a significant complication had occurred. The grading of regurgitation used is the subjective 1+ to 4+ system, with 1+ being mild and 4+ being severe. The grades recorded are those as reported at the joint catheterisation review meetings.

Table 6 below details the degree of angiographic mitral regurgitation. Mitral regurgitation post valvuloplasty was only assessed in those patients who were haemodynamically stable.

Table 6

Mitral regurgitation (Grade)	Pre-valvuloplasty (n = 120)	Post valvuloplasty n = 120
0	86 (72%)	61 (51%)
1+	30 (25%)	28 (23%)
2+	4 (3%)	18 (15%)
3+		8 (7%)
4+		3 (2%)
Not measured		2 (2%)

The degree of mitral regurgitation remained the same in 73 patients (61%), worsened by one grade in 25 patients, two grades in 12 patients (10%), three grades in 5 patients (4%) and four grades in 1 patient (1%). An improvement by one grade was noted in 6 patients (5%).

4) Comparing the two techniques

Table 7 details comparative data between the 2 types of balloon utilised at Groote Schuur hospital.

Table 7 **Comparison between the Inoue and Bifoil balloons**

	INOUE	BIFOIL	
Attempts	70	54	
Completed	67	53	
Successful	63(90%)	50(92%)	NS
Age	30.6(9.7)	30.5(10.6)	NS
MVA(pre)	0.98(0.27)	0.83(0.27)	p=0.06
MVA(post)	2.0(0.5)	2.1(0.6)	NS
LA/LV(pre)	16.0(6.0)	17.9(7.2)	NS
LA/LV(post)	5.6(2.7)	4.9(2.4)	NS
PA(pre)	42.1(16.2)	47.3(17.8)	NS
PA(post)	30.4(11.0)	33.9(15.8)	NS
MR(post)	3	8	NS
Early MVR	2	1	NS
Atrial perforation	3	0	NS
Ventricular perforation	0	2	NS
Deaths	0	2	NS

While the Inoue balloon group had a larger initial valve area, there are no differences between either group as regards success and significant complications.

5) Outcomes in selected groups

Eight pregnant patients with a mean age of 26 years underwent valvuloplasty. The outcome in five patients is known to have been successful. Mean duration of pregnancy was 27 weeks. (One additional patient was two months pregnant at the time of the procedure - unbeknown to the medical staff). Technical difficulties were experienced with manipulating the catheter in a 37 week pregnant patient - related to compression of the abdominal veins by the gravid uterus. Data relating to procedure duration are inadequately documented.

The mean valve area ($2.1cm^2$) achieved did not differ from the rest of the patients. One episode of false labour was documented. No other complications occurred in this group.

Fourteen patients had undergone a previous surgical commissurotomy. Their mean age was 33.6 years and mean valve area was $0.9(0.2)cm^2$. This increased to $2.1(0.5)cm^2$ after valvuloplasty. Only one patient had a significant procedure related complication.

Table 8 below details the differences between those patients with a good outcome and those with a poor outcome (serious adverse events and/or increase in valve area of less than 50%).

Table 8

	Good Outcome (n = 90)	**Bad** Outcome (n = 32)	
Age	30.3(9.1)	32.0(12.5)	NS
Sex	16 males	5 males	NS
Echo score	5	5	NS
Previous surgery	2	5	NS
Left atrial pressure	25.7(6.8)	26.6(7.8)	NS
Pulmonary pressure	43.2(15)	47.0(21.3)	NS
Dilating balloon	55 Inoue	13 Inoue	p = 0.046

While the comparison between the two groups ignores variables which cannot be retrieved retrospectively (such as number of dilatations per procedure), it is of interest that a poor outcome is more frequently associated with the use of a Bifoil balloon.

SECTION D: THE COMPLICATIONS OF BALLOON VALVULOPLASTY

1) Major Complications

The complications of balloon mitral valvuloplasty are shown in Table 9.

Table 9 **Major Complications**

Death	2(1.6%)
Severe Mitral Regurgitation	3(2.4%)
Pericardial Puncture	3(2.4%)
Ventricular Perforation	2(1.6%)
Hypotension post procedure	16(13%)
Stroke	1(0.8%)
Coronary embolus	2(1.6%)
Pulmonary embolus	1(0.8%)
Arterial embolus	1(0.8%)
Transient ischaemic attack	1(0.8%)
Deep vein thrombosis	3(2.4%)
Bacterial endocarditis	1(0.8%)
Late tamponade	1(0.8%)

70 patients (56%) had uneventful procedures and were discharged without any complication occurring. 38 major complications occurred in 35 procedures (28%). One patient had both a transient ischaemic attack and a deep vein thrombosis. One of the patients who was hypotensive post procedure also developed severe mitral regurgitation. The 'late' tamponade occurred in one of the patients who died.

Both deaths occurred several days following balloon valvuloplasty. A 32 year old female patient died three days following the procedure with unresolved pulmonary hypertension. The pathological features of established pulmonary hypertension were found at autopsy. A second patient aged 40 years died 14 days after the procedure during a pericardial aspiration. The patient had presented with a *Staphylococcus Aureus* pneumonia. The patient needed ventilatory support. The diagnosis of mitral stenosis was made at this time. An uneventful valvuloplasty procedure was performed and the patient improved. Six days following valvuloplasty the patient developed the signs of a septicaemic illness, including disseminated intravascular coagulopathy. *Staphyloccus Aureus* was cultured from the bloodstream. The patient developed a pericardial effusion and died during aspiration of a bloody exudate. Autopsy failed to reveal any evidence of iatrogenic cardiac injury.

Three patients had severe mitral regurgitation following the procedure. Two patients required mitral valve replacement within the first week following the procedure and one patient underwent valve replacement six months after valvuloplasty.

Two patients had left ventricular lacerations as a result of the tip of the Bifoil catheter straightening out with balloon inflation and tearing the ventricle. Both patients had uneventful surgical repairs of the ventricle. Both patients had echocardiographic documentation of an increase in mitral valve area.

Three patients had cardiac chamber (atrial) perforation at the time of the transseptal puncture. Two were recognised immediately and the procedure was stopped after contrast was shown to be entering the pericardial space. Both of these patients were observed in the intensive care

unit for twenty four hours and the procedure was then repeated. The third was not recognised until after valve dilatation had been performed and the patient became hypotensive, required emergency pericardial aspiration. This laceration was repaired surgically.

Two patients were noted to have ECG changes and moderate increases in cardiac enzymes following valvuloplasty procedures. One of these patients had a wall motion abnormality documented echocardiographically. Neither patient had chest pain. Both events were assumed to have been due to a coronary embolus at the time of procedure. Coronary angiography was not performed in these patients who did not experience any sequelae.

A pulmonary embolus was assumed to be responsible for a persistent decrease in oxygen saturation data post procedure in one patient.

An arterial embolus was diagnosed in a single patient who lost pedal pulses post procedure. The leg remained well perfused and embolectomy was not performed.

Two cerebral events were noted. In a single patient a dense left hemiparesis was noted in the ward 12 hours post procedure. A right parietal infarct was demonstrated by computerised tomography of the brain. The other patient developed a transient weakness affecting the left hand two days following the procedure.

One patient had a confirmed deep vein thrombosis diagnosed 14 days following the procedure. Deep vein thromboses were suspected (but not confirmed) in a further two patients who, following the procedure, developed a painful swollen leg.

Culture negative endocarditis was assumed to be responsible for the illness which resulted in the admission of a female patient one month after valvuloplasty. The original procedure had resulted in the creation of moderate mitral regurgitation which had not worsened the patient's symptoms. A large mass assumed to be a vegetation was seen in the patient's right ventricle. The illness had a 'septicaemic' character and was complicated by hypoxia and features compatible with septic pulmonary embolisation. The patient had severe tricuspid regurgitation. No organism was cultured. The patient improved and was submitted to mitral valve replacement and a tricuspid annuloplasty procedure. Large amounts of thrombus were found to be adhering to the tricuspid valve. Neither histological or bacteriological confirmation of endocarditis was obtained.

Hypotension following valvuloplasty was noted in 16 patients. The patients responded to intravenous crystalloid administration, although up to four litres of fluid were sometimes required. The complication followed 13 procedures using the bifoil balloon and followed three using the Inoue balloon. Hypotension following valvuloplasty occurred more frequently in the first two years that the technique was used; 9 out of 16 events occurred in 1988 and 1989. The immediate cause of the hypotension was not always apparent but may have related to the combined results of opiate analgesia, overnight starvation and the continued use of diuretics on the evening prior to the procedure. In addition vagal responses to removal of groin sheaths may have contributed to the hypotension. More recently, diuretic therapy has been routinely discontinued prior to the procedure. A separate sheath is not used with the Inoue balloon.

2) Minor Complications

Table 10 details the occurrence of minor complications. The difference between serious and minor complications is arbitrary and based on my perceptions of the time needed for the medical staff to 'sort out' the problem and its potential ability to cause death or severe morbidity.

Table 10 **Minor complications**

Complication	**Occurrence**
Vagal event	1 (1.6%)
Seizure	1 (1.6%)
Atrial arrhythmias	5 (4.0%)
Displaced guidewire	2 (3.2%)
Burst balloon	1 (1.6%)
Vomiting	1 (1.6%)
False labour	1 (1.6%)
Air in left atrium	1 (1.6%)
Minor bleeding	5 (4.0%)

These complications did not have any sequelae. The displaced wires resulted in inflation across the tricuspid valve in one patient and an inability to cross the atrial septum again in another. A brief seizure in one patient followed the administration of protamine sulphate.

The atrial arrhythmias consisted of one episode of atrial flutter, three episodes of atrial fibrillation and one 'supraventricular tachycardia'. Except for this event, which occurred during the procedure and necessitated electrical cardioversion, the other episodes were noted following the procedure. Two of these arrhythmic events required cardioversion.

SECTION E: SYMPTOMATIC IMPROVEMENT AND FOLLOW-UP

1) Symptomatic improvement following valvuloplasty

The improvement in effort tolerance following valvuloplasty is difficult to assess retrospectively. I have considered the symptoms as expressed at the first follow up visit as a better indication of symptoms following the procedure than the symptoms noted on discharge. Information is available in 102 patients. This information is summarised in Table 11.

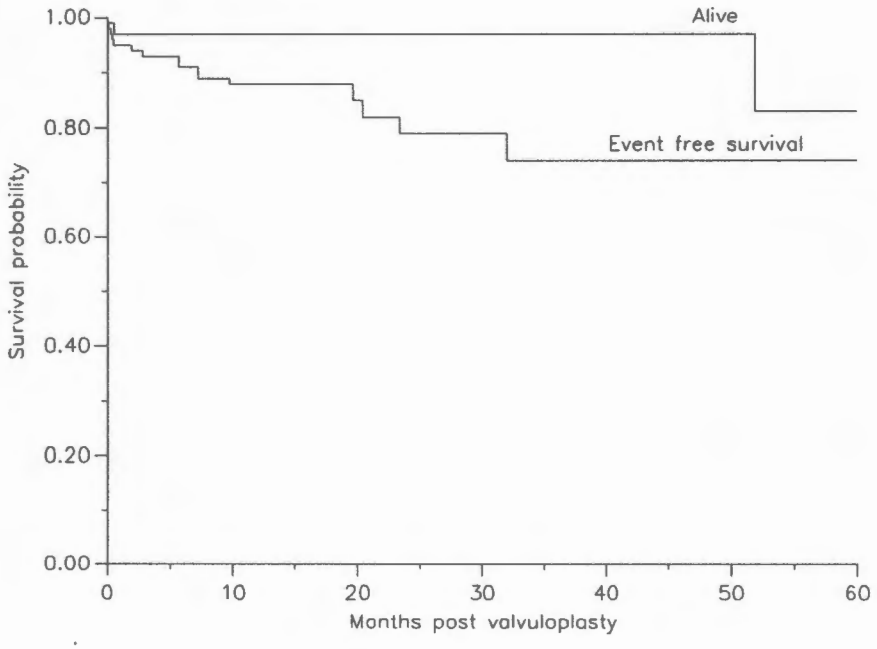
Table 11 **Symptoms post-valvuloplasty**

Lost to follow up	16 (13.5%)
Died	2 (1.6%)
Class I	59 (50%)
Class II	35 (30%)
Class III	4 (3%)
Class IV	2 (1.6%)
Worsened by 2 grades	1
Worsened by 1 grade	1
Remained unchanged	12
Improved by 1 grade	35
Improved by 2 grades	42
Improved by 3 grades	9

2) Survival following valvuloplasty

The figure below depicts the survival probabilities of the patient population following valvuloplasty. The method of Kaplan and Meier is used.[4] Only 1 late death occurred during the follow-up period and occurred in a patient with repeated cerebrovascular accidents post valve replacement who died 52 months post valvuloplasty. She had undergone mitral valve replacement and tricuspid annuloplasty 6 months after balloon valvuloplasty as a result of mitral regurgitation. The second curve depicts survival without mitral valve replacement, cerebrovascular accident, bacterial endocarditis or death.

Figure 2 Survival post valvuloplasty



The mean duration of follow-up was 16 months. (Range 0 to 56.6 months, median follow-up 10.7 months). Survival at 10.7 months was 97% (52 subjects at risk). Event free survival as defined above was 88% at 10.7 months. No data as to the incidence of echocardiographically determined restenosis are available. Two patients required a repeat balloon dilatation during the follow-up period (1.6%).

Seven patients underwent prosthetic mitral valve replacement during the follow-up period. The most common indication was persistent symptoms secondary to mitral regurgitation. One patient underwent open mitral valvotomy after the initial balloon dilatation could not be performed as a result of the inability to maintain the position of the guide wire in the left atrium.

REFERENCES FOR CHAPTER 8

1. Wilkins GT, Weyman AE, Abascal VM, Block PC, Palacios IF. Percutaneous balloon dilatation of the mitral valve: an analysis of echocardiographic variables related to outcome and mechanism of dilatation. Br Heart J 1988;60:299-308.

2. Cohen DJ, Kuntz RE, Gordon SPF, Piana RN, Safian RD, McKay RG, Baim DS, Grossman W, Diver DJ. Predictors of long-term outcome after percutaneous balloon mitral valvuloplasty. N Engl J Med 1992;327:1327-1325.

3. Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. Lancet 1986;1:307-310.

4. Altman DG. Practical Statistics for Medical Research. London:Chapman and Hall,1991:368-371

CHAPTER 9

DISCUSSION

The published South African experience with balloon mitral valvuloplasty is relatively small with just over 900 patients being reported in the literature.[1,2,3] The group at Groote Schuur hospital bears some resemblance to these patients in terms of demographic composition and pathology.

Table 1 below compares the Groote Schuur Hospital experience with the 2 largest Southern African series.

Table 1

	Groote Schuur n = 118	Baragwanath n = 235	Wentworth n = 530
Age(years)	30.7(10.2)	29(11)	30.5(12.5)
Balloon	I 70 B 54	I 145 D 90	I 430 B 100
Death	1.6%	0.8%	0.4%
Optimal result	76%	84%	83%

Note: Figures in parentheses denote 1 standard deviation. I - Inoue balloon, D - Double (Mansfield) technique, B - Bifoil/Trefoil balloon. Optimal result - mitral valve area ≥ 1.5cm^2 and < 2+ mitral regurgitation.

The Groote Schuur experience has involved younger patients than reported in the series originating in the first world.[4] Authors from India have reported a smaller series with a mean age of 19 years.[5]

Haemodynamic results

The haemodynamic results of balloon valvuloplasty at Groote Schuur do not differ markedly from other local centres.[1] The increases in valve area and decreases in transmitral gradients and pulmonary pressure are also equivalent to the results achieved by other major international investigators.[6]

Despite being a 'mixed' series, the haemodynamic results achieved are equivalent for the Inoue and the Bifoil/Trefoil balloons. This concurs with the results of the Johannesburg Hospital group.[2] Although reported in a series of older patients, Shaw et al reported similar equivalent haemodynamic results with the 2 balloon types in the United Kingdom.[7]

Complications

The incidence of 'major' complications (death, early valve replacement, thromboembolus and tamponade) in the Groote Schuur series is 8% which is similar to local[1] and international experience[8].

The incidence of severe mitral regurgitation and the need for early mitral valve replacement (2.4%) is comparable to the published results of other investigators who report figures

ranging from 2.4%[9] to 3.8%[8]. Mitral regurgitation increasing by 2 grades or more occurred in 15% of patients. This is similar to other patients from South Africa[1] and more than the 10% reported in the single operator series of Hung.[6]

The incidence of death (1.8%) is higher than the Baragwanath experience (0.8%)[1] or the Wentworth experience (0.4%)[3] but the absolute numbers of deaths in all series are small. (The differences fail to reach statistical significance). The deaths occurred in two extremely ill patients. Interestingly the mortality reported for closed mitral valvotomy at Groote Schuur Hospital for the period 1965 to 1977 was 2.97%.[10]

Ventricular perforation has not been reported with the Inoue balloon. The 1.6% incidence reported in the Groote Schuur hospital series occurred while the Trefoil or Bifoil devices were being used. This is similar to the experience of Shaw et al in Britain.[7] Ventricular perforation is also documented by authors using the Mansfield technique.[11] On the other hand, the occurrence of pericardial tamponade secondary to the transseptal puncture is common to all 'antegrade' procedures and the incidence in the Groote Schuur group (2.4%) is similar to the experience of other authors.[12] Only one of the three patients needed surgical repair of an atrial tear after emergency pericardial aspiration.

Embolic events (cerebral, coronary and arterial) occurred in 4.2% of the series. This is higher than the incidence reported at Baragwanath hospital (1%)[1] and in the large experience of Hung (2.5%)[6]. The absolute numbers of patients in whom these events occurred is small and differences in definition of events may account for these differences. Transoesophageal echocardiography was not used in the Groote Schuur hospital patients undergoing balloon

valvuloplasty. The incidence of embolic events at Baragwanath hospital was noted to drop after the introduction of routine transoesophageal echocardiography prior to valvuloplasty. (No embolic events were documented in the 140 patients who underwent transoesophageal echocardiography prior to valvuloplasty).[1]

The description of hypotension following valvuloplasty has not been reported in the literature. 13% of the Groote Schuur patients were significantly hypotensive. The possible reasons for this complication have been discussed in the results section (Chapter 8).

Technique

The perception that the Inoue balloon is easier to use as well as the fear of ventricular perforation with the Bifoil devices, have resulted in the Inoue balloon being favoured at Groote Schuur hospital. For similar reasons the Inoue balloon is favoured by the Johannesburg Hospital group[2] and the Wentworth group.[3] At Groote Schuur Hospital the use of the Bifoil catheter was statistically associated with a 'bad' outcome.

Special Groups

The good results of balloon valvuloplasty in pregnant patients at Groote Schuur are in keeping with those reported by Patel et al from Wentworth.[13] The efficacy of the procedure in patients who had undergone previous surgical commissurotomy is in keeping with the published literature.[14]

Symptomatic Improvement

92% of the group were in NYHA class I or II on first follow up visit. These results are equivalent to other local[1] and international experience.[6] 84% of patients described symptoms consistent with an improvement of at least one NYHA class.

Although survival data are available in the Groote Schuur hospital series up to 56.6 months post procedure, the median follow-up interval is 10.7 months. The 88% event free survival reported is equivalent to the results reported by Vahanian et al.[8] Total survival at 10.7months was 97%. The survival data are similar to those reported by Hung.[6]

Conclusion

The initial experience with balloon valvuloplasty at Groote Schuur confirms that the procedure can be learnt and applied with equivalent success to other centres. The position of balloon valvuloplasty as a replacement for closed mitral valvotomy is still to be established particularly in the developing world. Paradoxically while an expensive technique is being learnt, the 'time-out' for trainee surgeons may result in the training of surgeons who cannot reliably perform the closed procedure.

With increasing costs and the reallocation of health resources the use of expensive technology will become harder to justify. An alternative to the expensive Inoue balloon (which should not be resterilized[7]) will be needed. Balloon devices such as the Bifoil type which are more difficult and potentially more dangerous to use cannot be considered as a replacement.

REFERENCES FOR CHAPTER 9

1. Röthlisberger C,Essop MR,Skudicky D,Skoularigis J,Wisenbaugh T,Sareli P. Results of percutaneous balloon mitral valvotomy in young adults. Am J Cardiol 1993;72:73-77

2. Manga P, Landless P, Gebka M. Comparative results of percutaneous balloon mitral valvuloplasty using the Trefoil/Bifoil and Inoue balloon techniques. Int J Cardiol 1994;43:21-25

3. Patel JJ, Munclinger MJ, Pillay RJ, Mitha AS, Gouws E. Outcome analysis of 70 months experience with percutaneous mitral valvuloplasty. Cardiovasc J South Afr 1994;5(5 Suppl):2-6 (Abstract)

4. Herrmann HC, Ramaswamy K, Isner JM, Feldman TE, Carroll JD,Pichard AD, Bashore TM, Dorros G, Massumi GA, Sundram P, Tobis JM, Feldman RC, Ramee S. Factors influencing immediate results, complications, and short-term follow-up status after Inoue balloon mitral valvotomy: A North American multicenter study. Am Heart J 1992;124:160-166

5. Arora R, Nair M, Kalra GS, Nigam M, Khalilula M. Immediate and long-term results of balloon and surgical closed mitral valvotomy: A randomised comparative study. Am Heart J 1993;125:1091-1094

6. Hung JS, Chern MS, Wu JJ, Fu M, Yeh KH, Wu YC, Cherng WJ, Chua S, Lee CB. Short- and long-term results of catheter balloon percutaneous transvenous mitral commissurotomy. Am J Cardiol 1991;67:854-862

7. Shaw TRD, Turnbull CM, Currie P, Flapan AD, Pringle S, Lee BC. A comparison of cylindrical and Inoue balloon techniques for mitral valvotomy in patients in the United Kingdom. Br Heart J 1994;72:486-491

8. Vahanian A, Michel PL, Cormier B, Ghanem G, Vitoux B, Maroni JP, Cazaux P, Acar J. Immediate and mid-term results of percutaneous mitral commissurotomy. Eur Heart J 1991;12(suppl B):84-89

9. Hernandez R, Macaya C, Bañuelos C, Alfonso F, Goicolea J, Iñiguez A, Fernandez-Ortiz A, Castillo J, Aragoncillo P, Aguado MG, Zarco P. Predictors, mechanisms and outcome of severe mitral regurgitation complicating percutaneous mitral valvotomy with the Inoue balloon. Am J Cardiol 1992;70:1169-1174

10. Commerford PJ, Hastie T, Beck W. Closed mitral valvotomy: actuarial analysis of results in 654 patients over 12 years and analysis of preoperative predictors of long-term survival. Ann Thorac Surg 1982;33:473-479

11. Herrmann HC, Kleaveland JP, Hill JA, Cowley MJ, Margolis JR, Nocero MA, Zalewski A, Pepine CJ, for the M-Heart group. The M -Heart percutaneous balloon mitral valvuloplasty registry: Initial results and early follow-up. J Am Coll Cardiol

1990;15:1221-1226

12. Nobuyoshi M, Hamasaki N, Kimura T, Nosaka H, Yokoi H, Yasumoto H, Horiuchi H, Nakashima H, Shindo T, Mori T, Miyamoto AT, Inoue K. Indications, complications, and short-term outcome of percutaneous transvenous mitral commisurotomy. Circulation 1989;80:782-792

13. Patel JJ, Mitha AS, Hassen F, Patel N, Naidu R, Chetty S, Pillay R. Percutaneous balloon mitral valvotomy in pregnant patients with tight pliable mitral stenosis. Am Heart J 1993;125:1106-1109.

14. Davidson CJ, Bashore TM, Mickel M, Davis K. Balloon mitral commissurotomy after previous surgical commissurotomy. Circulation 1992;86:91-99.