Predicting the performance of mitral prostheses implanted in children under 5 years of age

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Abstract

Background: Mitral valve replacement (MVR) is occasionally indicated in infants and young children, necessitating the use of small prostheses. The performance of these small valves during somatic growth of the patient can lead to patient-prosthesis mismatch. This study examines performance of these valves over time to establish predicted performance and timing of replacement.

Methods: Records were reviewed of all patients under 5 years of age who underwent small mechanical MVR between 1988 and 2004 (n=24). Valve sizes were between 17 and 23 mm (Bileaflet 91.6%, Tilting Disc 8.3%) with a median size of 19 mm. Mean age of patients was 1.4 ± 1.3 years with a mean weight of 7.8 ± 3.4 kg.

Results: Early deaths (n=5, 20.8%) were excluded. There were two late deaths and five patients required redo-MVR: four for outgrowth and one for acute thrombosis at 3 months. Age at redo for outgrowth was 8.6 ± 6.6 years with mean body weight of 22.5 ± 17.5 kg. Mean time between original operation and redo was 8.6 ± 6.1 years in these four patients. Follow-up was a median of 7.5 years (range 0.1—15.7 years). Overall freedom from death or valve replacement was 82.6 ± 9.1% at 5 years and 75.7 ± 10.6% at 10 years. The performance of the original prostheses showed a peak blood flow velocity across the valves of 1.3 ± 0.6 m/s at 5 years and 2.2 ± 0.5 m/s at 10 years. Seventy-five percent of the survivors still have their original valve at a mean of 8.1 ± 4.4 years postoperative with New York Heart Association status of I or II. Actuarial curves suggest that gradients across the valves reach a peak of >10 mmHg at a mean between 6.5 and 7 years postoperative.

Conclusion: MVR in children under 5 years carries a high mortality. Nevertheless, small mechanical MVR perform remarkably well in young children with durable haemodynamics despite growth of the patients well beyond more than double the initial bodyweight. Valves can be expected to last over 8 years before requiring re-replacement.

Keywords: Mitral valve; Replacement; Children

1. Introduction

Mitral valve replacement (MVR) is occasionally indicated in infants and young children, necessitating the use of small prostheses. Valve repair is the procedure of choice and is generally feasible in most pathologies [1,2]. However, complex lesions such as congenital stenosis with a parachute valve may necessitate replacement as a primary procedure. Nevertheless, the majority of MVRs are performed following previous repairs. Replacement in this age group has a high-operative mortality ranging from 10 to 36% [3–5].

A mechanical valve is the prosthesis of choice in the mitral position in children because biological valves have a tendency to degenerate quicker in children compared to adults [6]. Also, availability of tissue valves in small sizes is extremely limited and the protrusion of the commissural pillars of these valves into the ventricular cavity may further limit suitability. Insertion of a mitral valve in smaller children is more difficult than in adults. Access may be a problem, for example, when the left atrium may not have had time to dilate in an infant to provide working space and this may necessitate a trans-septal approach via the right atrium. Supra-annular positioning of the prosthesis may be unavoidable and risk impingement of the sewing ring on the left ventricular outflow tract (LVOT) creating subaortic stenosis. Anticoagulation during follow-up is cumbersome [7,8]. In young neonates warfarin is contraindicated and unfractionated heparin is used until at least 3 months of age. Another concern is compliance with warfarin and repeated blood sampling in small children.

Furthermore, the performance of these small valves during somatic growth of the patient can lead to patient-prosthesis...
mismatch. This may result in haemodynamic dysfunction with consequent redo-MVR with a larger prosthesis [9]. This study aims to address the issue of patient-prosthetic mismatch of mechanical MVR in children as well as complications over time to establish predicted performance and timing of replacement.

2. Patients and methods

Children under 5 years of age who underwent small mechanical MVR between 1988 and 2004 (n = 24) were included in the study. Data were collected from computerised databases (HeartSuite®, Systeria Ltd, Glasgow, UK) and patient notes retrospectively. Early deaths (n = 5, 20.8%) were excluded from the analysis. MVR with prosthesis between 17 and 23 mm (Bileaflet 91.6%, Tilting Disc 8.3%) were included in the study. The median size was 19 mm (range: 17–23 mm). Mean age of patients was 1.4 ± 1.3 years with a mean weight of 7.8 ± 3.4 kg.

The primary diagnoses leading to mitral valve dysfunction were atrioventricular canal defect (n = 11) and mitral valve dysplasia (n = 13). There was one patient with Shone’s complex. Sixteen patients had previously undergone mitral valve repair surgery. The haemodynamic lesion responsible for the MVR was mitral regurgitation in 75% (n = 18), mitral stenosis in 16.6% (n = 4), and combined mitral stenosis and regurgitation in 8.3% (n = 2) of the patients. Two children had concomitant procedures, one had closure of ventricular septal defect and the other underwent mechanical aortic valve replacement plus Konno aorto-ventriculoplasty. Both patients were alive at 3.6 and 8 years of follow-up, respectively. The latter patient required a redo-MVR 8 years after the initial operation. Table 1 shows patient characteristics of 30-day survivors at initial mitral valve replacement.

Table 1

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (months)</th>
<th>Weight (kg)</th>
<th>Diagnosis</th>
<th>Primary haemodynamic pathology</th>
<th>Previous operations</th>
<th>z-score</th>
<th>Prosthetic valve size (mm)</th>
<th>BSA (m²)</th>
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<td>14.2</td>
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<td>17</td>
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</table>

Note that the BSA was calculated by using the formula of DuBois and DuBois (Arch Intern Medicine 1916;17:863–71). Abbreviations: BSA, body surface area; cAVSD, complete atrioventricular septal defect; CoA, coarctation of aorta; kg, kilograms; LAVV, left atrioventricular valve; LVOT, left ventricular outflow tract; MR, mitral regurgitation; MS, mitral stenosis; MV, mitral valve; pAVSD, partial atrioventricular septal defect; SubAS, subvalvular aortic stenosis; z-score, represents mitral annulus size at time of mitral valve replacement.

The operation was performed via a median sternotomy in all patients. Cardiopulmonary bypass with moderate hypothermia and crystalloid cardioplegic arrest was used. Deep hypothermic circulatory arrest was employed in small infants, only if deemed necessary to gain adequate surgical view. The posterior leaflet and subvalvar apparatus of the MV was preserved wherever possible. In the small annulus, in the one patient with Shone’s syndrome, all valve tissue was excised to maximise the mitral inflow. Also, in the patient with endocarditis, all valve tissue was excised. The mechanical valve was inserted within the mitral annulus except in one patient, in whom the valve was sutured in the supra-annular position because the smallest available prosthesis would not fit in the annulus. Interrupted pledged evertting ethibond sutures were used. The valves used were according to surgeons’ choice. Twenty-one (91.6%) St. Jude bileaflet prostheses (St. Jude Medical Inc, Minneapolis, USA), two (8.3%) Bjork–Shiley valves (Shiley Inc, CA, USA) and one (4.1%) ATS bileaflet prosthesis (ATS Medical Inc, Minneapolis, USA) were used.

Postoperatively, all patients received oral warfarin to maintain an INR between 3.0 and 4.0. All patients were seen at least every 12 months. Transthoracic echocardiography was performed immediately before surgery, in the postoperative period and at each follow-up visit. Follow-up was a median of 7.5 years (range 0.1–15.7 years).

3. Statistical analysis

The data are expressed as frequencies, mean ± SD or medians with ranges, as appropriate. Time to late mortality, redo-MVR and valve-related events were examined with Kaplan–Meier curves. A Cox proportional hazards model was used. Multivariate analyses were performed. A p-value <0.05
was considered statistically significant. SPSS software (6.1.2 for Windows, SSPS Inc, Chicago, IL, USA) was used.

4. Results

Insertion of a permanent pacemaker was needed in three patients after initial MVR. The postoperative course was complicated by a retroperitoneal haematoma in one patient and one patient required laparotomy and small bowel resection for small bowel infarction. The latter patient died at 8 months follow-up. This patient had Down’s syndrome and also underwent previous atrioventricular septal defect (AVSD) repair. The only other late mortality was a patient who had a previous aortic coarctation/mitral valve repair, then MVR followed by a redo-MVR. Six months following this, the child had a pulmonary venous dilatation for isolated pulmonary vein stenosis but succumbed 6 months later due to heart failure.

The performance of the original prostheses showed a peak blood flow velocity ($V_{\text{max}}$) across the valves of $1.5 \pm 0.6$ m/s at 5 years and $2.2 \pm 0.5$ m/s at 10 years for the entire study. Seventy-five percent of the survivors still had their original valve at a mean of $8.1 \pm 4.4$ years postoperative with NYHA status of I or II. Actuarial curves suggest that gradients across the valves reach a peak of $>10$ mmHg at a mean of between 6.5 and 7 years postoperative. Five patients required redo-MVR: four for acquired mitral stenoses secondary to somatic growth and one for acute thrombosis due to noncompliance with anticoagulation at 3 months follow-up. Successful redo-MVR with a similar prosthesis sized 21, 25, 21 and 21 mm was performed in the four patients where the size of the original prosthesis was 19, 21, 17 and 19 mm, respectively. Thus, at redo-MVR, a larger prosthesis was implanted in every case except for the early replacement for acute thrombosis where the same size was valve used. Constriction was, therefore, not a problem and there was no recorded incidence of significant pannus ingrowth. Age at redo for outgrowth was $8.6 \pm 6.6$ years with mean body weight of $22.5 \pm 17.5$ kg. Mean time between original operation and redo was $8.6 \pm 6.1$ years in these four patients. At the time of redo-MVR, $V_{\text{max}}$ was 2.8, 2.6, 3.2 and 2.7 m/s in these four patients. Redo-MVR was well tolerated with no early deaths, valve thrombosis or structural valve failure. Overall freedom from death or valve replacement was $82.6 \pm 9.1\%$ at 5 years and $75.7 \pm 10.6\%$ at 10 years (Fig. 1).

Four patients suffered from valve-related events during follow-up. One patient developed prosthetic valve endocarditis (pseudomonas aetiology, died), one developed anticoagulation-related minor rectal bleeding which settled with correction of coagulation (admitted but not transfused, no underlying bowel pathology), one developed pancreatitis, possibly warfarin-induced (settled with conservative treatment) and one developed prosthetic valve thrombosis (required redo at 3 months). There were no significant paravalvular leaks. Kaplan–Meier 10-year freedom from any valve-related event was $78.0 \pm 8.9\%$ (Fig. 2). Mapping of the weight on centile charts shows that children do relatively well and grow along their centile lines but remain smaller than average (mean 24th centile $\pm 25$ at 5 years and 32nd centile $\pm 30$ at 10 years). In relation to healthy controls, 91% of early survivors were NYHA I at 5 years (remaining two patients in NYHA II).

5. Discussion

The occurrence of uncorrectable mitral regurgitation after atrioventricular canal repair is the commonest cause of mitral valve replacement in children [10–12]. The evaluation of small children for MVR includes consideration of in-hospital mortality, late attrition associated with warfarin use or residual heart disease and the development of patient-prosthesis mismatch due to somatic growth leading to
subsequent MVR [4,5,13]. Therefore, morbidity and mortality associated with redo-MVR should be included in the decision-making at the time of initial MVR in children, especially under 5 years of age.

MVR in small children is a very challenging procedure. The frequently associated complex anatomy and pathology makes it high-risk especially in an unstable patient. It is well established that early mortality after MVR may be as high as >30% in patients under 5 years of age [2,4]. Early mortality rates of 17–35% are reported for this age group [2,3,14]. It has been reported that MVR under 5 years of age is becoming increasingly safer which is probably a result of overall better surgery, perioperative care of the paediatric surgical patients and improvements in cardiopulmonary bypass [5].

A bileaflet mechanical valve is the prosthesis of choice in the mitral position in children because of its low profile, excellent haemodynamics and durability versus a biological prosthesis [6]. Despite the need for anticoagulation, acceptable long-term results of mechanical MVR are reported in children [15–18]. Some authorities believe that contemporary low-profile mechanical valves are less thrombogenic and hence no anticoagulant needs to be prescribed [19]. The use of such strategy in one case series leads to a greater incidence of thromboembolic events [20]. Thus, we and others [21] have shown that routine warfarinisation of children after mechanical MVR is safe and well tolerated. Another theoretical option is the so-called Ross II operation using the upturned pulmonary autograft in the mitral position. This is a complex and untried procedure in small children but does not require anticoagulation and may be a viable alternative where warfarin cannot be used.

Structural valve failure with the use of present day mechanical prostheses is no longer a problem in small children. However, somatic growth may lead to patient-prosthesis mismatch in this age group. This may result in restriction of leaflet mobility, conduction defects and progressive LV dysfunction. The problem may be exacerbated by trying to insert a larger prosthesis at the initial MVR to avoid redo later, which may also result in circumflex artery injury. That the mitral annulus grows despite valve implantation, allowing the insertion of a larger prosthesis at subsequent MVRs is well known [13].

The incidence and timing of redo-MVR in children depends on the age at the initial MVR, the prosthesis used and the length of follow-up and thus incidence varies widely amongst studies ranging from 0 to 60% [5,13,15–17,22]. With the use of Starr–Edwards valve, it has been shown that body weight increase of 2.5 times at the time of initial MVR is a predictor of redo-MVR ([13], published in 1978). The haemodynamic performance of the valves used in our study has been good and the valve-related complication rate low. The rate of thromboembolism, PVE and bleeding complications compares with previously published reports [14,23,24]. The 10-year freedom from late death and redo-MVR is considerably better than previously reported (50–75%) [2,14,23,24]. However, caution should be used in making comparisons due to the heterogeneous nature of the studies.

In our study four study patients had redo-MVR for somatic growth. The decision for redo-MVR was based on a combination of clinical symptoms and an increased trans-

valvar gradient. Redo operation was carried out as an elective procedure in all the four patients. The indications of redo-MVR in these patients were evidence of symptomatic mitral stenosis (i.e. breathlessness, cough, exercise limitation) and/or echocardiographic finding of $V_{max}$ $>2.2$ m/s. Urgent redo-MVR was performed in the child who developed thrombosis on the valve after initial MVR. In all these patients it was possible to insert a larger prosthesis than the first time prophylaxis, a finding reported by other authors as well [4,9]. Furthermore, in all cases a bileaflet valve was inserted.

The results of this paper must be interpreted in the light of its limitations. The study is retrospective and the number of patients is relatively small. However, MVR in children <5 years of age is uncommon and therefore case series from single centres are useful to expand our understanding on the subject.

Our data agree with the findings of Caldarone et al. [18] who showed that the long-term outlook of low-risk children who undergo MVR is quite favourable despite the potential for requiring a second (and possibly third) MVR. MVR in children under 5 years carries a high mortality. Nevertheless, small mechanical MVR perform remarkably well in young children with durable haemodynamics despite growth of the patients well beyond more than double the initial body-weight. Valves can be expected to last over 8 years before requiring re-replacement. The ability to predict outcome after MVR in children aged <5 years may be useful in choosing between MVR and alternative therapeutic strategies.

References

Appendix A. Conference discussion

Dr J. Fragata (Lisbon, Portugal): Because the standard deviation for patient weight was so large, could you give us any idea what the proportion of valves were on the 'small size' group, I mean sizes 17 and 19, and how much did that number actually contribute to all the population? Because instead, if sizes 21 and 23 were more prevalent in your population, then one would expect those to be reoperated later. In summary, I would like to know how much a 17 or 21 and 23 were more prevalent in your population, one would then expect that number actually contribute to all the population? Because instead, if sizes 17 and one was 21.

Dr Vohra: The four valves that we redid, two of them were 19 size, one of them was 17 size and one was 21.

Dr Fragata: So still the great majority will be 20, 21 and 23?

Dr Vohra: The great majority of the patients received a size of 19 mm. Only one patient had a size of 23 mm at time of insertion of original prosthesis. Mostly they were 17 or 19. 21 size was again two or three patients, not more than that.

Dr Fragata: So the important message is that a 17 or 19 will easily last 8 years?

Dr Vohra: Yes, definitely.

Dr C. Brizard (Melbourne, Australia): Your paper is a nice demonstration of what has become a clear knowledge from the literature: the mitral valve replacement in itself, whatever the size of the prosthesis in children, is a low-risk factor for death. The risk factor for death is the mismatch between the annulus of the recipient and the size of the prosthesis you implant. In order to further demonstrate that, could you tell us about the cause of death of the four patients that have been excluded, the early deaths, and whether the late deaths received a prosthesis implanted in the supra-annular position?

Dr Vohra: None of the late deaths were implanted in the supra-annular position. And we did not, as part of the design of the study, look at the early years?

Dr Fragata: The four valves we replaced. The size that we used were 21 in three patients and 25 in one patient. So from 17, we went to 21 in one patient and from 19 we went to 21 in two patients. And the child who had a size of 21 inserted at the time of original operation had 25-mm size redo prosthesis inserted.

In answer to your second question, again, we would repair the mitral valve as our procedure of choice for all mitral regurgitations.

Dr G. Stellin (Padova, Italy): Obviously in the paediatric population, the mitral valve replacement is a major concern. And it is not clear from your presentation in how many cases you did attempt, indeed, a mitral valve reconstruction, and in which cases you couldn’t really obtain a mitral reconstruction, and you couldn’t reconstruct the valve and you had to replace it.

Dr Vohra: Probably I won’t have the full answer to your question. But as you can see, 9 of our patients had a previous AVSD repair, and 11 patients had a prior mitral valve repair. But at the time of surgery, every time we looked at the valve, there was an intention to repair the valve first. But if it was thought that it is not repairable, then, on table, it was decided to carry on, to replace the valve instead of continue to try to repair the valve.

Dr M. Wojtalik (Pozen, Poland): I would expect the reoperation at the time of the fast growth in the age of 12 to 14. Did you analyse this factor?

And the second question: in some children one can implant really a big size valve. And the size of future adults, the ex-pediatric patient, depends on the parents. Did you compare the size of the valve implanted to the size of parents or the future adult valve size?

Dr Vohra: We did analyse the redo rate. We did a univariate analysis first, and it did show that small size and age less than 1 year of age, impaired LV, prior cardiovascular procedures, did have effect on the outcome. But on multivariate analysis, none of these factors had any effect on the outcome or the need for a redo operation 8 years or 10 years afterwards. So we did not find any. This may be due to small number of patients from a single centre.

The second question is the need to insert a larger prosthesis at the time of original operation. One has to be really cautious. There is always a tendency to think that this patient will grow up and the mitral annulus will enlarge. And to avoid patient-prosthesis mismatch, one tends to put in a larger valve at the time of first operation.

It has been shown very clearly that if this is done, there are more chances of haemodynamic compromise after the first operation and the outcome is worse. There is a chance of circumflex artery injury and valve leaflet dysfunction. LV function gets impaired. And there are also chances of narrowing of the left ventricular outflow tract obstruction.

We did not look at the parental size and did not predict what the mitral annulus size will be once they grow up to 15 or 20 years of age. But this is a valid point. One has to be really careful not to oversize the valve at the time of initial surgery.

It has been very clearly shown by Caldarone in Circulation that a second AVR is a perfectly safe operation. It’s probably safer than the first. The child is bigger, it’s less a risk. And the first valve lasts you 8 to 10 years anyway, so what’s the point in trying to fit in a larger valve and get a worse outcome early after the original mitral valve replacement?

Dr Wojtalik: Well, I didn’t mean to force to put in a bigger size, but it just sometimes happens that in a child the age of 2 years, you might implant a valve 24. And if the parents are small size, the 24 might be suitable for the adult, too, in the future.

Dr Vohra: Sure, it may be suitable, but it should not be a routine to try and put in a bigger valve size. It may suit one patient. But generally if you look at a cohort of patients, it has been clearly shown that one can end up in trouble trying to do that.

Dr J. Mono (Southampton, United Kingdom): Just a brief point. I take what you’ve just said, that you don’t want to force in a big valve, but in a very small child it is useful to get in as big a valve as possible. And one trick I have used is to use an aortic valve upside down, so you’re putting it just back, and you can usually get in one size bigger.

Dr Vohra: Thank you for your suggestion.