Mitral Valve Repair in Children Below Age 10 Years: Trouble or Success?

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Background. **Mitral valve (MV) repair in children is challenging because of the broad spectrum of lesions and anticipated patient growth. The purpose of the study was to evaluate the outcome of MV repair in children below 10 years of age.**

Methods. **We reviewed all MV repair procedures performed in children below 10 years of age. Endpoints of the study were survival after MV repair and cumulative incidence of reoperation.**

Results. **MV repair was performed in 40 patients with congenital MV disease (MVD) and in 10 patients with acquired MVD. Median age at time of repair for congenital MVD was 1.2 years (range, 14 days to 9.8 years) and for acquired MVD 1.9 years (range, 10 days to 9.9 years). Indication for MV repair was mitral regurgitation in 31 congenital MVD patients (77.5%) and in all acquired MVD patients. In patients with congenital MVD operative mortality was 5% and late mortality was 10%. No deaths occurred in patients with acquired MVD. Patients with congenital mitral regurgitation showed a better, yet not significant, 6-year survival than patients with congenital mitral stenosis (85.3% ± 8.2% vs 60% ± 18.2%, $P = .1$). In patients with congenital MVD cumulative incidence of reoperation at 6 years was 38.6% ± 8.3%.**

Conclusions. **In children below 10 years of age, MV repair is an effective treatment option for MVD. However it often just delays the time to valve replacement.**

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Mitral valve disease (MVD) is rare in children, and the incidence of congenital MVD is about 0.5%.[1] Congenital MVD is often associated with other cardiac malformations such as ventricular septal defect (17%) and subaortic stenosis (12%).[2,3] Because any component of the valvar apparatus may be affected, resulting in stenosis, regurgitation, or combined lesions, decisions on the best surgical strategy are apt to be particularly challenging. However primary reconstruction of the valve seems preferable even though it may simply delay, rather than prevent, mitral valve (MV) replacement as a child grows.[4]

In adults a well-established classification of valvar pathology, addressing etiologic and morphologic aspects of MVD, has led to standardized surgical techniques that show excellent results up to 2 decades later.[5-7] Valvar pathology in the setting of congenital MVD is typically more complex, precluding a standardized approach to MV repair. However some reports do indicate favorable long-term results after MV repair, describing long-term survival rates of 86% to 93% and 79% freedom from reoperation after 10 and 20 years, respectively.[8,9] On the other hand children subjected to MV replacement face a high mortality (14%-24%) and greater long-term need for permanent pacemaker implantation (15%) because of atrioventricular block.[10,11] Other serious disadvantages of MV replacement in a growing child include patient–prosthesis mismatch, requiring serial valve replacements, and obligatory lifelong anticoagulation therapy.[12,13] The aim of our study was to evaluate the durability of MV repair in children below 10 years of age and to show if it is an advisable strategy for these patients.

Patients and Methods

Study Design and Study Population

In this retrospective study we reviewed all patients up to 10 years old undergoing MV repair between February 1975 and December 2017 at the German Heart Center Munich. Patients with complete or partial atrioventricular septal defects and single-ventricle physiology were excluded from analysis. Data for the present study were collected by reviewing medical records, operative notes, and telephone interviews with the referring pediatric cardiologist. Depending on the etiology and underlying diagnosis, patients were divided into 2 groups: patients

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with congenital MVD and patients with acquired MVD, with each group of patients with either prevalent mitral regurgitation (MR) or mitral stenosis (MS).

Follow-up was conducted in our outpatient clinic or by the referring pediatric cardiologists. Operative mortality was defined as described by the Society of Thoracic Surgeons.[14] Endpoints of the study were survival after MV repair and cumulative incidence of reoperation. This study was approved by the Institutional Review Board of the Technical University Munich, and the need for patient consent was waived.

Evaluation of the MV

Evaluation of the MV was performed by transthoracic echocardiography at baseline and discharge. Transthoracic echocardiography was performed according to the American Society of Echocardiography guidelines.[15,16] MR was graded as mild, moderate, moderate to severe, or severe based on qualitative parameters (color-flow jet area and mitral inflow) and semiquantitative or quantitative measures (vena contracta width and regurgitant fraction).[15] MS was graded on the basis of the mean pressure gradient across the valve as mild, <5 mmHg; moderate, ≥5 mmHg but ≤10 mmHg; and severe, >10 mmHg.[16] Left ventricular linear dimensions were assessed in the parasternal long-axis view, using 2-dimensional–targeted M-mode echocardiography.[17] Left ventricular volume and left ventricular ejection fraction were measured in the apical 2-chamber view, using the 2-dimensional modified Simpson's rule.[17] Classification of the predominant MV lesion was done according to Carpentier.[7,18]

Surgical Techniques

All patients were operated through a median sternotomy, with cardiopulmonary bypass and mild hypothermia at 32°C core temperature. Exposure of the MV was gained through a left atriotomy or by incision of the interatrial septum. Injection of saline solution into the left ventricle and careful examination with hooks were performed to assess the coaptation and mobility of the valve leaflets. Interrupted stitches or continuous locked sutures were used for direct closure of leaflet defects or clefts. Different patch materials, namely glutaraldehyde (0.2%)-treated autologous pericardium and decellularized bovine pericardium (CardioCel; Admedus Regen Pty Ltd, Perth, WA, Australia), were applied for leaflet augmentation. Gore-Tex neo-chordae (W.L. Gore & Assoc Inc, Newark, DE) were used for chordal replacement. Assorted annuloplasty rings, ranging from rigid complete to semirigid partial design, were also implanted.

Statistical Analysis

Data were analyzed using SPSS, version 25.0 for Windows (IBM Corp, Armonk, NY) and R (version 3.5.2; R Foundation for Statistical Computing, Vienna, Austria). All continuous variables were non-normally distributed and reported as median with range of minimum and maximum. For time-to-event analysis the mean with SD was reported for follow-up time. Categorical variables were expressed as numbers and percentages. The 2-tailed χ^2 test was used for analysis of categorical data, whereas continuous variables were compared using the Mann-Whitney U test. Kaplan-Meier analysis was applied to calculate estimated survival. The log-rank test was used to compare the survival of patients with congenital MR and MS. Competing risk analysis was used to calculate the cumulative incidence of reoperation. Estimated incidences of survival and reoperation were described at the mean follow-up time. Thus the Kaplan-Meier plots of survival and the competing risk analysis for death and reoperation were truncated at the mean follow-up time. An event-specific proportional risk model was used to calculate the hazard ratio for reoperation. Statistical significance was set at $P < .05$.

Results

Patients With Congenital MVD

Congenital MVD was present in 40 patients with a median age and weight at surgery of 1.2 years (range, 14 days to 9.8 years) and 8.2 kg (range, 2.9-41.9), respectively. Nineteen patients (47.5%) were below 1 year of age. Thirty-one patients (77.5%) showed a prevalent MR, whereas a significant MS was seen in only 9 patients (22.5%). The main reasons for MR were either leaflet clefts in 17 patients (42.5%) or leaflet restrictions due to shortened chordae in 5 patients (12.5%). MS was due to a supravalvar ring (n = 3), papillary muscle–commissural fusion (n = 3), or a parachute valve (n = 3). Further characteristics at baseline and details of MV pathologies are depicted in Tables 1 and 2. Surgical procedures are specified in Table 3.

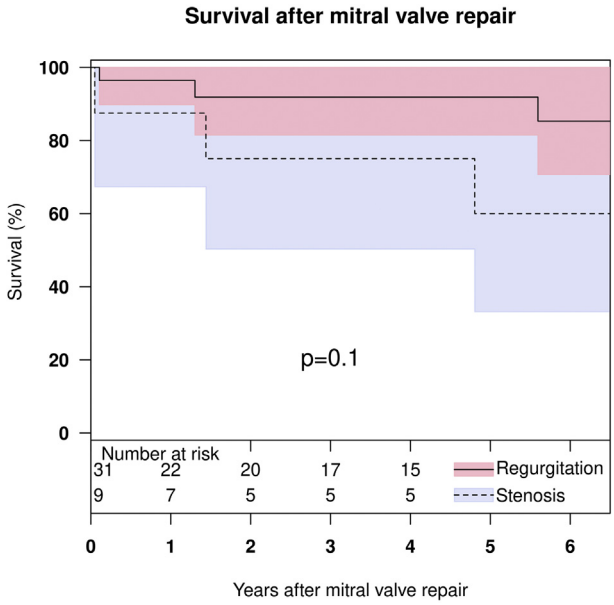

Figure 1. Kaplan-Meier plots of survival of patients with mitral regurgitation (red) or mitral stenosis (blue).

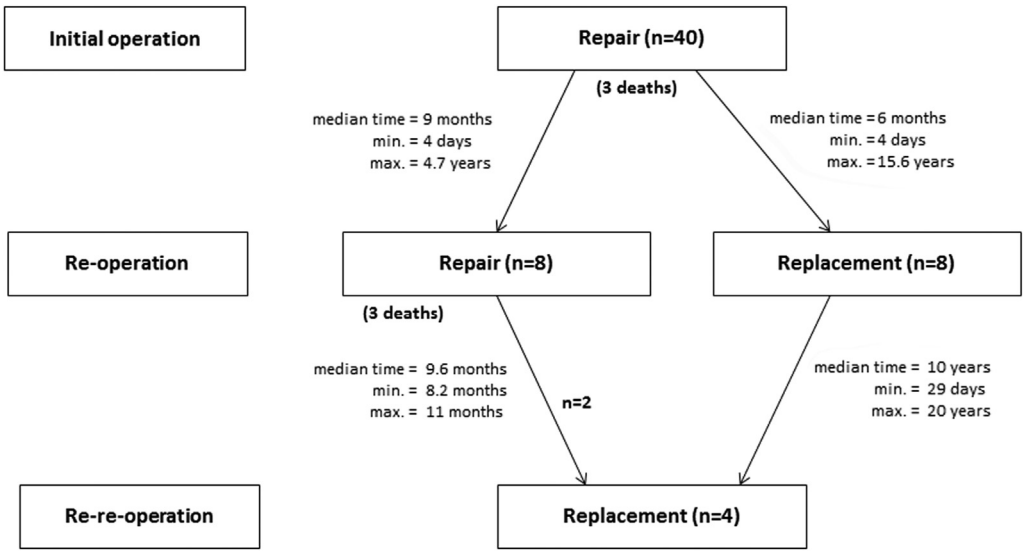

Figure 2. Clinical course and outcomes for patients with congenital mitral valve disease. (max., maximum; min., minimum.)

Autologous pericardium for leaflet augmentation was used in all patients. For ring annuloplasty the following ring prostheses were used: Puig-Massana-Shiley Ring (Shiley Incorporated, Irvine, CA) in 1, Colvin-Galloway Future Ring (Medtronic Inc, Minneapolis, MN) in 2, and Colvin-Galloway Future Band (Medtronic Inc) in 1 patient. Median cardiopulmonary bypass time was 94 minutes (range, 51-295), and median aortic cross-clamp time was 64 minutes (range, 16-177). The end-diastolic volume of the left ventricle decreased from a preoperative median volume of 54.5 mL (range, 11-161) to a postoperative median volume of 36.5 mL (range, 1.6-112; $P = .1$). The left ventricular ejection fraction decreased from a preoperative median ejection fraction of 75% (range, 46%-94%) to a postoperative median ejection fraction of 61.5% (range, 40%-88%; $P = .001$).

Operative mortality was 5% (n = 2). One patient with pulmonary atresia, intact ventricular septum, and severe MR died at age 7.2 months in low cardiac output, 39 days after MV repair, including 4 days after redo MV repair. One patient with congenital aortic stenosis and MS died at age 8.6 months, 18 days after commissurotomy of the aortic and MV due to severe residual aortic valve stenosis.

Late mortality was 10% (n = 4) within a median of 3.1 years (range, 1.3-5.6) after MV repair. Two patients with

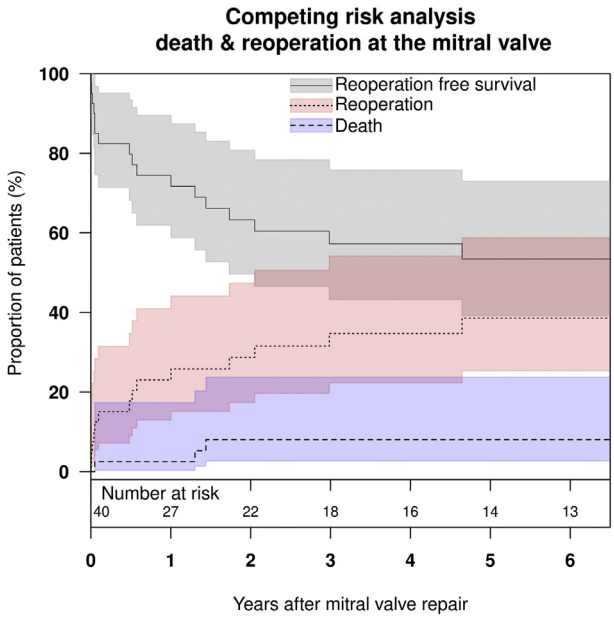

Figure 3. Competing risk analysis for death and reoperation at the mitral valve. Cumulative incidence of reoperation (red) and death with no reoperation (blue). The gray curve shows the patients alive without reoperation.

Table 1. Baseline Characteristics of Patients With Congenital Mitral Valve Disease

Characteristic	Value
Median age, y (range, days-years)	1.2 (14-9.8)
Median weight, kg (range)	8.2 (2.9-41.9)
Male sex	16 (40)
Mitral regurgitation	31 (77.5)
Mitral stenosis	9 (22.5)
Associated cardiac malformations	32 (80)
Ventricular septal defect	11 (27.5)
Aortic stenosis (valvar/subvalvar)	6 (15)
Atrial septal defect II (ostium secundum)	4 (10)
Shone's complex	3 (7.5)
Pulmonary atresia + intact ventricular septum	2 (5)
Tricuspid regurgitation	2 (5)
Transposition of the great vessels	2 (5)
Persistent foramen ovale	1 (2.5)
Patent ductus arteriosus	1 (2.5)

Values are n (%) or median (range).

Table 2. Classification of Congenital Mitral Valve Lesions

Classification	No. of Cases (%)
Mitral valve regurgitation	31 (77.5)
Type I (normal leaflet motion)	21 (52.5)
Annular dilatation	3 (7.5)
Cleft leaflet	17 (42.5)
Leaflet defect	1 (2.5)
Type II (leaflet prolapse)	3 (7.5)
Elongated chordae	3 (7.5)
Type III (restricted leaflet motion)	7 (17.5)
Short chordae	5 (12.5)
Fused commissures	1 (2.5)
Others	1 (2.5)
Mitral valve stenosis	9 (22.5)
Type A (normal papillary muscle)	6 (15)
Supravalvar ring	3 (7.5)
Papillary muscle–commissural fusion	3 (7.5)
Type B (abnormal papillary muscle)	3 (7.5)
Parachute valve	3 (7.5)

additional left ventricular obstruction died of right-sided heart failure more than 8 years after MV repair. In both patients a redo repair had been performed 3 and 4.7 years after MV repair, respectively. One patient died from a viral myocarditis caused by parvovirus B19 more than 1 year after successful MV repair. One patient with MS died of unknown cause 1.4 years after MV repair.

Survival rate at 6 years was 78.6% ± 8%. Patients with congenital MR showed a better, yet not significant, 6-year survival than patients with congenital MS (85.3% ± 8.2% vs 60% ± 18.2%; $P = .1$) (Figure 1).

Mean follow-up time was 6.2 ± 6.9 years, with a median follow-up time of 3.9 years (range, 3 days to 32 years). In

Table 3. Surgical Techniques of Congenital Mitral Valve Repair

Surgical Technique	No. of Cases (%)
Supravalvar repair	3 (7.5)
Resection of a supramitral membrane	3 (7.5)
Valvar repair	32 (80)
Ring annuloplasty	4 (10)
Rigid, complete ring	1 (2.5)
Semirigid, complete ring	2 (5)
Semirigid, partial ring	1 (2.5)
Leaflet procedure	22 (55)
Leaflet/cleft suture	19 (47.5)
Triangular leaflet resection	1 (2.5)
Leaflet augmentation	2 (5)
Alfieri stitch	3 (7.5)
Commissurotomy	3 (7.5)
Subvalvar repair	5 (12.5)
Chordal replacement	1 (2.5)
Papillary muscle splitting	4 (10)

16 patients (40%) a reoperation was performed after initial MV repair. Residual moderate to severe MR was seen in 6 patients, severe MR in 4 patients, moderate MS in 5 patients, and severe MS in 1 patient. In 2 patients reasons for persistent severe MR were functional causes, and in the other 14 patients residual MR or MS was due to morphologic dysfunction of the repaired MV. In 8 patients (50%) a redo repair was performed at a median time of 9 months (range, 4 days to 4.7 years), and 8 patients required a valve replacement at a median time of 6 months (range, 4 days to 15.6 years). During follow-up 4 patients required a MV replacement as re-reoperation. An overview of the clinical course of patients with congenital MVD is shown in Figure 2.

Cumulative incidence of reoperation at 6 years was 38.6% ± 8.3% (Figure 3). In patients with congenital MS risk for reoperation was not significantly higher than for patients with congenital MR (hazard ratio, 1.5; 95% confidence interval, 0.4-2.5; $P = .49$).

Patients With Acquired MVD

Ten patients presented with an acquired MVD, all with prevalent MR. Median age at time of surgery was 1.9 years (range, 10 days to 9.9 years). Reasons for acquired MVD were endocarditis in 5 patients (50%), anomalous left coronary artery originating from the pulmonary artery in 3 patients (20%), and iatrogenic in 2 patients (20%). Iatrogenic MVD included a perforation of the MV after dislocation of a septal occluder and perforation of the anterior MV leaflet during the resection of a left ventricular outflow tract obstruction. Surgical procedures were leaflet suture in 4 patients (40%), cleft suture in 2 patients (20%), and leaflet augmentation in 2 patients (autologous pericardium in 1 patient, CardioCel patch in 1 patient).

Further baseline and operative characteristics of this subgroup are shown in Table 4. No deaths occurred. In 3 patients (30%) a redo repair of the MV because of persistent moderate to severe MR was performed at 3, 30, and 45 days, respectively, after initial MV repair. One patient required a valve replacement 26 days after redo MV repair because of recurrent severe MR.

Comment

The present investigation shows that in children up to 10 years of age, surgical repair of the MV seems beneficial, achieving a survival rate of 79% at 6 years for patients with congenital MVD, whereas patients with congenital MR showed a better, yet not significant, 6-year survival than patients with congenital MS. In patients with congenital MVD cumulative incidence of reoperation at 6 years was 39%.

The most challenging issue in treating pediatric patients with congenital MVD is the range of pathologies affecting leaflets and subvalvar elements of the MV. Systematic classification is thus difficult, as are attempts at surgical standardization, unlike in adults with acquired valvar heart disease. There are excellent, reproducible means of classifying MV pathology in adults[7] contributing to the exceptional outcomes of surgical MV repair

Table 4. Baseline and Operative Data of Patients With Acquired Mitral Valve Disease

Characteristic	Value
Median age, y (range, days-years)	1.9 (10-9.9)
Median weight, kg (range)	10.6 (3.8-30.4)
Male sex	5 (50)
Mitral regurgitation	10 (100)
Endocarditis	5 (50)
Functional	3 (30)
Iatrogenic	2 (20)
Type I leaflet defect	4 (40)
Type I cleft leaflet	2 (20)
Type II elongated chordae	1 (10)
Type II ruptured chordae	1 (10)
Type III fused commissures	1 (10)
Type III others	1 (10)
Associated cardiac malformations	6 (60)
Anomalous left coronary artery originating from the pulmonary artery	3 (50)
Atrial septal defect II (ostium secundum)	2 (33)
Ventricular septal defect	1 (17)
Leaflet suture	4 (40)
Cleft suture	2 (20)
Leaflet augmentation	2 (20)
Chordal replacement	1 (10)
Chordal shortening	1 (10)

Values are n (%) or median (range).

in these patients.[19-21] In a prospective investigation conducted by David and coworkers,[20] the probability of MV reoperation 20 years after initial repair was 5.9% in patients 60 years of age, and freedom from recurrent severe MR was 90.7%. To repair a prolapsed posterior mitral leaflet in adult patients either resection (triangular or quadrangular) or nonresection (chordal replacement) is a viable approach.[22] Lange and colleagues[23] have achieved excellent midterm results through nonresection, which allows the use of larger annuloplasty rings and better physiologic repair, preserving leaflet mobility.

Given these encouraging experiences in adult patients, MV repair has increasingly been applied to children, with good long-term results.[8,9,24,25] However more in-depth analysis is needed to reconcile disparities in outcomes. In our cohort 6-year survival after MV repair was 79%, compared with an 86% survival at 15 years cited by Yakub and colleagues[8] and 93% survival recorded at 20 years by Vida and associates.[9] The median age of our patients with congenital MVD was 1.2 years and for patients with acquired MVD 1.9 years, as opposed to 11 years in the Yakub study and 7 years in Vida's analysis. There was also a higher incidence of associated cardiac defects in our patients, perhaps explaining the difference in survival. We found that patients with congenital MR showed a better, yet not significant, 6-year survival than patients with congenital MS. The frequent association of MS with other left heart obstructive defects may predispose to lethal events.[26]

In children the underlying morphology determines the available surgical options for MV repair. Most MV repairs performed in patients with congenital and acquired MVD were leaflet procedures. Leaflet augmentation was mainly used to treat restrictive leaflets. According to Shomura and coworkers,[27] use of glutaraldehyde-treated autologous pericardium for leaflet augmentation produced good late-term results, with 82% freedom from reoperation at 10 years. In patients with pure dilatation of the mitral annulus, prosthetic annuloplasty rings have proven similarly beneficial for children 11 years of age, with a freedom from reoperation of 79% at 10 years.[8] However the threshold for such implants is higher in smaller children, restricted by their future growth.

The inevitable growth of pediatric patients is both the chief limitation in MV surgery and the principal argument for primary repair instead of valve replacement. Valve replacement at early ages necessitates a multitude of reoperations due to outgrowth of the prosthesis. In terms of freedom from MV reoperation in patients with congenital MVD Vida and colleagues[9] reported a rate of 92% at 10 years, whereas in our study the cumulative incidence of reoperation at 6 years was 39%. This discrepancy can be explained by the higher age at operation and lower incidence of associated cardiac defects in the study by Vida and colleagues. In our study the risk for reoperation in patients with congenital MS was not significantly higher than for patients with congenital MR. This finding was confirmed by Stellin and colleagues[24] and Vida and colleagues.[9]

When comparing patients with congenital MVD and patients with acquired MVD, repair of primary congenital MVD is more complex than repair of acquired MVD reflected in the different reoperation rates and need for valve replacement after initial MV repair. In the present investigation patients with congenital MVD had a higher incidence of MS and left heart obstructive defects such as Shone's complex and severe aortic valve stenosis, resulting in an operative mortality of 5% and a late mortality of 10%. In the retrospective study by Vida and colleagues[9] early and late mortality for patients with congenital MVD was 5% and 9%, respectively, confirming our mortality rates in congenital MVD patients. In the group of patients with acquired MVD no deaths were seen, which underlines the lesser complexity of these patients. It is also important to stress that in our patients acquired MVD was mostly due to endocarditis or functional reasons, resulting in prevalent MR in contrast to most published investigations in which acquired MVD was due to rheumatic causes.[8,28] In patients with rheumatic etiology MV repair is usually more challenging and complex,[13] making a comparison with our subgroup of patients with acquired MVD difficult.

Limitations of this study include its single-center, nonrandomized, and retrospective design. The number of available patients was also rather small for such a long study period, and morphology of the MV was heterogeneous. Finally inconsistencies in preoperative, operative, and postoperative management may have affected our outcome parameters in a way not covered by our analysis.

In conclusion the durability of MV repair in children below the age of 10 years depends on the etiology of the MVD and the underlying morphology. Although MV replacement is not always inevitable in the long term, a repair of the valve should still be the goal of treatment in such patients, even if it just delays the time to replacement.

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