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ORIGINAL RESEARCH

Acute Hemodynamic Changes after Mitraclip Implantation Comparing Patients with Degenerative and Functional Mitral Regurgitation

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ABSTRACT

Background: MitraClip (MC) therapy is a percutaneous treatment option for surgical high-risk patients with both degenerative (DMR) and functional mitral regurgitation (FMR). We compared the acute hemodynamic outcomes of MC therapy in DMR and FMR patients.

Methods: A total of 339 successfully treated patients (77 ± 9 years; 215 men [63%]; 129 DMR [38%], 210 FMR [62%]; LV ejection fraction 40 ± 17%) were hemodynamically assessed pre- and post-MC.

Results: FMR patients had significantly higher pre- and post-MC filling pressures, as well as higher pulmonary capillary wedge and mean pulmonary artery pressures, than DMR patients, but the increase or decrease in these variables did not differ significantly between etiologies. Cardiac output increased significantly in both groups (DMR: 4.1–4.9 l/min; FMR: 4.1–5.1 l/min; both $p < 0.0001$); the increase was statistically not different between etiologies (DMR: 0.8 ± 1.1 l/min; FMR: 1.0 ± 1.1 l/min; $p = 0.06$). With heart rate constant pre- and post-MC, significant increases were also observed in forward stroke volume (DMR: 70–84 ml; FMR: 70–86 ml; both $p < 0.0001$). The mean left atrial v-wave was statistically not different between etiologies at baseline and decreased significantly after MC (DMR: 26–17 mmHg; FMR: 27–21 mmHg; both $p < 0.0001$).

Conclusion: Successful MC therapy results acutely in marked increases in cardiac output and forward stroke volume, and a significant decrease in the left atrial v-wave, in both DMR and FMR patients. Our results attest to the beneficial effect of percutaneous mitral valve repair, particularly in the latter patients with generally poorer baseline health.

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KEYWORDS Etiology; MitraClip; mitral regurgitation; percutaneous mitral valve repair; right-heart catheterization

Introduction

Mitral regurgitation (MR) is the most prevalent valvular heart disease in the general population, increasing along with population aging and heart failure progression.[1] The disease is classified according to its etiology into degenerative (or primary) MR (DMR) and functional (or secondary) MR (FMR). DMR is most commonly due to degeneration of connective tissue with localized or diffuse alterations of the annulus, leaflets, and chordae; FMR is characterized by left ventricular (LV) dysfunction in the absence of structural lesions.[2]

Percutaneous mitral valve repair is an expanding treatment option for patients with moderate-to-severe or severe MR deemed at high surgical risk. The MitraClip (MC; Abbott Vascular, CA, USA) is a percutaneous approach to mitral valve repair in patients with severe MR. The safety and efficacy of MC therapy has been demonstrated for both primary and secondary MR.[3–6] Implantation success is high with over 95% in the three largest MC registries with almost 2000 patients.[6–8] The 30-day mortality rates are not higher than 4.5% and procedural mortality does not exceed 0.1%.[6–8] Most common complications are bleeding complications with up to 7%, but severe complications such as cardiopulmonary resuscitation, tamponade, re-intubation due to respiratory failure or low-cardiac output are lower than 2%.[6–8]

To date, more than 40,000 MC procedures have been performed worldwide[9]; however, only a small amount of data on acute hemodynamic measurements has been published, predominantly in small patient cohorts and without differentiation according to MR etiology.[10–13]

The present study describes the hemodynamic findings before and after MC implantation in considerably larger cohorts of patients with purely degenerative or functional MR.

Materials and methods

Patients

Between September 2009 and November 2015, 578 patients with moderate-to-severe or severe MR underwent MC therapy at our center. Patient selection for the MC procedure was performed by an institutional Heart Team. The study was approved by the Hamburg Ethics Committee (trial

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number: WF-39/17). Hemodynamic parameters were assessed before and after MC implantation. Included in this retrospective analysis were patients with an acutely successful MC implantation (post-procedural MR ≤ 2+) and a complete set of pre- and post-MC data for cardiac output, forward stroke volume, and heart rate. This selection left a total of 339 patients (129 DMR [38%], 210 FMR [62%]).

Cardiac catheterization

Invasive hemodynamic measurements were performed with a Swan-Ganz catheter placed in the pulmonary artery and a pigtail catheter in the left ventricle. Before MC implantation, all measurements (apart from left atrial [LA] pressure and LA v-wave) were performed after femoral access was achieved and prior to transseptal puncture. Measured variables were systolic, diastolic and mean pulmonary artery pressure (PAP), pulmonary capillary wedge (PCW) pressure and PCW v-wave, right atrial pressure, cardiac output, forward stroke volume, LV end-diastolic pressure, as well as systolic, diastolic and mean aortic pressure. LA pressure and the LA v-wave were obtained from pressure measurement using the MC guide catheter placed in the left atrium. Cardiac output was determined by the thermodilution method to exclude hyperoxygenation, taking the average from at least three measurements.[14] After MC implantation, hemodynamic measurements were performed while the MC guide catheter was placed across the atrial septum. Systemic vascular resistance was calculated as (mean aortic pressure – right atrial pressure)/cardiac output × 80 and pulmonary vascular resistance as (mean PAP – PCW pressure)/cardiac output × 80.

MitraClip procedure

All procedures were performed in a hybrid operating room. The procedure was performed under general anesthesia using transesophageal echocardiography and fluoroscopic guidance. MC procedures were performed as previously described.[4,15]

Statistics

Continuous data are described as mean and standard deviation or as median plus first and third quartile; comparisons of hemodynamic variables were made by *t* tests. Categorical data are presented as absolute and relative frequencies; comparisons were made by chi-square or Fisher's exact test. A two-tailed *p*-value <0.05 was considered statistically significant. Statistical analyses were performed with StatView 4.5 (Abacus Concepts, Inc., Berkeley, CA, USA).

Results

Patients

The mean age of the overall patient population was 76 ± 9 years; 215 patients (63%) were men. The operative risk of mortality was reflected by a median logistic EuroSCORE of 18%. FMR patients were on average younger (75 vs. 80 years), were more often men (69% vs. 55%), had a higher logistic EuroSCORE (median 21% vs. 15%), a significantly lower LV ejection fraction (32% vs. 52%), and a higher prevalence of New York Heart Association (NYHA) class IV heart failure (40% vs. 20%). Cardioverter-defibrillators (with and without cardiac resynchronization modality) had been

Table 1. Baseline patient characteristics.

	Total (*N* = 339)	DMR (*n* = 129)	FMR (*n* = 210)	*p*
Men	215 (63)	71 (55)	144 (69)	0.0147
Age, years	76 ± 9	80 ± 8	75 ± 9	<0.0001
Body mass index, kg/m^2	26 ± 5	25 ± 5	26 ± 5	0.0130
Logistic EuroSCORE, %	18 [10, 32]	15 [9, 23]	21 [11, 37]	0.0001
LVEF, %	40 ± 16	52 ± 13	32 ± 13	<0.0001
NYHA functional class				0.0031
II	8/290 (3)	3/108 (3)	5/182 (3)	
III	188/290 (65)	83/108 (77)	105/182 (58)	
IV	94/290 (32)	22/108 (20)	72/182 (40)	
Hypertension	273/334 (82)	109/126 (87)	164/208 (79)	0.08
Hyperlipidemia	210/333 (63)	64/126 (51)	146/207 (71)	0.0004
Diabetes mellitus	85/334 (25)	23/126 (18)	62/208 (30)	0.0199
COPD	67/335 (20)	25/127 (20)	42/208 (20)	1.00
Pulmonary hypertension[a]	170/338 (50)	57 (44)	113/209 (54)	0.09
Atrial fibrillation	238/335 (71)	92/128 (72)	146/207 (71)	0.81
Renal insufficiency[b]	162/335 (48)	55/127 (43)	107/208 (51)	0.18
Coronary artery disease	216/336 (64)	74/128 (58)	142/208 (68)	0.06
Previous cardiac surgery	107/334 (32)	37/126 (29)	70/208 (34)	0.47
Peripheral arterial disease	55/335 (16)	16/127 (13)	39/208 (19)	0.17
Electrical therapy				<0.0001
None	151/232 (65)	56/58 (97)	95/174 (55)	
ICD	44/232 (19)	2/58 (3)	42/174 (24)	
CRT	37/232 (16)	0/58 (0)	37/174 (21)	

Note. Values are mean ± standard deviation, median [first quartile, third quartile], *n* (%), or *n*/*N* (%).
[a]Mean pulmonary artery pressure (right-heart catheterization) ≥25 mmHg.
[b]Glomerular filtration rate <50 ml/min/1.73 m^2.
COPD, chronic obstructive pulmonary disease; CRT, cardiac resynchronization therapy; DMR, primary (degenerative) mitral regurgitation; EuroSCORE, European System for Cardiac Operative Risk Evaluation; FMR, secondary (functional) mitral regurgitation; ICD, implantable cardioverter/defibrillator; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association.

Table 2. Procedural characteristics.

	Pre MitraClip	Post MitraClip
MR severity		
0/1+	–	266 (78)
2+	42 (12)	74 (22)
3+	147 (43)	–
4+	150 (44)	–
Clips implanted		
1		176 (52)
2		142 (42)
>2		21 (6)

Note. All values are *n* (%).

implanted in 45% of FMR, but only 3% of DMR, patients. Pertinent baseline patient characteristics are given in Table 1.

Procedural characteristics relating to MR severity and the MC procedure are shown in Table 2.

Overall hemodynamics

As shown in Table 3 for the total patient population, MC implantation resulted acutely in a significant increase in cardiac output from 4.1 ± 1.3 to 5.0 ± 1.5 l/min, corresponding to a mean increase of 27%. Accordingly, with the mean heart rate stable at 61 min^{-1} throughout the interventions, forward stroke volume increased significantly by 28% (from 70 ± 26 to 86 ± 31 ml). Statistically significant decreases, by a mean of 18%, were observed in the LA v-wave (from 27 ± 12 to 19 ± 8 mmHg) and, although clinically irrelevant, the PCW v-wave (20 ± 9 to 19 ± 7 mmHg) and the mean LA pressure (16 ± 6 to 13 ± 6 mmHg). MC implantation did not impact acutely on PCW pressure and LV end-diastolic pressure. Minor, yet statistically significant increases were noted for mean PAP (by 0.9 ± 6.6 mmHg) and right atrial pressure

Table 3. Hemodynamic variables.

	Total	*p*-value (pre vs. post)	DMR	*p*-value (pre vs. post)	FMR	*p*-value (pre vs. post)	*p*-value (DMR vs. FMR)
Cardiac output, l/min							
Pre	4.1 ± 1.3		4.1 ± 1.3		4.1 ± 1.2		0.92
Post	5.0 ± 1.5	<0.0001	4.9 ± 1.5	<0.0001	5.1 ± 1.2	<0.0001	0.18
Δ	0.9 ± 1.1		0.8 ± 1.1		1.0 ± 1.1		0.06
Δ, %	27 ± 33		23 ± 29		29 ± 35		0.07
Heart rate, min^{-1}							
Pre	61 ± 14		61 ± 15		61 ± 14		0.98
Post	61 ± 13	0.64	60 ± 13	0.32	61 ± 13	0.75	0.42
Δ	−0.2 ± 9.5		−1.0 ± 10.8		0.2 ± 8.7		0.28
Forward stroke volume, ml							
Pre	70 ± 26		70 ± 25		70 ± 26		0.91
Post	86 ± 31	<0.0001	84 ± 30	<0.0001	86 ± 31	<0.0001	0.58
Δ	16 ± 21		15 ± 21		17 ± 22		0.40
Δ, %	28 ± 37		25 ± 33		30 ± 40		0.23
V-wave (left atrium), mmHg							
Pre	27 ± 12		26 ± 12		27 ± 12		0.62
Post	19 ± 8	<0.0001	17 ± 7	<0.0001	21 ± 9	<0.0001	<0.0001
Δ	−8 ± 11		−10 ± 12		−6 ± 10		0.0026
Δ, %	−18 ± 45		−29 ± 35		−12 ± 49		0.0012
V-wave (pulmonary capillary wedge), mmHg							
Pre	20 ± 9		19 ± 9		21 ± 9		0.08
Post	19 ± 7	0.0006	17 ± 6	0.0143	20 ± 7	0.0167	0.0009
Δ	−2 ± 9		−2 ± 10		−1 ± 8		0.46
Left atrial pressure, mmHg							
Pre	16 ± 6		14 ± 5		16 ± 7		0.0019
Post	13 ± 6	<0.0001	11 ± 5	<0.0001	14 ± 6	<0.0001	<0.0001
Δ	−3 ± 6		−4 ± 6		−3 ± 6		0.25
Pulmonary capillary wedge pressure, mmHg							
Pre	16 ± 7		15 ± 6		16 ± 7		0.0207
Post	15 ± 6	0.33	14 ± 5	0.28	16 ± 6	0.69	<0.0001
Δ	−0.4 ± 6.4		−0.6 ± 6.2		−0.2 ± 6.5		0.61
LV end-diastolic pressure, mmHg							
Pre	14 ± 6		12 ± 5		15 ± 6		0.0002
Post	12 ± 7	0.05	10 ± 4	0.0045	14 ± 7	0.55	<0.0001
Δ	−1 ± 7		−2 ± 5		−0.5 ± 8		0.20
Mean pulmonary artery pressure, mmHg							
Pre	26 ± 8		25 ± 8		27 ± 8		0.0327
Post	27 ± 7	0.0168	25 ± 7	0.30	28 ± 7	0.0286	0.0008
Δ	0.9 ± 6.6		0.5 ± 6.4		1.1 ± 6.8		0.48
Right atrial pressure, mmHg							
Pre	8 ± 4		7 ± 4		8 ± 5		0.14
Post	9 ± 4	<0.0001	8 ± 4	0.0203	10 ± 4	<0.0001	0.0379
Δ	1.1 ± 4.3		0.9 ± 4.3		1.3 ± 4.4		0.47
Mean aortic pressure, mmHg							
Pre	70 ± 11		70 ± 12		69 ± 11		0.31
Post	73 ± 11	<0.0001	74 ± 12	0.0106	72 ± 11	0.0022	0.15
Δ	3 ± 13		3 ± 14		3 ± 12		0.70
Systemic vascular resistance, dyn·sec/cm^5							
Pre	1314 ± 491		1346 ± 517		1295 ± 475		0.37
Post	1098 ± 413	<0.0001	1149 ± 421	<0.0001	1067 ± 405	<0.0001	0.09
Δ	−205 ± 422		−179 ± 411		−222 ± 428		0.40
Pulmonary vascular resistance, dyn·sec/cm^5							
Pre	215 ± 147		216 ± 145		215 ± 149		0.97
Post	198 ± 113	0.0178	200 ± 115	0.15	197 ± 112	0.59	0.81
Δ	−18 ± 134		−18 ± 136		−18 ± 133		0.99

Note. All values are mean ± standard deviation (SD). Δ = post − pre.

(by 1.1 ± 4.3 mmHg). Mean aortic pressure increased significantly from 69 ± 11 to 73 ± 11 mmHg during the procedure. Mean systemic and pulmonary vascular resistances were both significantly reduced after MC implantation (1314 ± 491 to 1098 ± 413 dyn·sec/cm^5 and 215 ± 147 to 198 ± 113 dyn·sec/cm^5, respectively).

Comparison of DMR and FMR patients

Notably, the acute increase in cardiac output by about 1 l/min and forward stroke volume by about 16 ml following MC implantation was present in both DMR and FMR patients (Table 3; Figures 1 and 2). Significant differences between DMR and FMR patients were observed in the post-MC LA v-wave (17 ± 7 vs. 21 ± 9 mmHg, respectively; Figure 3),

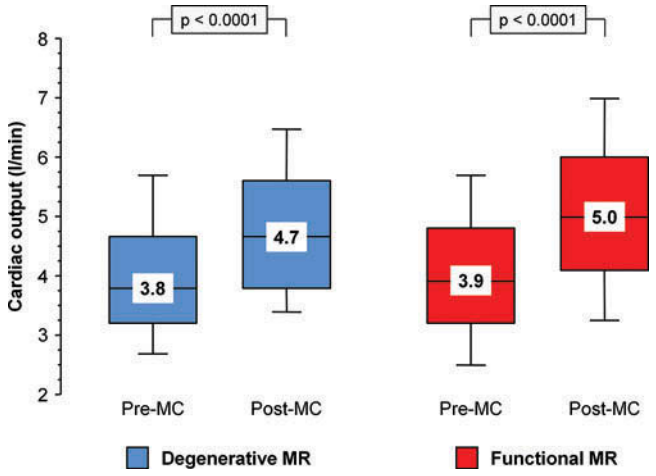

Figure 1. Distributions of cardiac output measurements before (pre-MC) and after MitraClip implantation (post-MC) according to MR etiology. In the box-and-whiskers plots, the horizontal line with number in box denotes median; top and bottom end of box denote 75th and 25th percentile, respectively; top and bottom ends of whiskers denote 90th and 10th percentile, respectively. Statistically significant increases in cardiac output by about 1 l/min are seen in both types of MR etiology. MR, mitral regurgitation.

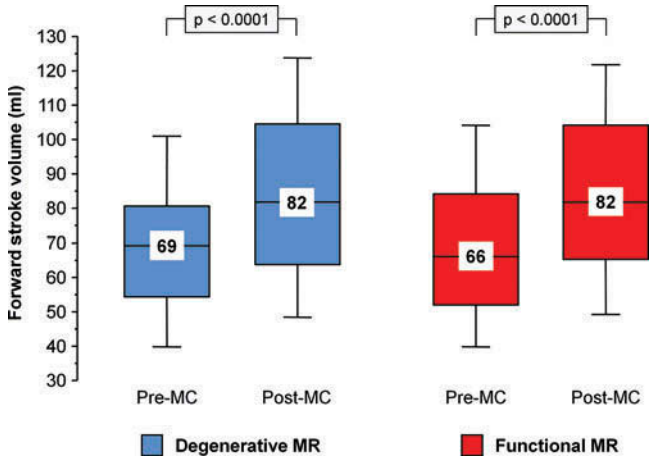

Figure 2. Distributions of forward stroke volume measurements before and after MitraClip implantation according to MR etiology. Statistically significant increases in forward stroke volume by about 15 ml are present in both types of MR etiology. For abbreviations and description of box-and-whiskers plots see Figure 1.

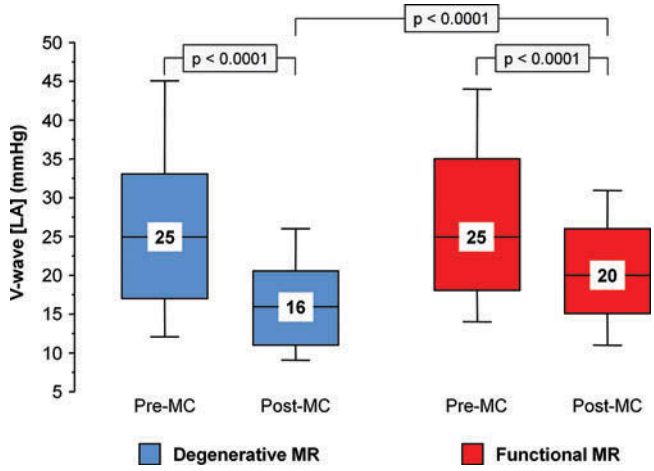

Figure 3. Distributions of left atrial v-wave measurements before and after MitraClip implantation according to MR etiology. Statistically significant decreases in left atrial v-wave are present in both types of MR etiology. Note that left atrial v-wave at baseline is not different between etiologies and that the decrease in left atrial v-wave is less pronounced in patients with functional MR. LA, left atrium; other abbreviations and description of box-and-whiskers plots as in Figure 1.

reflecting a lesser MC-induced LA v-wave decrease in FMR patients (−6 ± 10 mmHg [−12 ± 49%] vs. DMR −10 ± 12 mmHg [−29 ± 35%]). Such a difference between etiologies in MC-induced change was not found for the PCW v-wave. LA pressures pre- and post-MC were slightly higher in FMR patients, but the mean reduction in LA pressure induced by MC implantation was statistically not different between etiologies (DMR −4 ± 6 mmHg, FMR −3 ± 6 mmHg). Similarly, PCW pressure was marginally higher pre- and post-MC in FMR patients, with no significant difference between etiologies in the MC-induced change (DMR −0.6 ± 6.2 mmHg, FMR −0.2 ± 6.5 mmHg). LV end-diastolic pressure and mean PAP were also higher in FMR than DMR patients, both pre-MC (15 ± 6 vs. 12 ± 5 mmHg, respectively, for the former and 27 ± 8 vs. 25 ± 8 mmHg, respectively, for the latter) and post-MC (14 ± 7 vs. 10 ± 4 mmHg, respectively, for LV end-diastolic pressure and 28 ± 7 vs. 25 ± 7 mmHg, respectively, for mean PAP), but the change affected by MC implantation was statistically not different between the etiologies (Table 3). No clinically relevant differences between MR etiologies were observed for right atrial and mean aortic pressure as well as for systemic and pulmonary vascular resistance (Table 3). Intraprocedural cardiac rhythm (sinus rhythm or atrial fibrillation) has not been documented; however, the prevalence of atrial fibrillation at baseline was not different between DMR and FMR patients.

Discussion

Main findings

The main findings of this study comparing hemodynamics in DMR and FMR patients before and after a successful MC procedure are as follows:

- MC implantation resulted in significant increases in cardiac output and forward stroke volume in patients with either type of MR etiology, despite the fact that

baseline LV ejection fraction was significantly lower in patients with FMR.
- A significant reduction in the LA v-wave (greater than in the PCW v-wave), as an invasive sign of MR reduction, is seen in patients with either MR etiology.

Previous studies

In 2011, Siegel and colleagues reported on acute hemodynamic effects in 107 patients treated in the EVEREST (Endovascular Valve Edge-to-Edge Repair Study) phase I feasibility trial and the roll-in phase of the EVEREST II pivotal trial; only a minority (21%) of these patients had FMR.[12] The authors observed that successful MC implantation was associated with significant increases in forward stroke volume and cardiac output and a significant decrease in systemic vascular resistance; also, left ventricular (LV) end-diastolic pressure was significantly reduced post MC. No differentiation according to MR etiology was made. Gaemperli and co-workers, studying 50 patients (28 [56%] with FMR), observed a significant MC-related increase in cardiac index—thereby confirming Siegel and colleagues' results—as well as significant reductions in mean PAP and PCW pressure.[9] Again, results were not differentiated according to MR etiology. Pressure-volume loop recordings obtained by the same working group from 33 patients (15 [45%] with FMR) confirmed their previous findings of an MC-induced increase in cardiac index and a decrease in mean PCW pressure.[10] This time, the authors presented results separately for the 16 DMR patients and the remaining 17 patients, in which the 15 FMR patients were unfortunately grouped with two patients in whom MR of mixed etiology was present. Our study supports those authors' finding of an increased cardiac index after MC implantation; however, we did not observe a significant decrease in PCW pressure.

Baseline conditions in DMR and FMR patients

The present study of 339 patients who underwent MC treatment in a real-world scenario provides important hemodynamic information, also on baseline conditions, with a strict differentiation according to MR etiology.

Baseline cardiac output and forward stroke volume were not significantly different between MR etiologies, despite the fact that LV ejection fraction was significantly lower in patients with FMR than in patients with DMR. This is in line with published data and the pathomechanism of FMR with reduced LV function.[6] The lack of a difference in cardiac output and forward stroke volume (and heart rate) between these markedly different patient populations with respect to MR etiology and LV ejection fraction might be explained by afterload reduction, especially in FMR patients, due to general anesthesia; this was also described by Gaemperli and co-authors.[11]

Baseline v-wave amplitude in both the LA and the PCW position, as an invasive sign of a MR,[16] was not significantly different between the two MR etiologies. V-wave amplitudes reflecting the regurgitant flow into the LA seem to be elevated in patients with significant MR regardless of etiology.

In the present study, LA and LV filling pressures as well as pulmonary artery and PCW pressures were consistently higher in FMR patients. FMR patients may therefore be classified as sicker than DMR patients, a fact that is also reflected in a higher logistic EuroSCORE, lower LV ejection fraction, and more pronounced heart failure symptoms.

Effects of MC implantation in all patients

In a recent European survey, impaired LV ejection fraction, advanced age, and comorbidities were the most prominent characteristics of patients with severe symptomatic MR who were denied surgery.[17] For inoperable or surgical high-risk patients, MC therapy has been shown to be safe and efficacious; a low cardiac output state associated with the procedure, as seen in patients after cardiac surgery, is rare.[7,10,12] The finding of MC-associated overall increases in both cardiac output and forward stroke volume (in the presence of a stable heart rate) reflects the patients' hemodynamic improvement after correction of MR. Similar results were described in smaller patient populations by Siegel and colleagues and Gaemperli and colleagues, who unfortunately mixed successfully with unsuccessfully treated patients, measured forward stroke volume by echocardiography and cardiac output by invasive methods, and provided no information on heart rates pre- and post-intervention.[10–12]

The reduction or elimination of the low-impedance backflow into the LA is reflected in the present study primarily by the reduction in LA and PCW v-wave amplitude, as well as in LA pressure. The increase in forward stroke volume results most likely from blood flow redirected into the left ventricular outflow tract, thereby increasing the mean aortic pressure. The observation of an acute postoperative low cardiac output state after surgical correction of MR due to the acute elimination of backflow,[18,19] cannot be seen in the present data.

There is incongruent data on mortality after mitral valve repair in FMR patients.[20,21] Procedural mortality is rare in MC procedures. The significant increase in cardiac output and forward stroke volume observed in the present study was independent of the type of MR. Our findings implicate that even heart failure patients (predominantly FMR patients) benefit hemodynamically at least acutely from MC implantation.

Comparison of hemodynamic changes of DMR and FMR patients after successful MC implantation

Our study of 339 patients differentiated strictly between MR etiologies and assessed hemodynamic changes following a single, successful mitral valve repair procedure.

The effect of MR on the LV in DMR patients is predominantly volume overload, whereas in FMR patients ischemic or dilated cardiomyopathy with LV dysfunction causes MR and increases the volume overload on the already reduced contractility of the LV.[22,23] MR and volume overload will lead to reduced forward stroke volume and elevation of PCW, PAP, LV end-diastolic and LA pressures, particularly in chronic

stages of FMR.[22,23] These elevated filling pressures are also signs of invasive hemodynamic decompensation, as defined by Biner and co-authors.[13] In our study, there was no difference in the improvement in cardiac output and forward stroke volume between the two types of MR. This suggests that mitral valve repair redirects the regurgitant backflow into the LA into a forward flow towards the aorta regardless of the etiology of MR. The improved hemodynamics support the clinical superiority of MC therapy over medical treatment in FMR patients.[24,25]

Interestingly, the statistically significant, if small (2 mmHg), acute reduction in LV end-diastolic pressure after MC in DMR patients was not observed in FMR patients, possibly because of the ventricular disease present in the latter. It needs to be seen if the increase in forward stroke volume reduces LV end-diastolic pressure over time, and thereby slows or even stops the progression of heart failure, in FMR patients.

The amplitude of the v-wave is only an indicator of the presence of MR; grading of MR severity requires echocardiography. Measuring the LA v-wave or LA pressure during MC procedures can add information on the reduction of MR regardless of the etiology. This is in line with data from Horstkotte and co-authors who showed improved MR reduction by continuous LA pressure monitoring during MC therapy.[26]

Study limitations

This is a single-center study describing MC-induced hemodynamic changes. Data analysis was performed retrospectively. Data on fluid management and general anesthesia including catecholamine therapy was not collected. Correlations of hemodynamic findings with left ventricular volumes, dimensions, and MR severity were not performed.

Conclusions

MC therapy resulted in significant increases by 27% in cardiac output and 28% in forward stroke volume in the overall patient cohort, with no significant differences between MR etiologies. The latter finding is all the more remarkable, since FMR patients were generally in poorer health (higher logistic EuroSCORE, lower LV ejection fraction, higher prevalence of NYHA class IV heart failure, elevated filling and pulmonary pressures) than DMR patients. MC implantation results acutely in improved hemodynamic conditions in patients with either MR etiology.

Disclosure statement

Christian Frerker and Felix Kreidel have received lecture honoraria from Abbott Vascular, Inc. Ulrich Schäfer has received lecture honoraria as well as research grants from Abbott Vascular, Inc. and is a member of Crossroads Abbott Vascular. Karl-Heinz Kuck has received lecture honoraria as well as research grants from Abbott Vascular, Inc. The other authors report no conflict of interest.

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