

A Cardiovascular Magnetic Resonance Study on the short and long-term effects of Coronary Artery Bypass Graft Surgery on the Right Ventricular Systolic Function

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Article Info

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Received: March 11, 2017

Accepted: March 24, 2017

Published: March 30, 2017

Citation: Becker MAJ, Robbers LFHJ, Brouwer WP, et al. A Cardiovascular Magnetic Resonance Study on the short and long-term effects of Coronary Artery Bypass Graft Surgery on the Right Ventricular Systolic Function. *Madridge J Cardiol.* 2017; 2(1): 14-20.

doi: 10.18689/mjc-1000105

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Published by Madridge Publishers

Abstract

Purpose: Right ventricular (RV) systolic function is an important prognostic factor in various cardiovascular diseases. Echocardiography, by measuring the tricuspid annular plane systolic excursion (TAPSE), often demonstrates an apparent decreased RV function after coronary artery bypass grafting (CABG). Cine cardiovascular magnetic resonance imaging (CMR) with steady state free precession (SSFP) is considered the gold standard for evaluation of RV systolic function. We used this technique to evaluate RV systolic function at baseline (T0), short term (T1, 4±1 months), mid-term (T2, 7±1 months) and long-term (T3, 32±11 months) follow-up after CABG. Also, we assessed if grafting of the right coronary artery (RCA) had any influence on the postoperative RV function.

Methods: Thirty-four patients (29 men; mean age 63±9 years) had CABG and underwent serial CMR examinations. Dedicated software (Qmass, Medis, Leiden, the Netherlands) was used for quantification of RV ejection fraction (RVEF), on a stack of short-axis cine SSFP images. TAPSE was measured on a four chamber cine image.

Results: During all follow-up time points, RVEF remained unchanged (57±14%, 59±14%, and 58±12% respectively, all p-values: 1.0), compared to baseline measurement (61±11%). TAPSE, however, showed a significant decrease after CABG at all time points (17±5mm vs. 10±4mm, vs. 9±3mm and vs. 10±3mm respectively; all p-values <0.001). No differences in RV systolic function were found between patients with or without revascularization of the RCA.

Conclusion: Our CMR study showed a preserved RV systolic function after CABG at short-, mid- and long-term follow-up while TAPSE showed a persistent decrease after surgery, thereby underestimating true systolic function of the RV. Revascularization of the RCA had no influence on the postoperative RV systolic function.

Keywords: Right ventricular function; TAPSE; Cardiovascular Magnetic Resonance Imaging; Coronary Artery Bypass Graft.

Introduction

The right ventricular (RV) systolic function is an important prognostic variable in a wide range of cardiovascular diseases [1-5]. A frequently observed phenomenon after coronary artery bypass graft (CABG) surgery is an apparent decrease in RV systolic function, when the tricuspid annular plane systolic excursion (TAPSE) is measured on Echocardiography [6-9]. However, cardiovascular magnetic resonance imaging (CMR) is considered the gold standard

for quantitative non-invasive assessment of ventricular function [10-16]. Currently, only limited data are available on the assessment of RV systolic function after CABG with CMR. Furthermore, several studies reported conflicting findings about the change in RV systolic function after CABG. Due to these ambiguous results, the effects of CABG on long-term RV systolic function are still unclear. The aim of this study was to investigate the long-term effects of CABG on the RV systolic function, using CMR to assess TAPSE and RV volumes and function [1,10,12,15]. Secondly, we assessed whether grafting of the right coronary artery (RCA) had any influence on the postoperative RV systolic function.

Methods

Study Population

The patients for this study were recruited from the CMR database of the cardiology department of the VU University Medical Center, Amsterdam, the Netherlands. CMR data of 34 patients who underwent CABG surgery between 2001 and 2004 were analysed. Exclusion criteria were contra-indications for CMR (e.g. cardiac pacemaker, ferromagnetic implants, claustrophobia, inability to stay in a supine position for 30-45 minutes) [12]. The study was approved by the local institutional review board and patients provided informed consent.

CMR

All CMR acquisitions were performed at baseline (preoperatively) (T0), at short term (T1, 4±1 months), mid-term (T2, 7±1 months), and long-term (T3, 32±11 months) follow-up after CABG on a 1.5 Tesla clinical MR system (Sonata, Siemens, Erlangen, Germany). Functional imaging was performed by using retrospectively ECG-gated steady-state free precession (SSFP) cine imaging with breath-holding. In each patient standard long-axis cine images were acquired. A total of 8-10 short-axis slices were obtained every 10mm, starting at the mitral valve annulus and covering the entire volume of both ventricles.

CMR analysis

Analysis of the CMR data for the assessment of RV volumes was performed with dedicated post processing software (Qmass, V2011, Medis, Leiden, the Netherlands) based on methods described before [12]. Endocardial borders of RV were outlined on short-axis cine images at end-diastole and end-systole. RV volumes and RVEF were measured and calculated using the disk-area summation method (modified Simpson's rule) [17,18]. Trabeculae and papillary muscles were considered part of the blood pool volume. The four-chamber long-axis images were used to verify whether or not the basal short axis slice was part of the RV and included or excluded from the analysis.

In addition to the right ventricular volumes and function, the left ventricular ejection fraction was evaluated at T0 and T1 as well, to exclude LV dysfunction as a cause of eventual changes in RV function post CABG.

TAPSE was calculated on a four-chamber cine image using different software (Centricity Radiology RA600 v6.1, GE Healthcare, Buckinghamshire, United Kingdom). A fixed point on the thoracic wall, in line with the interventricular septum

was chosen as the reference point. The distance between the reference point and the lateral side of the tricuspid annulus was measured during end-diastole (end-diastolic length [EDL] in mm) and end-systole (end-systolic length [ESL] in mm). TAPSE was defined as the difference between EDL and ESL [12] (figure 1).

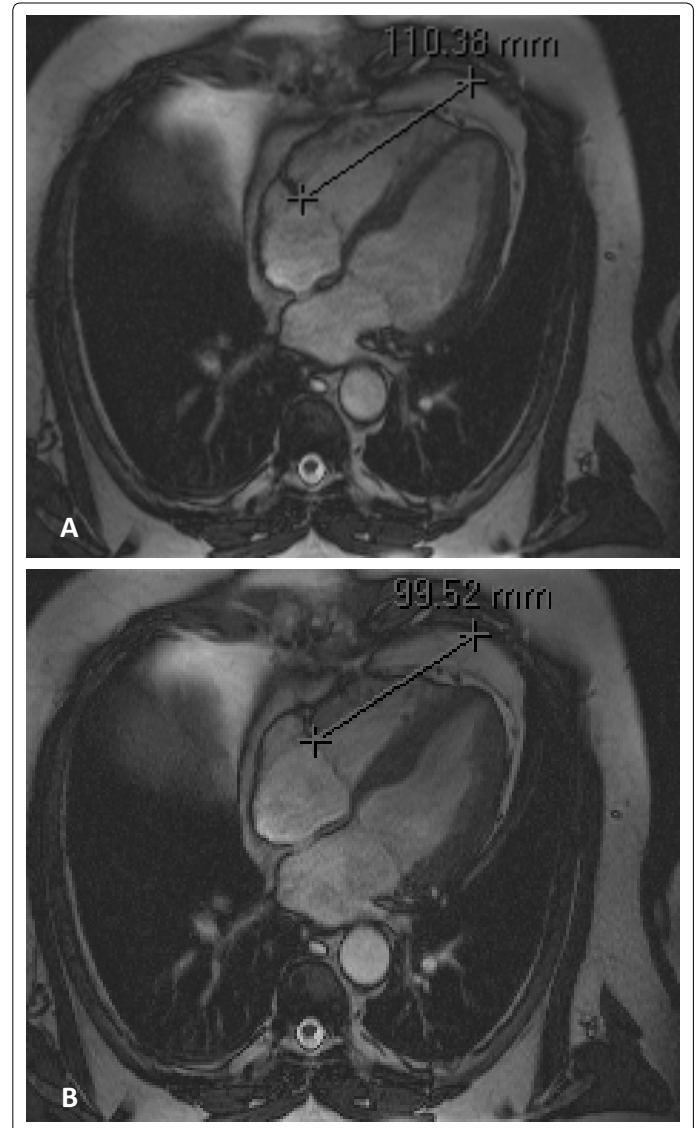


Figure 1: TAPSE was defined as the difference between End Diastolic Length (A) and End Systolic Length (B).

We divided our patients in two groups; one group with revascularization of the right coronary artery (RCA-group) and the other group with revascularization of coronary arteries other than RCA (non-RCA-group).

Statistical analysis

Statistical analysis was performed using SPSS 15.0 for Windows (SPSS Inc, Chicago, Illinois, USA). The repeated measurements during the follow-up period were initially compared using one-way ANOVA-analysis with Bonferroni correction. To compare the LVEF before and after CABG, a paired-samples t-test was used. Comparisons between the RCA- and non-RCA-group were made with the independent-samples t-test. The results are expressed as the mean and one standard deviation (mean±SD). A p-value of less than 0.05 was considered to be statistically significant.

Results

Study population

In total, 34 patients (29 men and 5 women) underwent baseline CMR acquisitions. The mean age was 63±9 years. At T1 33 patients had a follow-up CMR, 32 patients had a follow-up at T2 and 27 had a follow-up CMR at T3. Co-morbidities of the population were systemic hypertension (n=11), chronic myocardial infarction (n=17), diabetes mellitus (n=5), hypercholesterolemia (n=7), arrhythmia (n=6) and valvular disease (n=6). Twenty-six patients had angina complaints of different severity (New York Heart Association class I [n=2], II [n=4] and III [n=20]). In 27 patients the RCA was involved in the revascularization and in 7 patients the RCA was not involved in the revascularization. The baseline and follow-up characteristics are shown in table 1.

Table 1. Characteristics of the RCA- and non-RCA-group at baseline and follow-up

	RCA	non-RCA	P-value
N			
T0	27	7	
T1	27	6	
T2	26	6	
T3	24	3	
Age (years)			
T0	64 ± 10	61 ± 8	p=0.45
T1	64 ± 10	60 ± 8	p=0.35
T2	65 ± 10	62 ± 8	p=0.52
T3	67 ± 10	60 ± 7	p=0.26
Gender			
Men / women	22 / 5	7 / 0	p=0.02
LVEF (%)			
T0	40 ± 12	38 ± 13	p=0.69
T1	40 ± 13	36 ± 9	p=0.45
EDV (ml)			
T0	112 ± 24	134 ± 47	p=0.27
T1	103 ± 31	128 ± 34	p=0.10
T2	98 ± 34	128 ± 35	p=0.06
T3	108 ± 37	142 ± 40	p=0.16
ESV (ml)			
T0	43 ± 15	56 ± 29	p=0.29
T1	46 ± 22	54 ± 26	p=0.43
T2	41 ± 20	51 ± 24	p=0.29
T3	44 ± 16	58 ± 27	p=0.22
SV (ml)			
T0	69 ± 21	79 ± 22	p=0.30
T1	59 ± 24	74 ± 13	p=0.15
T2	57 ± 25	77 ± 16	p=0.08
T3	64 ± 29	84 ± 12	p=0.24
RVEF (%)			
T0	62 ± 12	60 ± 8	p=0.77
T1	56 ± 15	60 ± 11	p=0.61
T2	58 ± 15	61 ± 10	p=0.63
T3	58 ± 13	61 ± 8	p=0.67
TAPSE (mm)			
T0	16 ± 5	20 ± 5	p=0.08
T1	10 ± 4	11 ± 3	p=0.61
T2	10 ± 4	9 ± 3	p=0.64
T3	10 ± 3	11 ± 3	p=0.53
Co-morbidity			
- AP:			
NYHA I	1	1	p=0.34
NYHA II	3	1	p=0.88
NYHA III	17	3	p=0.24
NYHA unknown	3	2	p=0.30
- EH	7	4	p=0.16
- DM	4	1	p=0.92
- MI	12	5	p=0.29
- valve diseases	6	0	p=0.01
- arrhythmia	5	1	p=0.74
- HC	6	1	p=0.60
- others	11	3	p=0.96

Table 1: Between both groups, significant differences were only found in gender and concomitant valve disease.

LVEF: left ventricular ejection fraction; EDV: end diastolic volume; ESV: end systolic volume; SV: stroke volume; RVEF: right ventricular

ejection fraction; TAPSE: tricuspid annular plane systolic excursion; AP: angina pectoris; EH: essential hypertension; DM: diabetes mellitus; MI: myocardial infarction; HC: hypercholesterolemia.

CMR findings

The LVEF did not change significantly at T1 compared to T0 (40±12% at T0 to 39±12% at T1; p=0.71).

The end diastolic volume (EDV) showed no significant change at follow-up compared to baseline measurements (T0: 116±31ml vs. T1: 108±32ml, T2: 104±35ml, T3: 112±38ml. P-values are respectively 1.0, 0.89 and 1.0) (figure 2A).

The end systolic volume (ESV) did not change significantly after CABG (45±19ml at T0, 47±22ml at T1, 43±21ml at T2 and 46±17ml at T3. P-values = 1.0) (figure 2B).

Stroke volume (SV) showed no significant change either after CABG (71±22ml at T0 to 61±23ml at T1, 61±25ml at T2 and 66±28ml at T3; p=0.63, p=0.60, p=1.0 respectively) (figure 2C).

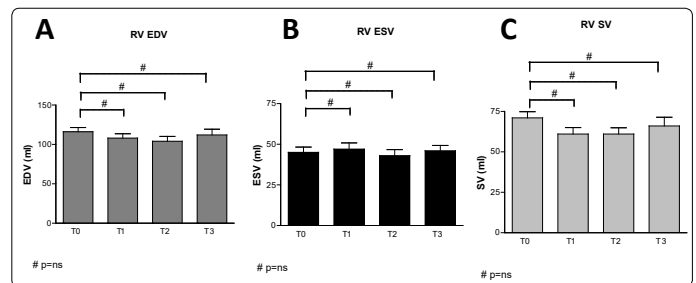


Figure 2: No significant changes were found in EDV (A), ESV (B) or SV (C) at either point of follow-up after CABG.

RVEF was not significantly changed at T1 compared to T0 (T0: 61±11% to T1: 57±14%, p=1.0). During the follow-up at T2 and T3, RVEF did not change either (59±14% and 58±12%, respectively. Both p-values=1.0) (figure 3A).

The TAPSE, however, was significantly decreased at T1 compared to T0 (17±5mm at T0 to 10±4mm at T1, p<0.001). During follow-up at T2 and T3, TAPSE remained significantly decreased in comparison to T0 values (at T2 9±3mm and at T3 10±3mm, both p-values <0.001). However, TAPSE showed no further decrease at T2 and T3 in comparison to T1 measurements (both p-values=1.0) (figure 3B).

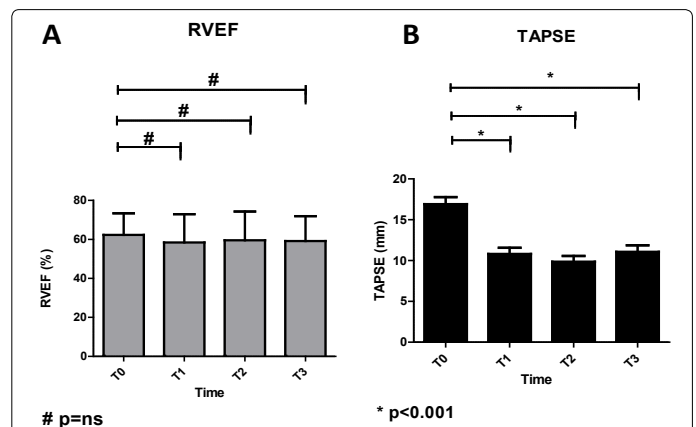


Figure 3: No significant change in RVEF was found at T1 compared to T0 (A), whilst TAPSE decreased significantly (B). At T2 and T3, there were no further changes in either RVEF or TAPSE.

RCA vs. non-RCA

At baseline, the RCA and the non-RCA CABG groups were

well matched for age, however, the non-RCA revascularized group did not contain any women ($p=0.02$). Concomitant valvular pathology (mitral valve regurgitation: $n=5$; tricuspid valve regurgitation: $n=1$) was significantly more seen in the RCA group than in the non-RCA-group ($n=6$ and $n=0$ respectively; $p=0.01$). At baseline, no significant differences in RV volumes, RVEF, LVEF and TAPSE were seen between the two groups (table 1).

In both the RCA- and the non-RCA-group, the LVEF showed no significant change at T1 compared to T0 (RCA: $40\pm 12\%$ at T0 to $40\pm 13\%$ at T1, $p=0.98$; non-RCA: $38\pm 13\%$ at T0 to $36\pm 9\%$ at T1, $p=0.38$). No significant difference was seen in LVEF between both groups at either time point (T0: $p=0.69$ and T1: $p=0.45$).

The differences in RV volumes between RCA- versus non-RCA-revascularization are outlined in figure 4 A-C. In both groups, we found no significant difference in EDV after CABG (RCA: $103\pm 31\text{ml}$ at T1, $98\pm 34\text{ml}$ at T2, $108\pm 37\text{ml}$ at T3 compared to $112\pm 24\text{ml}$ at T0, all p -values: 1.0; non-RCA: $128\pm 34\text{ml}$ at T1, 128 ± 35 at T2, $142\pm 40\text{ml}$ at T3 compared to $134\pm 47\text{ml}$ at T0, $p=0.80 - 1.0$) (figure 4A). Differences between both groups were not significant ($p=0.06 - 0.27$) (table 1).

The ESV showed no significant differences in follow-up measurements compared to T0 values (RCA: $46\pm 22\text{ml}$, $41\pm 20\text{ml}$, $44\pm 16\text{ml}$, respectively T1, T2, T3, in comparison to $43\pm 15\text{ml}$ at baseline, all p -values: 1.0; non-RCA: at T1 $54\pm 26\text{ml}$, T2 $51\pm 24\text{ml}$, T3 $58\pm 27\text{ml}$, compared to $56\pm 29\text{ml}$ at T0, all p -values: 1.0) (figure 4B), nor between the RCA- and the non-RCA-group ($p: 0.22 - 0.43$) (table 1).

Evaluation of SV showed no significant differences either between follow-up measurements (RCA: 59 ± 24 at T1, $57\pm 25\text{ml}$ at T2 and 64 ± 29 at T3, in comparison to $69\pm 21\text{ml}$ at T0, $p=0.56 - 1.0$; non-RCA: $74\pm 13\text{ml}$, $77\pm 16\text{ml}$ and $84\pm 12\text{ml}$ at respectively T1, T2 and T3, compared to $79\pm 22\text{ml}$ at T0, all p -values: 1.0) (figure 4C), nor between the two groups ($p: 0.08 - 0.30$) (table 1).

During follow-up measurements, RVEF was not significantly changed at T1 compared to T0 in either the RCA-group (T0: $62\pm 12\%$ vs. T1: $56\pm 15\%$, $p=0.95$) or the non-RCA-group (T0: $60\pm 8\%$ vs. T1: $60\pm 11\%$, $p=1.0$). RVEF remained unchanged during T2 (RCA: $58\pm 15\%$, $p=1.0$; non-RCA: $61\pm 10\%$, $p=1.0$) and T3 (RCA: $58\pm 13\%$, $p=1.0$; non-RCA: $61\pm 8\%$, $p=1.0$) in comparison to T0 values (figure 5A). Differences between the RCA and non-RCA group in RVEF were not significant at any time point ($p: 0.61 - 0.77$) (table 1).

TAPSE decreased significantly in the RCA- as well as the non-RCA-group at T1 compared to T0 (RCA: $16\pm 5\text{mm}$ at T0 to $10\pm 4\text{mm}$ at T1, $p<0.001$; non-RCA: $20\pm 5\text{mm}$ at T0 to $11\pm 3\text{mm}$ at T1, $p=0.003$). TAPSE remained reduced, but did not decrease further at T2 and T3 compared to T1 in either the RCA- or the non-RCA-group (RCA: $10\pm 4\text{mm}$ at T2 and $10\pm 3\text{mm}$ at T3, both p -values: 1.0; non-RCA: $9\pm 3\text{mm}$ at T2 and $11\pm 3\text{mm}$ at T3, both p -values: 1.0) (figure 5B). Intergroup differences were not significant ($p: 0.08 - 0.64$) (table 1).

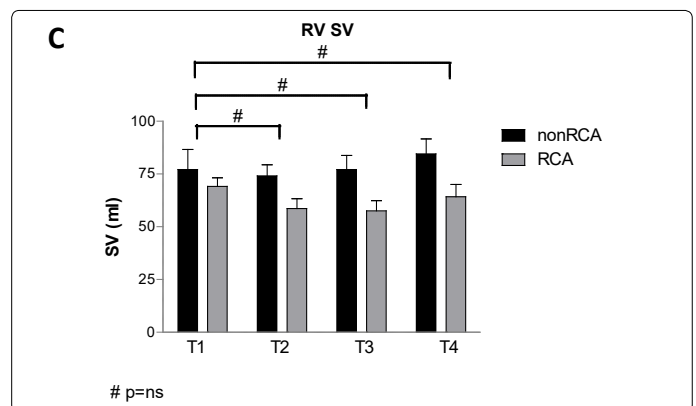
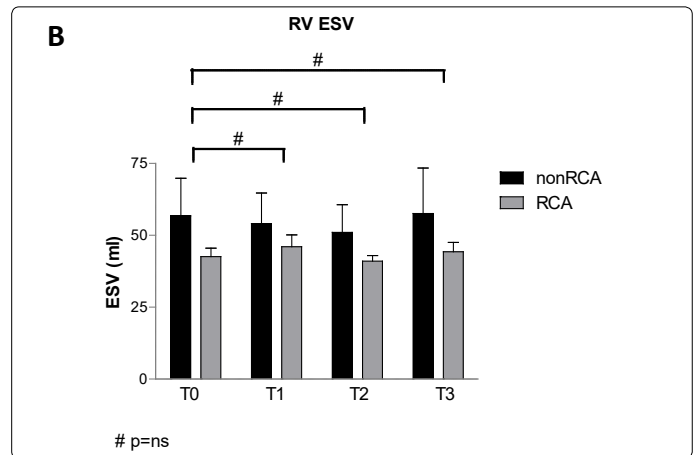
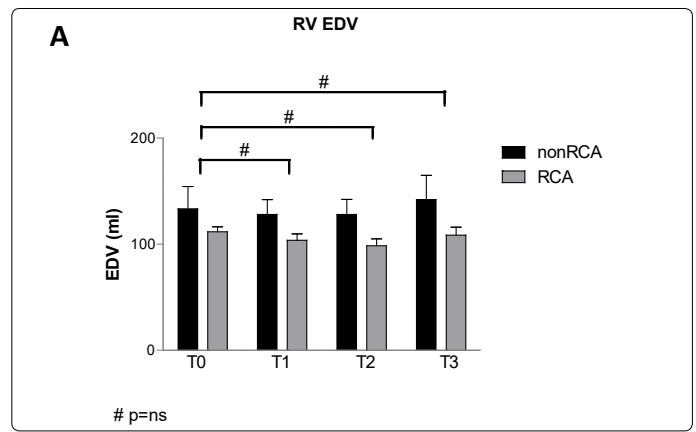
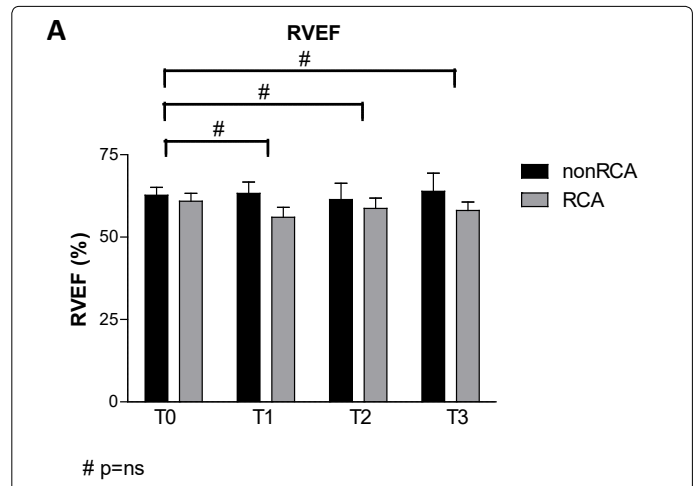


Figure 4: Measurement of the EDV (A), ESV (B) and SV (C) in the RCA-group vs. non-RCA-group showed no significant changes at either time point after CABG. Differences between both groups were not significant either.



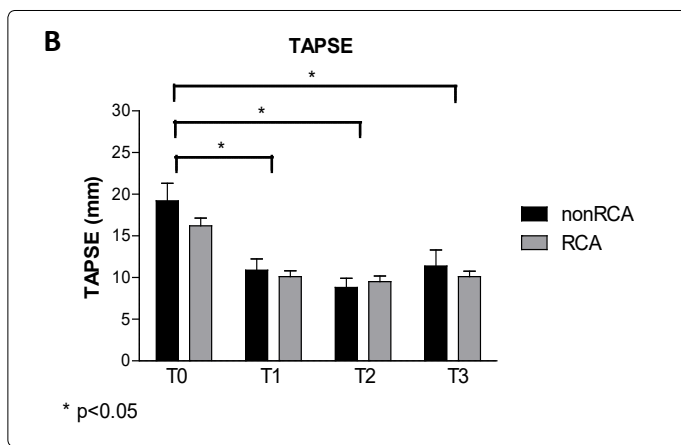


Figure 5: There was no significant change in RVEF, both in the RCA- and the non-RCA-group during the whole follow-up (A), whereas the TAPSE showed a significant decrease in both the RCA-group and the non-RCA-group at T1 compared to T0. TAPSE did not decrease further at T2 and T3 compared to T1 (B). Intergroup differences were not significant.

Discussion

This CMR study showed a significant decrease of TAPSE within 4 months after CABG which remained decreased during the whole long-term follow-up, even though the RVEF and the RV volumes remained unchanged after CABG. Whether the RCA was involved in the revascularization or not did not influence the outcome.

Prior studies have shown conflicting findings about the changes in RV parameters after CABG. An impairment of TAPSE was reported by some of them [6-8,11,19-21]. For example, Unsworth et al reported echocardiographic findings of a decreased longitudinal systolic right ventricular function after CABG, while the RVEF did not significantly change [9]. The impaired TAPSE together with the unchanged RVEF, as we also found in our study, suggests postoperative pericardial adhesion of the RV free wall [6,11,20-23]. Which can occur after partial removal of the parietal pericardium during open heart surgery [11]. Therefore RV systolic function visually seems to be impaired. It is believed that the paradoxical anterior motion of the ventricular septum during systole towards the RV, found after cardiac surgery, may be responsible for the preserved RVEF [6-8,11,13,19,23-25]. The effects of CABG on the RVEF are not entirely clear. While some studies reported an unchanged RVEF after CABG [9,26], others found a significant decrease in the RVEF [13,27-31]. Furthermore, the decreased RVEF found by Stein et al returned towards baseline values after manipulation with pharmacotherapy [31], and Joshi et al as well as Schirmer et al reported an increase in RVEF after CABG [32,33].

The controversies between the aforementioned studies may be caused by differences in techniques used to assess RV function. Due to its high accuracy and reproducibility, cardiovascular MRI has become the tool of choice for non-invasive assessment of cardiac function [1,10,16,34-36], with superior reproducibility in comparison to echocardiography [34]. In clinical practice however, 2D echocardiography still remains the primary imaging modality for assessing ventricular function. A simple and easily obtained echocardiographic

parameter that reflects the function of the RV is the measurement of the TAPSE [10,12,26,37,38]. Longitudinal excursion (as reflected by TAPSE) plays a dominant role in RV systolic function [39]. Thus TAPSE is regarded as a validated indicator of right ventricular function, and has been correlated with RVEF assessed by CMR and radionuclide angiography [1,10,12,37].

In patients who underwent CABG, however, the longitudinal excursion of the RV might be reduced postoperatively and thus relying only on measuring the TAPSE may lead to an underestimation of the actual RV systolic function. Since RV function is a major determinant of quality of life in patients with cardiovascular diseases, measurement of TAPSE after CABG [26] should therefore be carefully interpreted.

Little is known about the long-term effect of CABG on the RV systolic function. The follow-up of the present study (T3: 32±11 months) is, to the best of our knowledge, the longest published so far. Previous studies with follow-up periods of 6-12 months showed conflicting results: some of them demonstrated no further changes [7,8], while others reported alterations in RV systolic function during the follow-up [6,33]. For example, Pegg et al reported a significant decreased RVEF after CABG, which recovered to preoperative levels in 6 months follow-up [13]. However, we could not find any further changes in the RV volumes and ejection fraction, nor a recovery of the decreased TAPSE during the whole follow-up after CABG.

Various explanations of the reported changes in the function of the RV found after CABG have been suggested. Revascularization of a specific coronary artery, for example, was reported in some studies to be the cause of significant differences in RV function after CABG [9,27-32]. Nevertheless, in the present study, revascularization of the RCA did not have any influence on the outcome, which is in agreement with findings from other studies [7,8,13,20,31].

However, our study consisted of 34 patients only. Therefore, lack of significant differences may also be caused by this small sample size, in particular the non-RCA-group. Not every patient had follow-up CMR at all 3 time points. Six out of 7 patients of the non-RCA-group had a CMR at T2, and only 3 patients had CMR at T3, compared to respectively 26 and 24 patients in the RCA-group (table 1). Further assessment in larger cohorts is required to ascertain possible differences in RV volumes and function due to revascularization of the proximal RCA.

The first follow-up measurement was approximately 4 months after CABG, which is longer than the majority of studies. Previous studies reported RVEF changes within the first 6 hours to 6 days after CABG [13,27,29,40]. Since we determined the RV systolic function 4 months after CABG, transient changes immediately postoperatively have not been measured and are therefore unknown. Similarly, the interval between T2 (7±1 months) and T3 (32±11 months) was approximately two years. Transient changes in this interval could not be assessed as well.

Susceptibility artefacts caused by sternal wires after CABG may hamper accurate RV analysis. Previous studies reported

good inter-observer variability when artefacts are absent [10,12,36]. In our study there were only minor susceptibility artefacts from sternal wires in the RV region which did not interfere with accurate measurement. Lastly, many variables may influence changes in RV function after CABG, such as the use of a cardiopulmonary bypass [9,13,19,29-33,41], varying surgical procedures [11,21,22,26] and the effects of the sternotomy itself [9,20]. Future studies are needed to assess all variables influencing RV functional changes after CABG.

Conclusion

The present study shows that CABG does not influence RV systolic function, even after long-term follow-up. In contrast to the RVEF, TAPSE showed a significant decrease after CABG, implying that TAPSE underestimates the actual RV systolic function after CABG. This suggests that TAPSE alone might be an unreliable parameter for determination of right ventricular systolic function after CABG.

Acknowledgements

We would like to thank the participating staff that was involved in acquiring the data and processing the results, but most of all the patients that participated in the trial. No grants were received for this study.

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