ethics committee on human research, and informed consent was obtained. A frequency-domain OCT system, nonocclusive technique, motorized pullback at 20 mm/s, and rotation speed of 100 frames/s were used. Cross-sectional analysis of the OCT images was performed offline. Stent and lumen contours were outlined semi-automatically, and the stent and luminal cross-sectional areas, NIH thickness, NIC, and apposition were evaluated for the entire circumference of the vessel. If tissue/fibrin was found on the stent strut surface, it was calculated as NIH thickness, especially at 2 weeks. The image of each frame was evaluated for the presence of thrombi, defined as unusual masses protruding beyond the stent strut into the lumen on signal attenuation. Strut malapposition was defined as a distance of >100 μm between the stent strut surface and the inner vessel wall. The rates were calculated in each series.

Continuous variables are expressed as mean ± SD. The Friedman 2-way analysis of variance by ranks was used to determine statistical significance ($p < 0.05$).

No serious complications were observed. Representative OCT images and data at 2, 4, and 12 weeks are shown in **Figure 1**. The mean NIH thickness of all struts in the entire lesion increased uniformly from 2 to 12 weeks. NIC rapidly progressed from 2 to 4 weeks and was almost complete at 12 weeks. Malapposition was almost fully resolved at 4 weeks and was completely resolved at 12 weeks. Immediately after percutaneous coronary intervention, edge dissections were observed in 7 stents and intrastent mural thrombi were observed in 5; however, these completely resolved by 4 weeks. Of the 32 stents, 29 were completely covered with neointima after 12 weeks, and few uncovered stent struts remained (0.7% to 1.6%) in the other 3 stents.

We found that the percentage of uncovered struts rapidly decreased by 55.2% from 2 to 4 weeks after implantation, and all were almost completely covered within 12 weeks and that malapposition of struts completely resolved within 4 weeks. The favorable vascular response after Xience stent implantation may have resulted from not only decreased arterial injury and accelerated re-endothelialization related to the thin strut configuration, but also to the biocompatible fluorinated-copolymer with thromboresistant and hemocompatible properties (3). The recently approved second-generation drug-eluting stent includes information in the CE Mark instructions for use indicating a low thrombosis risk after only 1 or 3 months of DAPT. Additionally, based on clinical evidence from drug-eluting stent trials, the American College of Cardiology/American Heart Association guidelines were updated in March 2016 with a Class IIb recommendation that DAPT discontinuation after 3 months may be reasonable in patients with stable ischemic heart disease and high bleeding risk. Our data further support a short-duration DAPT with the use of the Xience stent. NIC of struts and apposition were almost completely achieved at 3 months after implantation. In conclusion, the Xience stent has a favorable, rapid vascular healing process that may justify short-duration DAPT.

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Prognostic Value and Determinants of CMR-Derived Left Atrial Function Assessed in STEMI

In recent years, assessment of left atrial (LA) volumes and function as prognostic markers after acute myocardial infarction (AMI) have gained increasing attention (1). Impairment of both systolic and diastolic left ventricular (LV) function is a frequent finding in patients after AMI and is mainly caused by the amount of infarcted tissue and scar formation (2). The diastolic dysfunction based increase in LA volumes and reduction of LA contractility have been found to be independent predictors for adverse clinical outcome after AMI (1). However, the direct

TABLE 1 Predictors for MACE in Univariate and Multivariate Cox Regression Analysis

	Univariate		Stepwise Multivariate	
	Hazard Ratio (95% CI)	p Value	Hazard Ratio (95% CI)	p Value
Smoker	2.03 (1.06-3.89)	0.033	–	–
Peak creatine kinase	1.01 (1.00-1.02)	0.02	–	–
TIMI-risk score	1.43 (1.27-1.60)	<0.001	1.27 (1.07-1.50)	0.006
LAEF	0.95 (0.93-0.97)	<0.001	0.96 (0.94-0.98)	0.001
LVEF	0.93 (0.91-0.96)	<0.001	–	–

Hazard ratio for categorized LAEF (LAEF ≤53%) was 2.89 (95% CI: 1.05 to 7.93; p = 0.04). All variables with p < 0.05 in univariate analysis was entered into the multivariate model. Myocardial salvage, infarct size and microvascular obstruction were not included into the model as the main purpose of this study was to identify an independent predictive value of the LAEF in comparison to the LVEF.

CI = confidence interval; LAEF = left atrial ejection fraction; LVEF = left ventricular ejection fraction; TIMI = Thrombolysis In Myocardial Infarction.

influence of myocardial damage and reperfusion injury obtained by cardiac magnetic resonance (CMR) on LA function is unknown. The aim of the present study was therefore to evaluate the prognostic value of LA function in the setting of acute reperfused ST-segment elevation myocardial infarction (STEMI) and the relationship between LA performance and established CMR markers of myocardial damage. We hypothesized that impaired LA systolic function is associated with myocardial injury and an increased rate of future cardiovascular events.

The present trial is a CMR substudy of the randomized AIDA STEMI (Abciximab Intracoronary versus intravenously Drug Application in STEMI) multicenter trial. Study design with inclusion and exclusion criteria were described elsewhere (3). No difference was observed regarding the combined primary endpoint of all-cause mortality, recurrent AMI, or new congestive heart failure within 90 days of randomization between both treatment modalities (3). In addition to the main trial, a CMR substudy with consecutive patient enrolment was conducted at 8 sites with proven expertise in CMR imaging. Central blinded image assessment was performed at the CMR core laboratory at the University of Leipzig—Heart Center (4). LA volumes were measured by manual tracing in balanced steady-state free precession images. LA volumes were traced both in long-axis 2-chamber and 4-chamber views in all patients, and final calculation of LA volumes was based on biplane measures. Left atrial ejection fraction (LAEF, corresponding to global LA function) was defined as:

$$\text{LAEF} = \frac{(\text{maximum LA volume} - \text{minimum LA volume}) \times 100}{\text{maximum LA volume}}$$

The primary endpoint was defined as the composite of all-cause mortality, recurrent AMI, or new congestive heart failure (major adverse cardiac events [MACE]) within 1 year following the index event.

From July 2008 to April 2011, 684 patients were included in the present analysis. The median age was 62 years (interquartile range [IQR]: 51 to 71 years) and 523 patients (77%) were male. A total of 36 patients (6%) had history of myocardial infarction prior to the index event. The median time between the index event and CMR was 3 days (IQR: 2 to 4 days). Median LAEF was 53% (IQR: 46% to 59%). LAEF correlated with LVEF ($r = 0.40$; $p < 0.001$) and was inversely correlated with infarct size ($r = -0.35$; $p < 0.001$). Other established CMR markers of myocardial damage (microvascular obstruction, myocardium at risk, hypointense infarct core) showed no significant association with LAEF.

The composite primary endpoint of all-cause mortality, recurrent AMI, or new congestive heart failure at 1 year occurred in 48 patients (7%). Patients with LAEF ≤53% had a significantly poorer 1-year outcome than did those with LAEF >53% (primary endpoint 12% vs. 2%; p log rank <0.001). In multivariate Cox regression analysis, TIMI (Thrombolysis In Myocardial Infarction)-risk score and LAEF were identified as independent predictors for MACE at 1 year (**Table 1**). However, using C-statistics, LAEF did not show an additional prognostic value over and above LVEF in predicting MACE (area under the curve for LAEF = 0.72 vs. for LVEF = 0.69; p = 0.54) or its individual components.

The present trial is the largest and first multicenter study assessing LA function after STEMI and its association with myocardial damage and clinical outcome. The main finding is that LAEF strongly correlates with classical prognostic markers for AMI such as LVEF and infarct size. Impaired LAEF was also found to be an independent predictor for MACE at 1-year follow-up. However, LAEF did not have an additive value in terms of MACE prediction after AMI over and above LVEF. Nonetheless, LAEF can be easy and rapidly assessed, and, therefore, it may be considered as an additional strong prognostic indicator for future cardiovascular events in STEMI survivors.

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Aortic Dilatation in Repaired Tetralogy of Fallot

Although the high prevalence of aortic root dilatation in adults with repaired tetralogy of Fallot (rTOF) is well established (1,2), evidence to guide clinical follow-up and decision making remains sparse.

We sought to define the features, determinants, and rate of progression of aortic dilatation in adults with rTOF using cardiovascular magnetic resonance (CMR).

We retrospectively identified adults with rTOF who had 2 interval CMR scans. Aortic dimensions were measured at sinus, sinotubular junction (STJ), and mid-ascending aortic level at both time points blinded to scan order and other clinical data. Dilatation was defined as diameter >2 SD larger than our published normal CMR aortic dimensions adjusted for age (3).

We retrospectively studied 110 patients (57 male; median age 30.9 years [interquartile range (IQR): 22.9 to 39.4 years]). One patient with aortic valve endocarditis requiring aortic valve surgery was excluded. Forty had a shunt prior to repair (median age at repair 4.5 years [IQR: 2.1 to 9.2 years]); 14 were repaired before 1 year of age; 9 had pulmonary atresia; 24 had right-sided aortic arch; and 11 were successfully treated for systemic hypertension. Twenty-nine patients (27%) had mild and 6 (5%) had moderate aortic regurgitation.

Seventy-six patients (69%) had aortic dilatation. Dilatation was present in 30 patients (27%) at sinus level, in 73 (66%) at STJ level, and in 24 (21%) at ascending aortic level. Thirty- five patients (31%) had normal aortic dimensions (**Figure 1A**). Patients repaired before 1 year of age were less likely to have aortic dilatation at any level compared with the remaining patients ($p = 0.001$).

Male sex and history of palliative shunt were independent predictors of aortic dilatation at any level ($p < 0.0001$ and $p = 0.023$, respectively) and were independent predictors of STJ dilatation ($p = 0.0001$ and $p = 0.033$, respectively). Male sex and pulmonary atresia were independent predictors of aortic sinus dilatation ($p = 0.008$ and $p = 0.0009$, respectively). Male sex, later repair, and pulmonary atresia were independent predictors of ascending aortic dilatation ($p = 0.008$, $p = 0.006$, and $p = 0.0004$, respectively).

During a median interval of 6.3 (IQR: 5.1 to 7.6) years, aortic diameters increased in 47% patients (25% at sinus, 21% at STJ, and 35% at ascending aortic level) at rates between approximately 0.2 to 0.4 mm/year (**Figure 1B**). Even among patients with sinus diameter ≥45 mm at baseline ($n = 5$), there was no increase.

Predictors of aortic diameter increase at STJ level were older age, later repair, and right aortic arch. No predictors of aortic diameter increase at other levels were ascertained.

There were no aorta-related events during follow-up. Aortic regurgitation progressed from mild to moderate in only 2 patients without progressive aortic dilatation.

In conclusion, our data show that aortic dilatation is common, most frequently at STJ level (97% of patients with dilated aorta). Aortic dimensions increased in approximately 50% of patients during