For results see **Table 1**. Mitral annular parameters with the exception of AH, AHICR, tenting height, and tenting volume were larger in men, and all parameters were similar after indexing for BSA. The indexed MVA area for the entire cohort was 5.00 ± 0.95 cm^2/m^2, with an upper reference limit of 6.86 cm^2/m^2. Independent associates of MVA area were as follow: BSA (beta = 0.513; $p < 0.001$); indexed LA volume (beta = 0.287; $p = 0.001$); and indexed LV systolic volume (beta = 0.204; $p = 0.040$). Sex, age, indexed LV mass, indexed LV diastolic volume, ejection fraction, and RV size were not independently associated with MVA area.

Intraobserver variability (coefficient of variation and 95% limits of agreement calculated using the Bland-Altman method): area: 3.3% ± 0.60 cm^2; AP diameter: 2.3% ± 1.3 mm; IC diameter: 2.1% ± 1.6 mm; AH: 4.4% ± 0.6 mm; AHICR: 5.7% ± 1.9%; tenting height: 4.7% ± 0.4 mm; tenting volume: 6.6% ± 0.2 ml; leaflet area: 3.8% ± 0.8 cm^2. Interobserver variability: area: 5.0% ± 0.91 cm^2; AP diameter: 4.9% ± 2.9 mm; IC diameter: 4.0% ± 3.0 mm; AH: 9.8% ± 1.2 mm; AHICR: 8.2% ± 2.8%; tenting height: 6.3% ± 0.6 mm; tenting volume: 10.0% ± 0.3 ml; leaflet area: 7.7% ± 1.5 cm^2.

The present study has defined reference values for the normal MVA in humans using 3D TEE, which will be of use when assessing the mitral valve. Key associates of the MVA area were BSA and indexed LA and LV systolic volumes, which is consistent with previous studies undertaken with TTE (2).

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Intra-Aortic Balloon Pump Optimizes Myocardial Function During Cardiogenic Shock

The intra-aortic balloon pump (IABP) has been the most widely used mechanical device for hemodynamic support in patients with cardiogenic shock complicating acute myocardial infarction (AMI) for more than 40 years. Recently, the use of IABP has been questioned due to limited evidence of clinical value (1). However, selected patients may benefit from IABP and improved methods for patient selection are warranted (2). The cardiomechanical effect of IABP on myocardial function and hemodynamics are mostly studied in experimental and animal models (3,4), but are lacking in humans with cardiogenic shock after AMI. Our aim was to investigate the cardiomechanical and hemodynamic effects of IABP as measured by echocardiographic strain on left ventricular (LV) function in patients with cardiogenic shock after AMI, in order to identify patients who may respond to IABP treatment.

In this 2-center study, 45 patients with cardiogenic shock complicating AMI treated with IABP were included. Echocardiography was performed during IABP counterpulsation (IABP on) and repeated after 5 min of standby position of the IABP (IABP off), with synchronized intra-aortic pressure recordings. Peak systolic strain was measured using speckle-tracking echocardiography. Global longitudinal strain (GLS) and circumferential strain were calculated as average peak systolic strain in a 16-segment LV model. Left ventricular end-diastolic volume, end-systolic volume, stroke volume (SV), and ejection fraction (EF) were calculated using the Simpson biplane method. Patients were classified as IABP-responders if GLS improved (lower values) during IABP counterpulsation compared with during standby. Exclusion criteria were aortic aneurism and/or dissection, severe valvular heart disease, intracardiac shunts as cause of cardiogenic shock, and pregnancy. The study

was designed by the authors and approved by the regional ethics committee. A single observer blinded to patient data and state of IABP analyzed the echocardiographic recordings.

The average age was 63 ± 8 years. Culprit artery was the left anterior descending in 33 patients, circumflex in 8 patients, and right coronary artery in 4 patients. Nineteen patients suffered cardiac arrest prior to IABP insertion. Hemodynamic and cardiomechanic data are given in **Table 1**.

With IABP on, systolic aortic pressures decreased and diastolic aortic pressures increased, but mean aortic pressure did not change significantly. LV volumes decreased during counterpulsation and probably reflect a combined effect of reduced LV afterload due to reduced systolic aortic pressure and improved coronary perfusion due to higher diastolic aortic pressure. GLS, global circumferential strain, and LVEF significantly improved during IABP on, however SV, derived from the Simpson biplane method, did not change significantly. Cardiac output measured as the product of SV (derived from biplane measures) and heart rate was marginally higher during IABP off, mainly driven by a small, but statistically significant change in heart rate.

Analysis of segmental strain demonstrated less systolic longitudinal passive stretch in ischemic segments and improved systolic circumferential strain in nonischemic segments during IABP on.

Thirty patients were classified as IABP-responders and 15 as IABP-non-responders, based on change in GLS. The average baseline LV volumes were significantly smaller among the IABP-responders compared with non-responders, end-diastolic volume 136 ml versus 178 ml and end-systolic volume 90 ml versus 124 ml, $p < 0.05$ for both. The average change in mean aortic diastolic pressure during IABP counterpulsation was significantly larger among IABP-responders (10 mm Hg) than among nonresponders (5 mm Hg), $p < 0.05$. There were no significant differences between responders and nonresponders with respect to baseline LVEF, change in SV, mean aortic pressure, or mean aortic systolic pressure, peak augmentation pressure, or balloon size.

To conclude, in patients with cardiogenic shock complicating AMI, IABP reduced LV volumes and improved global LV function by reducing passive myocardial stretch and by increasing circumferential contraction in nonischemic segments. These cardiomechanical responses to IABP can be monitored bedside by use of strain echocardiography and may be a useful method to verify positive cardiomechanical response to IABP with potential clinical benefit. The results are consistent with earlier studies from animal models and suggest a small, but favorable cardiomechanical response to treatment with IABP counterpulastion in selected patients with cardiogenic shock complicating AMI (3,4).

TABLE 1 Data From Aortic Pressure and Echocardiographic Analysis

	IABP On	IABP Off	p Value
Peak systolic aortic pressure, mm Hg	93.0 ± 10.0	105.0 ± 12.0	<0.01*
Mean diastolic aortic pressure, mm Hg	86.0 ± 9.0	79.0 ± 8.0	<0.01*
Mean aortic pressure, mm Hg	85.0 ± 8.0	84.0 ± 8.0	0.32
End-diastolic aortic pressure, mm Hg	66.0 ± 11.0	72.0 ± 10.0	<0.01*
Heart rate, beats/min	87.0 ± 2.0	90.0 ± 2.0	<0.01*
LV ejection fraction, %	35.0 ± 10.0	33.0 ± 10.0	<0.01*
LV end-diastolic volume, ml	134.0 ± 8.0	156.0 ± 9.0	<0.01*
LV end-systolic volume, ml	88.0 ± 6.0	107.0 ± 7.0	<0.01*
LV stroke volume, biplane, ml	46.0 ± 3.0	49.0 ± 3.0	0.32
Cardiac output, biplane SV · HR, l/min	4.0 ± 0.3	4.5 ± 0.3	0.05
Global longitudinal strain, %	−9.2 ± 4.4	−8.6 ± 4.3	<0.01*
Global circumferential strain, %	−13.6 ± 7.2	−12.2 ± 6.3	<0.01*
Normokinetic segments			
Longitudinal strain, %	−19.0 ± 0.3	−18.7 ± 0.3	0.41
Circumferential strain, %	−23.2 ± 0.4	−20.6 ± 0.4	<0.05*
Hypokinetic segments			
Longitudinal strain, %	−8.5 ± 0.2	−8.0 ± 0.2	0.06
Circumferential strain, %	−8.5 ± 0.4	−8.5 ± 0.4	0.95
Dyskinetic segments			
Longitudinal strain, %	2.6 ± 0.3	3.7 ± 0.4	<0.05*
Circumferential strain, %	2.2 ± 0.9	3.4 ± 0.9	0.40

Values are mean ± SE. *$p < 0.05$ were considered significant.
HR = heart rate; IABP = intra-aortic balloon pump; LV = left ventricular; SV = stroke volume.

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Stasis Mapping Using Ultrasound

A Prospective Study in Acute Myocardial Infarction

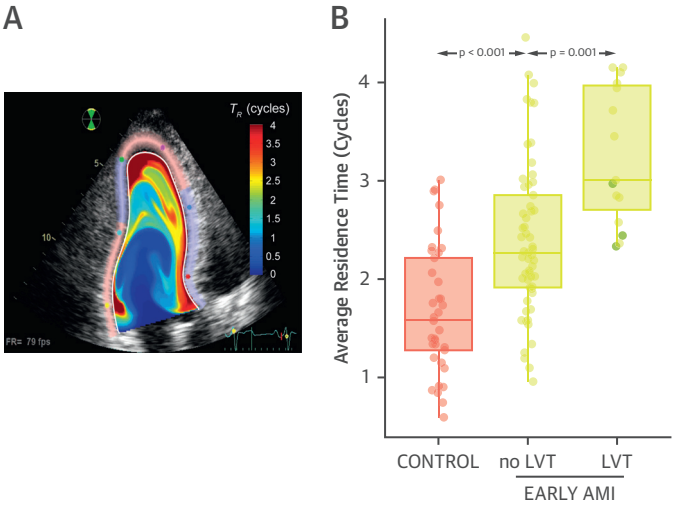
During the subacute phase of acute myocardial infarction (AMI), the incidence of left ventricular thrombosis (LVT) can be as high as 15% to 20%. A method for assessing the risk of LVT would be of particular value in the setting of AMI, because prophylactic anticoagulation must be balanced against the bleeding risk of triple antithrombotic regimens. Recently, it has become possible to obtain patient-specific maps of left ventricular (LV) stasis using conventional echocardiography (1,2). To test the potential of stasis mapping in AMI, we prospectively studied 73 patients admitted to our institution for a first anterior ST-segment elevation AMI from July 8, 2013 to January 2, 2016. Additional inclusion criteria were sinus rhythm, absence of greater than mild aortic regurgitation, LV ejection fraction ≤45% within the first 72 h of AMI onset, stable clinical status, and Killip class less than IV. All patients underwent a full echocardiographic examination both in the early phase (within 72 h of admission) and after 4 to 5 months of follow-up. Contrast ultrasound was used to rule out LVT. For comparison, we studied 37 control subjects of similar age based on the absence of cardiovascular disease, no history of diabetes mellitus or hypertension, and a normal electrocardiogram and echocardiogram. The institutional review board approved the study, and all participants provided written informed consent.

We used color Doppler velocimetry to obtain the unsteady 2-dimensional (2D+t) blood flow field in the apical long-axis view (3). From the 2D+t velocity field, we mapped the residence time in the LV, a magnitude that accounts for the time spent by blood particles in transit through the chamber (**Figure 1A**) (4). To characterize global stasis, we measured the average residence time of the entire blood volume inside the LV after 8 beats (1). Additionally, because local stasis can be particularly meaningful for mural thrombosis, we identified and characterized stagnant regions, defined as regions with all their blood particles having residence time ≥2 cycles.

The AMI patient population consisted of 73 patients, and follow-up data were available in 62 (85%; median follow-up of 4.6 months). Global residence time was >50% higher in the early phase of AMI than in control subjects (2.6 ± 0.9 cycles vs. 1.7 ± 0.9 cycles; $p < 0.001$) (**Figure 1B**). Stagnant regions were larger (44 ± 15% vs. 27 ± 20% of total LV area; $p < 0.001$) and had longer regional residence times (4.4 ± 1.1 cycles vs. 3.8 ± 1.1 cycles; $p = 0.01$) in the early phase of AMI than in control subjects. All global and regional metrics of stagnant regions improved toward control values in the follow-up studies.

LVT was found in 15 patients (20%; blind analysis), 3 of them in the follow-up study. LVT-positive patients showed significantly different stasis metrics than LVT-negative patients in early-phase studies, as demonstrated by a longer global residence time (3.2 ± 0.7 cycles vs. 2.4 ± 0.8 cycles; $p = 0.001$) (**Figure 1B**), larger stagnant regions (52 ± 10% vs. 42 ± 20% of total LV area; $p = 0.004$), and longer regional residence times (5.1 ± 0.9 cycles vs. 4.3 ± 1.0 cycles; $p = 0.02$). Although apical wall motion score (AWMS) was higher in LVT-positive patients (18.4 ± 3.3 vs. 16.0 ± 5.8; $p = 0.04$),

FIGURE 1 Residence Time Maps and Stasis Indices



(A) An example of the residence time (T_R) map in a patient who developed left ventricular thrombosis (LVT). The color scale represents the number of beats that each blood volume element remained in the ventricle. **(B)** Boxplots and scatterplots of average residence time for the control group **(pink)** and patients with acute myocardial infarction (AMI) at the early phase **(dark yellow)** with and without LVT. **Green** dots represent the 3 patients who showed LVT in the follow-up study but not in the early phase.