Off-pump epicardial ventricular reconstruction restores left ventricular twist and reverses remodeling in an ovine anteroapical aneurysm model

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Objective: The loss of normal apical rotation is associated with left ventricular (LV) remodeling and systolic dysfunction in patients with congestive heart failure after myocardial infarction. The objective of the present study was to evaluate the effect of epicardial ventricular reconstruction, an off-pump, less-invasive surgical reshaping technique, on myocardial strain, LV twist, and the potential alteration of myocardial fiber orientation in an ovine model of LV anteroapical aneurysm.

Methods: LV anteroapical myocardial infarction was induced by coil embolization of the left anterior descending artery. Eight weeks after occlusion, epicardial ventricular reconstruction was performed using left thoracotomy under fluoroscopic guidance in 8 sheep to completely exclude the scar. The peak systolic longitudinal/circumferential strains and LV twist were evaluated using speckle tracking echocardiography before (baseline), after device implantation, and at 6 weeks of follow-up.

Results: Epicardial ventricular reconstruction was completed in all sheep without any complications. Immediately after device implantation, LV twist significantly increased (4.18 ± 1.40 vs baseline 1.97 ± 1.92; $P = .02$). The ejection fraction had increased 17% and LV end-systolic volume had decreased 40%. The global longitudinal strain increased from −5.3% to −9.1% ($P < .05$). Circumferential strain increased in both middle and apical LV segments, with the greatest improvement in the inferior lateral wall (from −11.4% to −20.6%, $P < .001$). These effects were maintained ≥6 weeks after device implantation without redilation.

Conclusions: Less invasive than alternative therapies, epicardial ventricular reconstruction on the off-pump beating heart can restore LV twist and systolic strain and reverse LV remodeling in an ovine anteroapical aneurysm model. (J Thorac Cardiovasc Surg 2014;148:225-31)

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Despite improved therapies to enhance survival, congestive heart failure associated with myocardial infarction (MI) remains a major health problem. Throughout disease progression, the formation of scar tissue after MI leads to changes in left ventricular (LV) shape and function. The normal elliptical LV deteriorates, with sphericity and chamber dilation.[1] A LV twisting motion (torsion), in which the apex rotates counterclockwise and the base rotates clockwise about the long axis during systole, is determined by the contractile force and has been as a proposed sensitive marker of LV function. Experimental and clinical studies have demonstrated that apical rotation represents the dominant contribution to LV twist,[2] and decreased apical rotation and loss of LV twist have been shown to be associated with significant LV remodeling, reduced systolic function, and increased filling pressure in patients with congestive heart failure.[3] Surgical ventricle reconstruction (SVR), a technique developed to restore LV shape, has been shown to improve symptoms and, potentially, the prognosis in patients with congestive heart failure after anterior MI with LV aneurysm.[4-8] Few modified SVR techniques have been reported to improve LV twist by re-creating a more elliptical LV geometry.[9,10] However, those SVR procedures require ventriculotomy and cardiopulmonary support. We introduce a novel device (PliCath HF; BioVentrix, San Ramon, Calif) that is designed to treat post-MI scars in a remodeled ventricle by excluding the noncontractile portion of the circumference of the left ventricle through an off-pump, less-invasive, epicardial ventricular reconstruction (EVR) procedure. EVR can achieve the same results as the traditional Dor procedure but less invasively and without the necessity for cardiopulmonary bypass.

In the present study, we evaluated the effect of EVR on myocardial strain and LV twist and the potential alteration

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Abbreviations and Acronyms
EDV = end-diastolic volume
ESV = end-systolic volume
EVR = epicardial ventricular reconstruction
LAD = left anterior descending artery
LV = left ventricular
MI = myocardial infarction
SVR = surgical ventricle reconstruction

of myocardial fiber orientation in an ovine model of LV anteroapical aneurysm.

METHODS

Study Design

The study was conducted in accordance with the *Guide for Care and Use of Laboratory Animals* and was approved by the Institutional Animal Care and Use Committee of the Jack H. Skirball Center for Cardiovascular Research of the Cardiovascular Research Foundation. LV anteroapical acute MI was induced in 8 Dorsett hybrid sheep (weight, 36 ± 3 kg) by coil embolization of the left anterior descending artery (LAD). At 8 weeks after MI creation, EVR was performed using an open chest surgical procedure, and the device was implanted under fluoroscopic guidance. LV performance was evaluated by echocardiography for all sheep before MI creation (naive), before device implantation (baseline), immediately after device implantation, and at 6 weeks of follow-up.

Coronary Coil Embolization–Induced MI

In the present study, LV anteroapical MI was induced by LAD coil embolization. All sheep received intramuscular Telazol (4 mg/kg) injections for induction and were then intubated and mechanically ventilated using 1.5% to 2.5% isoflurane. Heparin (100 U/kg) was injected to maintain an activated clotting time of ≥250 seconds. Under fluoroscopic guidance, the coronary coil (2.0-3.5 mm; Cook Medical, Bloomington, Ind) was delivered to the middle LAD, at a point 40% to 50% of the distance from the apex to the base and to the corresponding diagonals to block the coronary blood flow and induce MI. Coronary angiography was performed to confirm complete and persistent occlusion. All catheters and sheaths were removed 2 hours after LAD occlusion, and the sheep were allowed to recover.

EVR (Device Deployment)

The EVR procedure was performed at 8 weeks after MI creation. The sheep were intubated and anesthetized as previously. The PliCath HF System (BioVentrix, San Ramon, Calif) consists of implantable anchor pairs (including a hinged internal anchor with a tether and a locking external anchor) and a delivery system (Figure 1). The heart was exposed by left thoracotomy, and the LV anteroapical and anterolateral scarred segments were identified using epicardial echocardiography. The device components were pinpointed using fluoroscopy. After intravenous heparin administration (activated clotting time ≥ 250 seconds), EVR was performed. In brief, a custom-made puncture needle, which was connected to a pressure transducer (DTX Plus DT-XX; BD, Franklin Lakes, NJ), was used to puncture the LV free wall and interventricular septum. After confirming the position of the needle tip within the right ventricle using both fluoroscopy and ventricular chamber pressures, a flexible wire was advanced into the right ventricular outflow tract to the pulmonary artery under fluoroscopic guidance. Next, the needle was removed, and a 14F catheter with a dilator was placed over the wire into the right ventricle (Figure 1, *A*). The dilator was removed, and 1 anchor was deployed onto the right ventricular side of the septum (Figure 1, *B*). The wire was removed, and the anchor was pulled back to the septum (Figure 1, *C*). Next, an epicardial anchor was placed on the anterior wall of the left ventricle. Additional anchor pairs were placed to achieve the desired volume reduction (2-3 pairs; Figure 1, *D*). The anchors were plicated together (Figure 1, *E*) with a measured compression force of 2 to 4 N, and contrast was injected to confirm exclusion of the newly formed compartment (Figure 1, *F*). The number of anchor pairs used was determined by the preoperative LV volume and infarct area measurements from echocardiography. The target was either complete scar exclusion or an end-systolic volume reduction (ESV) of >30%. Figure 2 shows the heart before and after device implantation. A chest tube was placed for drainage, and all incisions were then closed using standard methods.

Echocardiography

Echocardiograms were performed at the naive stage, before device implantation (baseline), immediately after device implantation, and at 6 weeks of follow-up. Two-dimensional echocardiography images were acquired with the sheep in the right lateral decubitus position using a 5-MHz probe (iE33; Philips Medical Systems, Bothell, Wash) from standard parasternal long- and short-axis planes. The LV end-diastolic volume (EDV) and ESV were calculated using the Simpson's method, and the ejection fraction (EF) was calculated using a standard formula: ejection fraction = [(EDV − ESV)/EDV] × 100.

Grayscale images for offline speckle tracking echocardiography analysis were acquired at a high frame rate (65-90 frames/s) from standard long-axis and apical 4-chamber views. Short-axis images were acquired at 3 different levels (base, mid, and apical for a total of 16 segments). At least 2 consecutive cardiac cycles were recorded for offline analysis (QLab 6.0 software; Philips).[9] When a cardiac cycle with a good quality image was selected, a region of interest for speckle tracking was defined first at end-diastole using a semiautomated border detection method. The locations of the tracking points extending from the endocardial to epicardial borders were adjusted, and then the segmental myocardial strain curves were automatically generated by the system (Figure 3). The basal and apical rotations were analyzed as previously described,[11] and the data were exported to a spreadsheet program (Excel; Microsoft, Redmond, Wash) to determine the LV peak systolic twist. The LV peak systolic twist was calculated as the net difference in LV rotation at isochronal points between the apical and basal short-axis planes.[12]

For each sheep, the global peak negative systolic circumferential strain was derived from the mean value of all short-axis segments.[12] The peak systolic longitudinal strain in all LV segments (basal septum, mid-septum, apical septum, apex, apical lateral, mid-lateral, and apical lateral) was averaged to obtain a global value (global longitudinal strain).[13] The regional circumferential/longitudinal strain from the basal, middle, and apical level was averaged from the corresponding segments.[14]

Statistical Analysis

Statistical analyses were performed using Statistical Analysis Systems statistical software, version 9.2 (SAS Institute, Cary, NC). All measurements were tabulated as the mean ± standard deviation. The differences within groups at distinct points were assessed using 2-way analysis of variance with the post-hoc test (Bonferroni method). $P < .05$ was considered statistically significant.

RESULTS

Preoperative transthoracic echocardiography confirmed the absence of LV thrombus in all the sheep. EVR was completed in all cases without any adverse hemodynamic consequences. All the sheep recovered from the

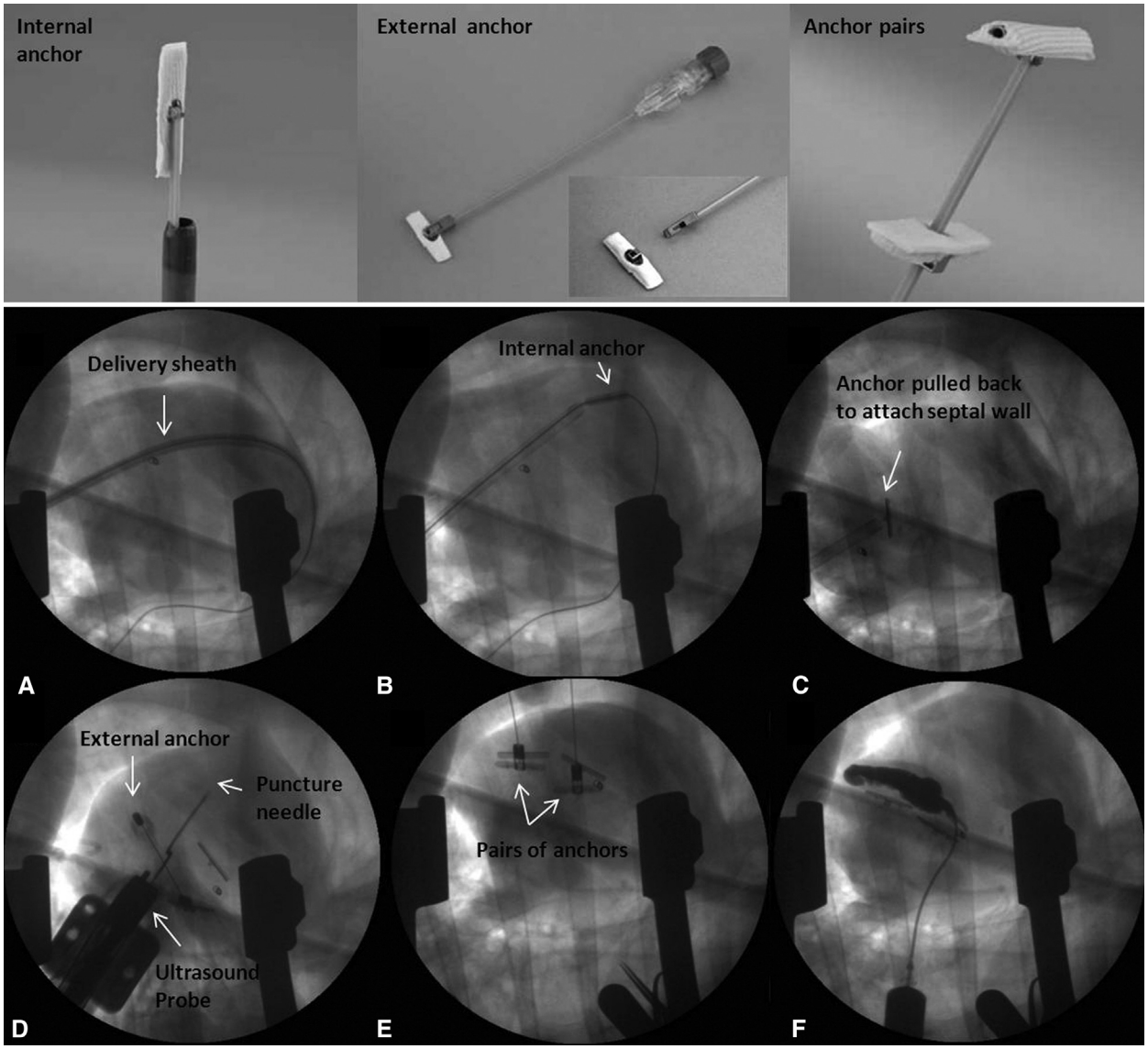

FIGURE 1. *Upper*, The PliCath HF System and, *lower*, the key procedural steps of the off-pump epicardial ventricular reconstruction approach. A, A 14F dilator was passed over the wire into the right ventricle. B, The inner implantable anchor was deployed. C, The inner anchor was pulled back to contact the septum. D, After the epicardial anchor was placed on the anterior wall of the left ventricle, the second puncture site was identified under epicardial echocardiographic guidance. E, Additional anchor pairs were placed and plicated together. F, Contrast injection was performed to confirm exclusion of the newly formed compartment.

surgical procedure and survived to euthanasia with no complications.

Postoperative and follow-up echocardiography confirmed that no communication was present between the restored ventricular chamber and the excluded ventricular space and that no ventricular septal defect was present. The standard 2-dimensional echocardiographic measurements are summarized in Table 1. Immediately after device implantation and compared with baseline, the ejection fraction had significantly increased by 17% ($P = .0005$). The LV EDV and ESV had decreased by 22% ($P = .01$) and 40% ($P = .002$), respectively. The stroke volume was preserved (27.1 ± 6.9 vs baseline 22.7 ± 3.8, $P = .16$). These effects were maintained for ≥6 weeks after device implantation with no redilation.

The parameters from speckle tracking echocardiography are listed in Tables 1 and 2. The apical peak-systolic rotation, LV twist, and global longitudinal and circumferential strain were significantly reduced 8 weeks after the anteroapical infarct. The greatest strain decrease was at the apical level (longitudinal strain, −0.2% ± 1.5% vs naive, −14.2% ± 3.7%; $P < .0001$; circumferential strain, −4.1% ± 4.6% vs naive, −17.2% ± 1.6%, $P < .001$) and in the anteroseptal wall (from −18.0% to −0.3%,

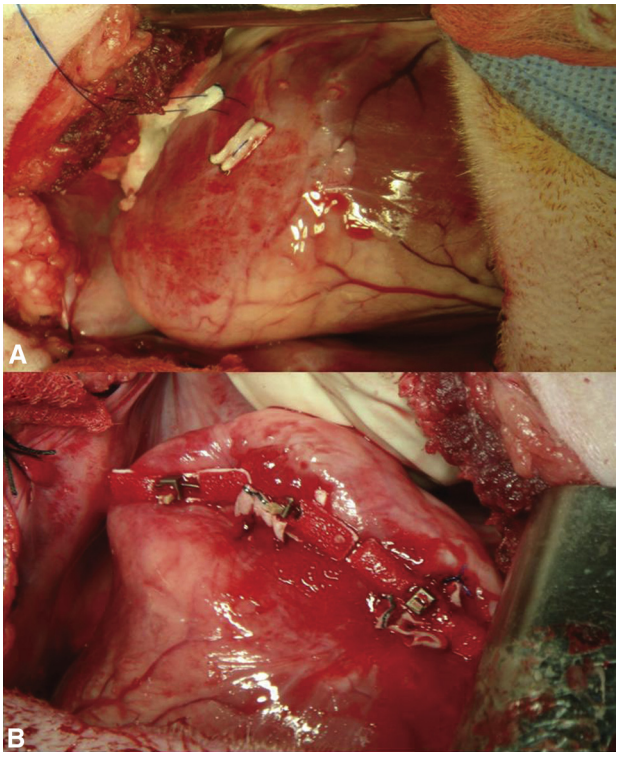

FIGURE 2. Representative heart images before and after device implantation. A, A dilated heart with anterior wall scar before device implantation. B, Approximation of 3 pairs of anchors over a tether resulted in a line of apposition of the lateral wall to the septum.

$P < .0001$). Immediately after device implantation and ventricle reconstruction, the LV twist (4.2° ± 1.4° vs baseline, 1.9° ± 1.9°; $P = .02$) had significantly recovered, although the apical rotation did not dramatically improve (2.3° ± 0.8° vs baseline, 1.3° ± 1.8°; $P = .2$). Compared with baseline, the global longitudinal strains had significantly improved. The middle and apical level circumferential strains were significantly improved after EVR, especially in the inferior lateral wall (from −11.4% to −20.6%, $P < .001$) but the circumferential strain at the basal level had not changed dramatically. Figure 3 shows the strain improvement from before device implantation to 6 weeks after the ventricle reconstruction procedure.

DISCUSSION

The formation of scar tissue after MI leads to various changes in LV shape and function. The left ventricle becomes less elliptical and dilates dramatically.[1] A variety of surgical techniques and devices have been developed to restore the LV shape and improve LV function by interrupting the remodeling process.[15] Large-scale clinical studies have demonstrated that LV reconstruction surgery is safe and effective in terms of a survival benefit and LV functional recovery for patients with ischemic cardiomyopathy.[4-8] Comparable animal studies have revealed mixed results in ventricular volume reduction. Ratcliffe and colleagues[16] reported a trend toward redilation 6 weeks after off-pump anteroapical aneurysm plication in a sheep apical infarct model. Redilation was not seen in the present study. Studies from the same laboratory have demonstrated that, unlike linear repair, patch repair (Dor procedure) significantly reduces the LV volume by 29% without redilation 6 weeks after the procedure.[17] This might be because the linear repair technique cannot exclude the septal scar and also carries the risk of creating a restrictive residual LV cavity, especially in large aneurysms, leading to diastolic dysfunction and LV failure. In the present study, on the off-pump beating heart, we have shown that the PliCath HF device successfully achieves ventricular reconstruction akin to patch repair by circumferentially excluding the nonfunctional scar from both anterior and septal walls through EVR. Significant reductions in the LV chamber size and volumes were seen immediately after device

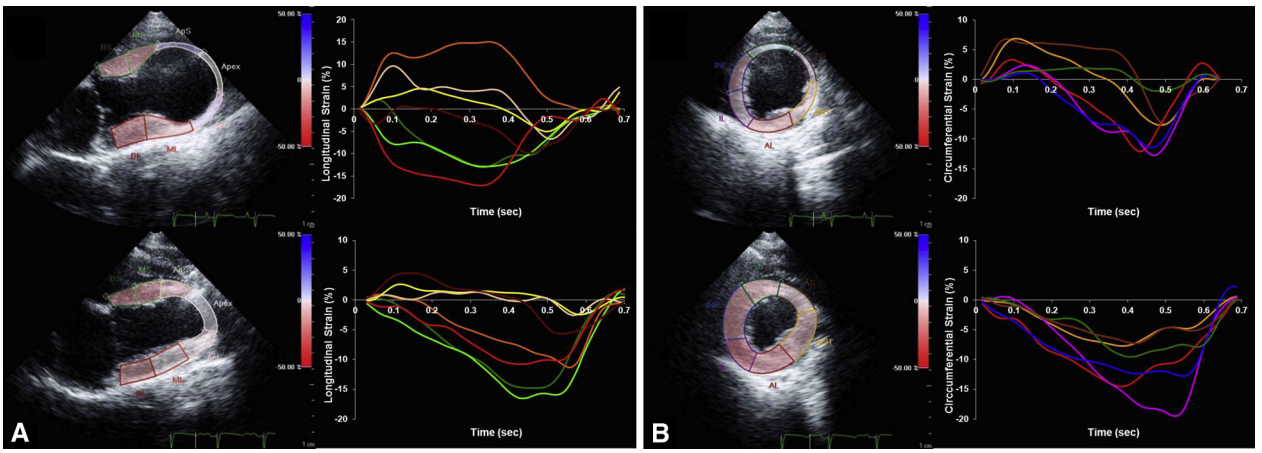

FIGURE 3. Representative images of longitudinal and circumferential strain. A, Longitudinal strain, *upper*, before and, *lower*, 6 weeks after device implantation. B, Circumferential strain, *upper*, before and, *lower*, 6 weeks after device implantation.

TABLE 1. Echocardiography parameters assessed before (baseline, 8 weeks after MI), immediately after device implantation, and 6 weeks after epicardial ventricular reconstruction

Variable	Naive (before MI)	Baseline (before device implantation)	After device implantation	At 6 wk of follow-up
LV EDV (mL)	44.1 ± 7.8	70.4 ± 14.6*	54.7 ± 14.4*,†	57.9 ± 17.6*,†
LV ESV (mL)	19.6 ± 4.0	47.7 ± 13.9*	27.6 ± 6.9*,†	30.8 ± 11.1*,†
SV (mL)	24.5 ± 4.3	22.7 ± 3.8	27.1 ± 6.9	27.1 ± 7.4
EF (%)	55.7 ± 3.0	33.1 ± 7.5*	49.7 ± 3.2*,†	47.3 ± 6.1*,†
Apical rotation (°)	5.2 ± 1.9	1.3 ± 1.8*	2.3 ± 0.9*	2.4 ± 0.8*
LV twist (°)	5.3 ± 1.9	1.9 ± 1.9*	4.2 ± 1.4†	3.8 ± 1.5†
Global longitudinal strain (%)	−13.1 ± 2.2	−5.3 ± 3.0*	−9.1 ± 3.0†	−9.0 ± 2.5†
Basal level	−12.7 ± 4.2	−10.2 ± 3.9	−16.3 ± 6.4	−16.9 ± 5.2
Middle level	−11.7 ± 4.8	−7.9 ± 5.5	−10.1 ± 4.6	−10.4 ± 4.9
Apical level	−14.2 ± 3.7	−0.2 ± 1.5*	−3.7 ± 3.3*,†	−2.8 ± 1.8*
Global circumferential strain (%)	−16.9 ± 2.2	−10.0 ± 2.7*	−14.2 ± 3.2†	−13.4 ± 2.7†
Basal level	−16.9 ± 2.2	−16.4 ± 5.2	−17.5 ± 2.3	−16.1 ± 2.7
Middle level	−16.6 ± 3.3	−10.5 ± 2.1*	−14.8 ± 3.9†	−14.6 ± 3.5†
Apical level	−17.2 ± 1.6	−4.1 ± 4.5*	−11.3 ± 3.2*,†	−10.2 ± 2.2*,†

Data presented as mean ± standard deviation. *MI*, Myocardial infarction; *LV*, left ventricular; *EDV*, end-diastolic volume; *ESV*, end-systolic volume; *SV*, stroke volume; *EF*, ejection fraction. *$P < .05$ versus naive. †$P < .05$ versus baseline.

implantation and maintained ≥6 weeks after device implantation with no redilation.

LV strain and twist are validated indexes of regional and global myocardial function, and 2-dimensional speckle tracking echocardiography provides an accurate method for assessing LV function in patients with heart failure.[18,19] LV twist deformation is generated by contraction of the left ventricle's helically oriented myofibers. The twisting motion of the left ventricle about its long axis results from contraction of the opposite, obliquely oriented epicardial and endocardial fibers, which produce the contemporary clockwise rotation of the base and counterclockwise rotation of the apex, obtaining the torsion of the heart along its long axis. LV torsion represents a critically important mechanism for both ejection and filling.[20,21] After MI, the progressive LV dilation and increased sphericity lead to changes in myofiber orientation and torsional dynamics. Decreased LV torsion has been described in patients with ischemic cardiomyopathy.[2] Therefore, the recovery of LV torsion corresponds to a physiologic rearrangement of the myocardial fibers. In the present study, LAD coil embolization successfully created an anterior wall scar, and all sheep developed heart failure characterized by a dilated LV chamber. The enlarged ventricle resulted in changes in the LV rotational behavior, characterized by significant

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TABLE 2. Regional peak systolic longitudinal and circumferential strain assessed before (baseline, 8 weeks after MI), immediately after device impanation, and 6 weeks after epicardial ventricular reconstruction

Variable	Naive (before MI)	Baseline (before device implantation)	After device implantation	At 6 wk of follow-up
Longitudinal strain (%)				
Basal septum	−12.2 ± 2.8	−12.3 ± 3.8	−19.4 ± 8.4*,†	−17.5 ± 5.8*,†
Basal lateral	−13.2 ± 6.4	−8.0 ± 8.5	−13.3 ± 5.6	−16.5 ± 6.2
Mid-septum	−12.6 ± 6.0	−9.7 ± 6.2	−9.7 ± 4.6	−9.6 ± 5.1
Mid-lateral	−10.8 ± 5.3	−6.2 ± 9.4	−10.5 ± 7.9	−11.2 ± 5.8
Apical septum	−17.9 ± 5.0	−1.0 ± 3.5*	−4.7 ± 4.7*	−3.2 ± 3.1*
Apical lateral	−10.3 ± 5.0	−3.2 ± 3.6*	−2.6 ± 5.6*	−3.5 ± 3.5*
Apex	−14.5 ± 6.9	3.5 ± 3.3*	−3.9 ± 4.9*,†	−1.9 ± 3.5*,†
Circumferential strain (%)				
Anterior septum	−18.0 ± 2.3	−0.3 ± 2.4*	−7.6 ± 7.7*,†	−7.1 ± 4.9*,†
Anterior	−16.6 ± 3.8	−4.4 ± 1.3*	−8.9 ± 5.3*	−9.2 ± 3.5*,†
Anterior lateral	−19.2 ± 5.5	−13.2 ± 5.8	−15.9 ± 2.7	−17.2 ± 3.5
Inferior lateral	−17.0 ± 1.9	−11.4 ± 4.7*	−20.6 ± 4.7†	−17.5 ± 3.1†
Inferior	−15.3 ± 3.8	−9.7 ± 2.9*	−16.2 ± 3.8†	−12.8 ± 2.6
Inferior septum	−15.2 ± 2.8	−5.1 ± 2.8*	−9.1 ± 5.3*	−10.6 ± 4.5†

Data presented as mean ± standard deviation. *MI*, Myocardial infarction. *$P < .05$ versus naive. †$P < .05$ versus baseline.

decreases in systolic apical and LV rotation (Table 2). The evolving techniques of surgical reconstruction of the left ventricle after an anterior MI have progressed toward an increasingly physiologic restoration of the ventricular shape and volume, with increasing attention paid to the multilayered structure of myocardial fibers.[22] We have shown that LV twist was significantly improved after PliCath HF device implantation, resulting in significant improvement in the ejection fraction and contractility.

It has been reported that circumferential strain serves as a short-axis restraint to prevent additional LV geometric expansion and also helps identify patients at risk of future LV remodeling. A reduced circumferential strain has been associated with worse systolic and diastolic LV sphericity indexes. In contrast, the longitudinal strain was more sensitive to myocardial damage and a reduced longitudinal strain was only associated with a worse diastolic index.[23] In the present study, we found that the global longitudinal and circumferential strains were significantly decreased after anteroapical MI creation and scar maturation. A series of large animal studies of SVR using a chronic ovine MI model has been performed with magnetic resonance tagging analysis.[16,17] Zhang and colleagues[17] found that the Dor procedure not only improved the LV ejection fraction but also decreased the average circumferential wall stress by 23.1% and the systolic circumferential strain in nearly all noninfarcted LV regions at 6 weeks after the procedure. Longitudinal shortening increased 2 weeks after the Dor procedure but then decreased to a near preprocedure level at 6 weeks of follow-up.[17] Our present study also showed that the global circumferential strain was significantly improved after EVR, and the greatest regional circumferential strain improvement was found at the apical level and inferior lateral wall. The similar circumferential strain improvement indicated that less-invasive EVR can achieve the same results as the Dor procedure in decreasing the LV sphericity index and improving ventricular systolic function. Furthermore, unlike the study by Zhang and colleagues,[17] the global and regional longitudinal strains in the present study increased immediately after EVR and maintained improvement for $\leq$6 weeks of follow-up. Compared with patch aneurysmorrhaphy, catheter-based ventricular reconstruction without ventriculotomy has had a more positive effect on systolic longitudinal strain and caused less damage to the myocardium. Hung and colleagues[23] reported that reduced longitudinal and circumferential strains were strongly associated with a greater risk of all-cause mortality. Patients with chronic ischemic heart disease and dilated cardiomyopathy treated with traditional surgical and medical management have had a poor prognosis (2-year mortality, 35%-50%).[24] Therefore, the sustained improvements in the global and regional longitudinal and circumferential strains can be extrapolated to report clinical improvements, leading to better New York Heart Association functional class, reduced admissions for congestive heart failure, and improved long-term survival.[25] At the very least, this therapy could attenuate the progressive LV remodeling expected in these patients.

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