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Transatrial Intrapericardial Tricuspid Annuloplasty

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Abstract

OBJECTIVES—This study sought to demonstrate transcatheter deployment of a circumferential device within the pericardial space to modify tricuspid annular dimensions interactively and to reduce functional tricuspid regurgitation (TR) in swine.

BACKGROUND—Functional TR is common and is associated with increased morbidity and mortality. There are no reported transcatheter tricuspid valve repairs. We describe a transcatheter extracardiac tricuspid annuloplasty device positioned in the pericardial space and delivered by puncture through the right atrial appendage. We demonstrate acute and chronic feasibility in swine.

METHODS—Transatrial intrapericardial tricuspid annuloplasty (TRAIPTA) was performed in 16 Yorkshire swine, including 4 with functional TR. Invasive hemodynamics and cardiac magnetic resonance imaging (MRI) were performed at baseline, immediately after annuloplasty and at follow-up.

RESULTS—Pericardial access via a right atrial appendage puncture was uncomplicated. In 9 naïve animals, tricuspid septal-lateral and anteroposterior dimensions, the annular area and perimeter, were reduced by 49%, 31%, 59%, and 24% ($p < 0.001$), respectively. Tricuspid leaflet coaptation length was increased by 53% ($p < 0.001$). Tricuspid geometric changes were maintained after 9.7 days (range, 7 to 14 days). Small effusions (mean, 46 ml) were observed immediately post-procedure but resolved completely at follow-up. In 4 animals with functional TR, severity of regurgitation by intracardiac echocardiography was reduced.

CONCLUSIONS—Transatrial intrapericardial tricuspid annuloplasty is a transcatheter extracardiac tricuspid valve repair performed by exiting the heart from within via a transatrial puncture. The geometry of the tricuspid annulus can interactively be modified to reduce severity of functional TR in an animal model.

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All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

APPENDIX For supplemental material and video, please see the online version of this article.

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Keywords

annuloplasty; structural heart interventions; transcatheter repair; tricuspid regurgitation

Tricuspid regurgitation (TR) is a predictor of mortality, increasing with regurgitation severity and independent of age, biventricular function or dimensions, or pulmonary artery pressure (1). In patients with mitral regurgitation (2) or undergoing left-sided valve surgery (3), TR is associated with heart failure and worse outcome. Persistent TR after left-sided valve surgery predicts poor outcome (4). Isolated tricuspid valve surgery is associated with high mortality (5,6) and, therefore, is seldom justifiable.

Most clinical TR is “functional” and is caused by dilation of the right ventricle and tricuspid annulus (7), resulting from volume or pressure overload. Less commonly, dilation results from primary right ventricular (RV) pathology such as RV infarction or cardiomyopathy. The severity of TR corresponds to tricuspid annulus dimension (8) and is dependent on individual patient preload and afterload conditions, which vary with time.

Surgical annuloplasty restores annular geometry and improves leaflet coaptation (9). Replacement tricuspid valves are more susceptible to thrombosis compared with prostheses in the mitral or aortic positions. Transcatheter repairs have been developed for functional mitral regurgitation (10–12) but not functional TR.

Transatrial intrapericardial tricuspid annuloplasty (TRAIPTA) is a novel transcatheter tricuspid valve repair, guided by x-ray fluoroscopy. The pericardium is accessed via a puncture of the right atrial appendage (RAA) from within. A circumferential implant, which exerts compressive force over the tricuspid annulus, is delivered along the atrioventricular groove within the pericardial space. Tension on the implant is adjusted interactively to modify tricuspid annular geometry and reduce TR. The RAA puncture is sealed using off-the-shelf nitinol closure devices.

In this study, we tested pre-clinical feasibility of transcatheter extracardiac tricuspid annuloplasty in swine and showed that substantial modification of tricuspid annular geometry is achieved and sustained for at least 7 days. In animals with induced functional TR, we showed that TRAIPTA effectively reduces the severity of tricuspid regurgitation. We also explored the feasibility of clinical translation through human cardiac computed tomography (CT) analysis.

METHODS

Procedures were approved by the institutional animal care and use committee. TRAIPTA was performed in 16 Yorkshire swine (mean 48 ± 14 kg) under general anesthesia and mechanical ventilation. Of these, 9 naïve animals were survived for 7 to 14 days, 4 with functional TR were survived for up to 48 days and 3 underwent nonsurvival characterization of TRAIPTA tension and coronary artery protection elements. Hemodynamics and geometry were characterized at each time point by angiography, magnetic resonance imaging (MRI) at

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mid-systole at 1.5-T, and/or 320-detector row CT. Selective coronary angiography was performed before and after TRAIPTA.

TRAIPTA SYSTEM CONFIGURATION

We designed and built a catheter system comprising a delivery device and a permanent implant (Figure 1). The delivery device is formed from an interrupted 0.035-inch nitinol wire, pre-shaped into a self-expanding loop to open inside the pericardium and encircle the heart. The TRAIPTA implant comprises a 2.5-mm diameter hollow tube of braided 0.005-inch nitinol wire. The braided design allows the implant to shorten longitudinally by more than 50%. A braided polyester suture (Ti-Cron size 2, Covidien, Waltham, Massachusetts) runs within the hollow implant with a pre-tied sliding Roeder knot that allows interactive tightening after deployment and that ensures continuous myocardial apposition. The implant is pre-mounted on the delivery device loop and is pre-loaded into a 12-French braided sheath (Cook Medical, Bloomington, Indiana) for deployment.

TRANSATRIAL PERICARDIAL ACCESS

The RAA was engaged from the femoral vein with a 4-French multipurpose catheter (Figure 2) and punctured using the back end of a 0.035-inch guidewire. The catheter was exchanged for a 14-French sheath (Cook Medical); 10 ml of 50% iopamidol/saline was injected into the pericardial sheath to visualize epicardial structures (Online Appendix, Online Figure 1).

DELIVERY AND TIGHTENING OF TRAIPTA IMPLANT

The nitinol loop of the TRAIPTA system opened inside the pericardium to encircle the heart along the atrioventricular groove (Figure 1). The delivery device was then withdrawn, leaving the TRAIPTA implant in place. The suture was tightened interactively during real-time 1.5-T MRI (Aera, Siemens, Erlangen, Germany) to achieve the desired tension.

In animals with functional TR, implant tension was adjusted during intracardiac echocardiography. In 2 naïve animals, we assessed the tension-geometry relationship by applying progressive tension measured with a force meter, 3 times in each animal, during real-time MRI.

CLOSURE OF THE RAA PUNCTURE

After TRAIPTA deployment, pericardial contrast was washed out and 2 mg/kg triamcinolone was infused to impede pericardial adhesions (13). Alongside a 0.014-inch “buddy” guidewire (Ironman, Abbott Vascular, Santa Clara, California) for emergency bailout, a 5- or 6-mm nitinol atrial septal occluder (Amplatzer, St. Jude Medical, St. Paul, Minnesota or Lepu Medical, Bejing, China) closed the appendage puncture site. A final RAA angiogram confirmed hemostasis before all wires and sheaths were withdrawn.

ANIMAL MODEL OF FUNCTIONAL TR

Ten naïve Yorkshire swine underwent procedures to cause right ventricular (RV) volume and/or pressure overload or tricuspid papillary restriction. This included combinations of (1) isolated RV infarction by ethanol infusion into the right coronary artery during balloon protection of the posterior descending artery (n = 8); (2) interatrial septostomy with a 20-

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mm balloon (n = 4); (3) pulmonary artery infusion of 8 ml of 300- to 700-μm polyvinyl-alcohol spheres to induce pulmonary hypertension (n = 5); (4) ethanol needle injection directly into the RV papillary muscles (n = 1); and (5) stenting open the pulmonary valve to cause severe regurgitation (n = 1). Of these 10 animals, 4 were euthanized for intractable ventricular fibrillation or severe right heart failure after RV infarction. The remaining 6 developed a dilated right ventricle, but only 4 of these developed moderate-severe TR after a mean of 117 days. These 4 animals underwent TRAIPTA with intracardiac echocardiography and were survived for up to 48 days.

HUMAN IMAGING FOR SUITABILITY

We studied human cardiac CT angiograms of patients with RV enlargement in the anonymized and delinked National Heart, Lung, and Blood Institute database. This does not constitute human subjects research under US45CFR§46.102(f). Suitability criteria for transatrial access were the presence of a discrete lobe from which to puncture and anterior orientation. Patients were evaluated for the presence of a clearly demarcated atrioventricular groove suitable for TRAIPTA. Epicardial coronary arteries at risk of compression were identified.

STATISTICAL ANALYSIS

Data were analyzed using SPSS 19.0 (IBM, Armonk, New York) and reported as mean ± SD. Differences were examined by 1-way repeated-measures analysis of variance, with Bonferroni post-hoc tests as appropriate. A $p \leq 0.05$ was considered significant.

RESULTS

TRAIPTA was performed in a total of 16 animals (9 naïve survived for 7 to 14 days, 4 with functional TR survived up to 48 days, and 3 were not survived to characterize tension-geometry and coronary protection elements). Transatrial pericardial access was successful in a single pass, and the TRAIPTA device was consistently delivered to the atrioventricular groove in 16 of 16 animals. These 2 stages required <10 min to perform (Online Video).

SURVIVAL EXPERIMENTS IN 9 NAÏVE ANIMALS

Significant tricuspid annular geometric reduction was achieved by tightening the TRAIPTA implant (Table 1, Figures 3 and 4), with 49% ($p < 0.001$) reduction in the septal-lateral dimension and 31% ($p < 0.001$) in the anteroposterior dimension. The tricuspid annular area and perimeter were reduced by 59% ($p < 0.001$) and 24% ($p < 0.001$), respectively. Tricuspid leaflet coaptation length (Figure 4) was significantly increased by 53% ($p < 0.001$). Lesser mitral annular geometric change was observed with a mean reduction in the septal-lateral and anteroposterior dimensions of 15% ($p < 0.05$) and 15% ($p < 0.05$), respectively. No significant hemodynamic changes and no sustained arrhythmias were observed (Online Table 1). Coronary artery compression was not seen in any animal (Online Figure 2).

The RAA puncture was consistently closed using a nitinol closure device in all 9 animals. Post-procedure MRI demonstrated small pericardial effusions (mean, 46 ± 44 ml), but there

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was no evidence of tamponade (Online Table 1). No animal required pericardial drainage. At follow-up (mean, 9.7 days; range, 7 to 14 days), the TRAIPTA implant remained in place without migration, and pericardial effusions had resolved. On necropsy (Figure 5), the implant was encased in fibrous tissue and fused to the myocardium along its entire course. There were no adhesions between the visceral and parietal pericardial layers.

SURVIVAL EXPERIMENTS IN 4 ANIMALS WITH FUNCTIONAL TR

TRAIPTA successfully reduced the severity of TR by intracardiac echocardiography (Figure 4). This was maintained up to 48 days of follow-up. After euthanasia, the TRAIPTA implant remained intact in the correct anatomic position on necropsy and was completely endothelialized and fused to the myocardium (Figure 5).

NONSURVIVAL EXPERIMENTS IN 3 NAÏVE ANIMALS

Implant tension correlated with changes in annular geometry (Figure 3), and the feasibility of coronary artery protection was demonstrated (Online Figure 2), although, in fact, no coronary compression was observed in any of these experiments.

ANATOMIC SUITABILITY IN HUMANS

We reviewed 14 cardiac CT angiograms for anatomic suitability from patients with an intracardiac shunt and a dilated right heart. RAA morphology and anatomic position were consistent across subjects. All 14 met both suitability criteria for transatrial pericardial access. Thirteen of 14 patients (93%) had a clearly defined atrioventricular groove. Of those, all had at least 1 epicardial coronary artery that crossed the projected course of the TRAIPTA implant.

DISCUSSION

To our knowledge, this is the first description of a transcatheter tricuspid valve repair and the first mechanical intervention accomplished via a right atrial exit to the pericardium. In contrast with the mitral valve, where primary leaflet or subvalvular apparatus pathology often contributes to regurgitation, most symptomatic TR is caused by annular dilation with intact valvular apparatus. For this reason, annuloplasty is the preferred surgical repair. We showed a dose-response relationship between TRAIPTA tension and tricuspid geometry and leaflet coaptation; we showed that TRAIPTA treats functional TR in a clinically relevant animal model, that the right atrial exit port is reliably closed, that the procedure is rapid and reproducible, and that eligible humans appear to have suitable anatomy.

TRANSATRIAL PERICARDIAL ACCESS

Transatrial pericardial access was first described by Verrier et al. (14) to sample pericardial fluid, drain effusions, and deliver drugs. A number of interventional (15) and electrophysiology procedures require pericardial access, which is usually obtained through a “dry” subxiphoid puncture and which risks hemopericardium from right ventricular or coronary artery laceration.

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TRAIPTA is the first mechanical intervention performed by exiting the heart from within via a transatrial puncture. This approach provides direct access to the base of the heart in the plane of the atrioventricular groove. The puncture was sealed with off-the-shelf nitinol closure devices, and hemostasis was consistently achieved in all animals. However, right atrial pressures were low in these animals, and the risk of bleeding could be higher in patients with severe TR or coagulopathy. Small post-procedural pericardial effusions were observed but without tamponade, whereas in patients, a temporary pericardial drain would likely be placed. Complete resolution of these effusions was observed in all animals at follow-up, even with the permanent TRAIPTA implant in situ. Our current technique requires inversion of the appendage to ensure proper nitinol closure device positioning. We believe that the device redistributes force adequately to protect against appendage injury.

INTRAPERICARDIAL TRICUSPID ANNULOPLASTY

Aortic and mitral valve disease commonly coexist with tricuspid valve disease. Despite the adoption of transcatheter aortic and mitral valve interventions, there remains an unmet need for transcatheter tricuspid valve repair (16), particularly because untreated TR after aortic or mitral surgery confers a poor outcome. Percutaneous orthotopic (17,18) or heterotopic (19,20) prosthetic tricuspid valve replacement has been described, but these risk thrombosis without anticoagulation. TRAIPTA would not require anticoagulation because the implant is extravascular, although some patients may have other indications (e.g., atrial fibrillation). Transcatheter tricuspid replacement valves are contraindicated in the presence of transvenous pacing or defibrillator leads, but TRAIPTA is possible because it is extravascular. Transatrial exit is possible in the presence of right atrial pacing leads because the puncture can be performed distant to the lead insertion point. TRIAPTA may also be a treatment strategy for pediatric and adult congenital systemic single right ventricle patients with TR.

In this study, we correlated implant tension with reduction in tricuspid annular dimensions, especially in the septal-lateral dimension, which is the main axis of annular dilation (7) and the principal target for surgical annuloplasty. The magnitude of geometric modification easily exceeded ranges reported for surgical annuloplasty (21). We observed lesser geometric modification of the mitral annulus, likely reflecting the higher pressures and increased myocardial thickness of the left heart. Nevertheless, this technique could potentially be adapted to treat both atrioventricular valves. Unlike surgery, TRAIPTA can be performed in the beating heart and titrated in real time under varying loading conditions imposed by hemodynamic provocations such as exercise and volume. Importantly, we found geometric change was maintained over the follow-up period.

ANIMAL MODEL

TR has been induced in animals by surgical annular disruption (22,23), but there are no transcatheter large animal models of functional TR. In this study, we found that to create TR required multiple insults, including volume and pressure overload, as well as considerable time to allow the right ventricle to remodel.

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STUDY LIMITATIONS AND POTENTIAL FAILURE MODES

TRAIPTA requires the pericardial space to be free of adhesions, which could impede implant delivery. This likely precludes TRAIPTA in patients with previous pericardiotomy or pericarditis. However, the technique might be useful in conjunction with transcatheter aortic valve replacement and/or mitral valve repair. Because of its circumferential position around the heart, 1 potential complication is coronary artery and coronary sinus compression. At the level of tension exerted to achieve 40% reduction in the tricuspid septal-lateral dimension, we did not observe any coronary artery or sinus compression (Online Figure 2), albeit in animals without severe RV hypertension. To address this potential complication, we demonstrated that a protection element could be incorporated into the TRAIPTA implant (Online Figure 2) and that its position along the TRAIPTA implant was adjustable interactively in situ. This bridgelike element, originally developed in our lab for mitral cerclage annuloplasty (12), directs compressive force away from an entrapped coronary artery or sinus. Finally, in common with all permanent implants, potential failure modes are implant migration and tissue erosion. We did not observe any implant migration, and on necropsy, the TRAIPTA device was fully adhered to the myocardium with no macroscopic evidence of tissue erosion (Figure 5A). Similar macroscopic findings were observed for the nitinol closure device at the right atrial puncture site. No pericarditis or pericardial adhesions were observed, perhaps because we administered intrapericardial glucocorticoids after deployment. These finding suggests a very low likelihood of late device migration or tissue erosion, but this will be evaluated in a future long-term animal study.

CLINICAL TRANSLATION AND SUITABILITY OF HUMAN ANATOMY

RAA morphology was suitable for transatrial pericardial access in all patients, and most had a clearly defined atrioventricular groove suitable for TRAIPTA. Most patients also had epicardial coronary arteries crossing the projected course of the TRAIPTA implant, and we therefore anticipate that protection elements would be required in human implementation (Online Figure 2).

CONCLUSIONS

We report a novel transcatheter tricuspid annuloplasty technique using transatrial pericardial access and pericardial deployment of a permanent implant. We demonstrate interactive adjustment of tricuspid annular and leaflet geometry in naïve swine, comparable to that achieved with surgical annuloplasty. In animals with functional tricuspid regurgitation, this geometric adjustment reduces the severity of regurgitation.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

ACKNOWLEDGMENTS

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ABBREVIATIONS AND ACRONYMS

CT	computed tomography
MRI	magnetic resonance imaging
RAA	right atrial appendage
RV	right ventricular
TR	tricuspid regurgitation
TRAIPTA	transatrial intrapericardial tricuspid annuloplasty

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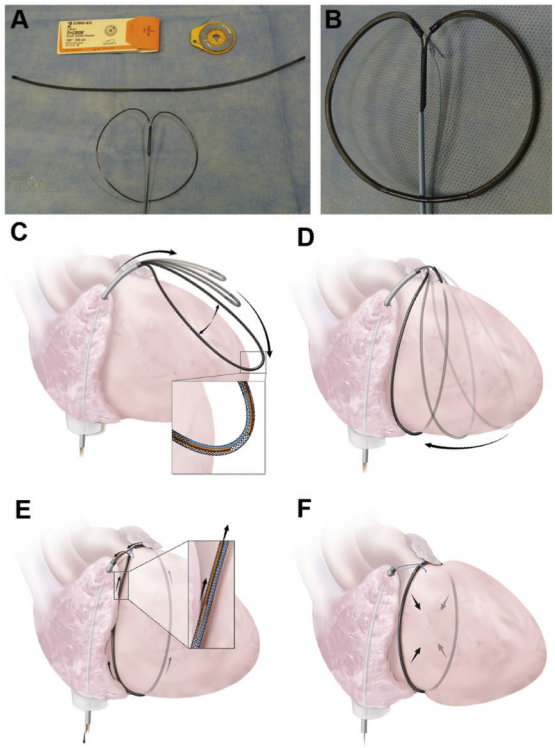

FIGURE 1. TRAIPTA System and Procedural Steps

(A) Braided suture (packaging and spool), transatrial intrapericardial tricuspid annuloplasty (TRAIPTA) implant and delivery device. **(B)** TRAIPTA implant loaded onto delivery device. The suture is housed within the hollow TRAIPTA implant with a pre-tied Roeder sliding knot. **(C)** The system is advanced into the pericardium through the right atrial appendage. **(D)** The nitinol delivery device ensures that the system opens into a loop inside the pericardium and reaches the atrioventricular groove. **(E)** The delivery device is withdrawn. **(F)** The implant is tightened. See Online Video for demonstration of TRAIPTA procedure. TRAIPTA = transatrial intrapericardial tricuspid annuloplasty.

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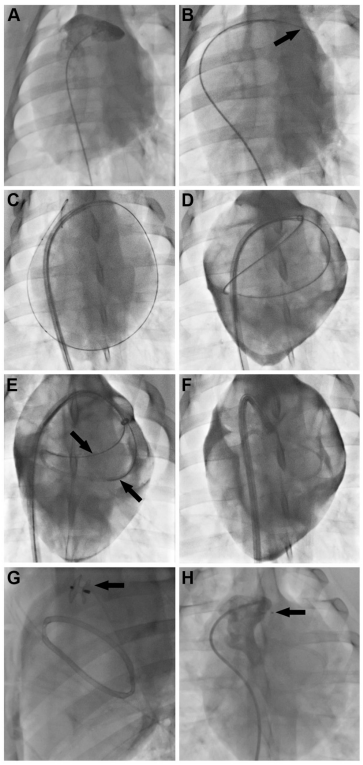

FIGURE 2. TRAIPTA Procedure In Vivo

(A) Right atrial appendage (RAA) angiogram. **(B)** RAA puncture with 0.035-inch guidewire. **(C)** A 14-French sheath introduced into the pericardium. **(D)** The transatrial intrapericardial tricuspid annuloplasty (TRAIPTA) implant is deployed around the heart within the contrast-filled pericardial space. **(E)** Delivery system (**arrows**) withdrawal leaving the TRAIPTA implant in the atrioventricular groove. **(F)** The TRAIPTA implant is tightened by a sliding Roeder knot. **(G)** Closure of the RAA puncture with a nitinol closure device **(arrow)**. **(H)** RAA angiogram 7 days later (**arrow** indicates the closure device).

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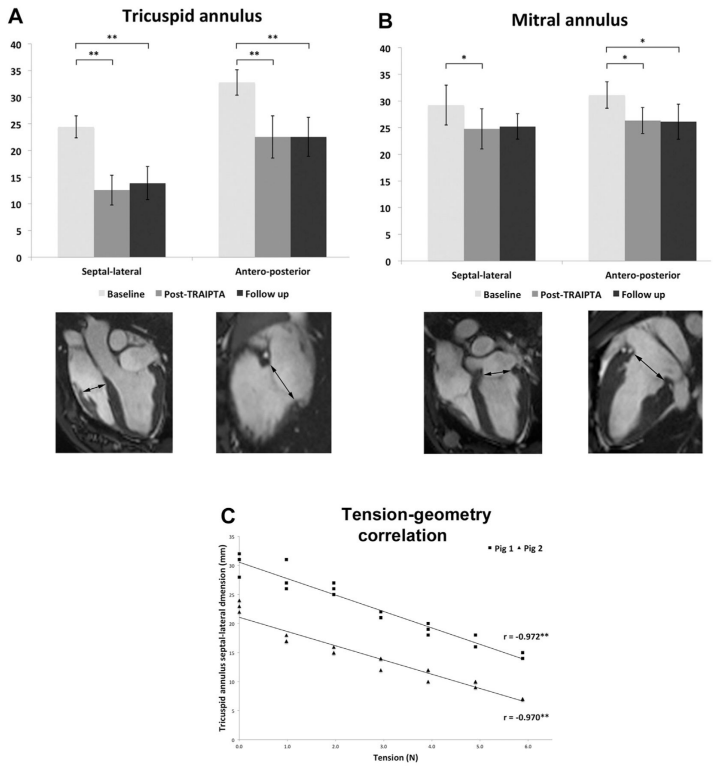

FIGURE 3. Geometric Change Resulting From Annuloplasty

(A) Mean tricuspid valve annular dimensions in 5- and 2-chamber MRI views. Arrows denote measured annular dimensions. **(B)** Mean mitral valve annular dimensions in 4- and 2-chamber magnetic resonance imaging views. **(C)** The transatrial intrapericardial tricuspid annuloplasty (TRAIPTA) implant was tensioned using a force meter attached to the suture. *$p < 0.05$; **$p < 0.001$.

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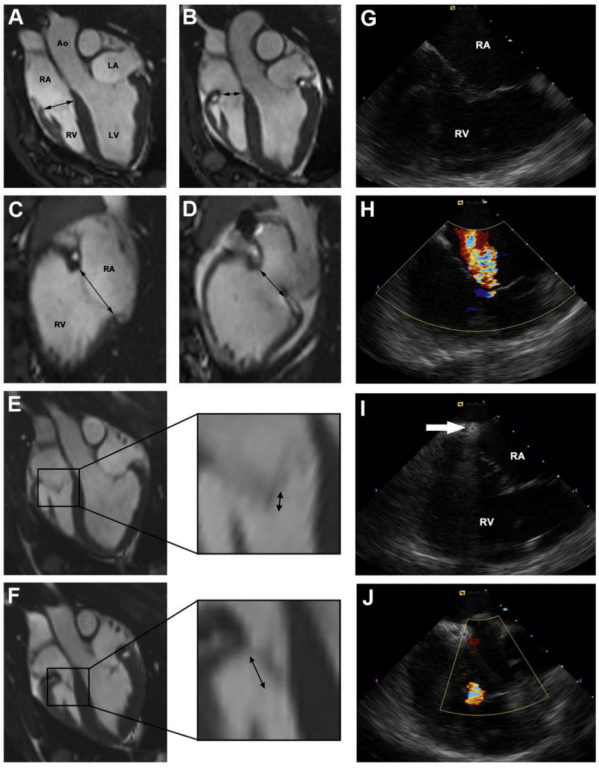

FIGURE 4. Imaging of Tricuspid Valve and Annulus Before and After Annuloplasty

(A) Magnetic resonance imaging 5-chamber view at baseline. **Arrow** denotes tricuspid annulus. **(B)** A 5-chamber view after transatrial intrapericardial tricuspid annuloplasty (TRAIPTA). **(C)** A 2-chamber view at baseline. **(D)** A 2-chamber view after TRAIPTA. **(E)** Tricuspid leaflet coaptation at baseline. **Arrow** denotes coaptation length. **(F)** Tricuspid leaflet coaptation after TRAIPTA. Arrow denotes coaptation length. **(G)** Intracardiac echocardiography of the tricuspid valve before deployment of the TRAIPTA implant. **(H)** Color Doppler shows a central jet of moderate-severe tricuspid regurgitation (peak velocity 1.8 m/s). **(I)** Tensioned TRAIPTA implant at the level of the tricuspid annulus **(arrow)**. **(J)** Color Doppler showing significant reduction in tricuspid regurgitation severity after TRAIPTA. Ao = aorta; LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle.

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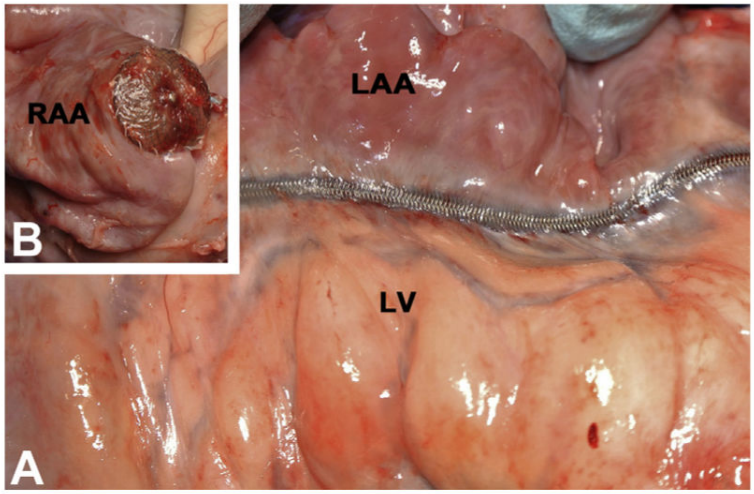

FIGURE 5. Necropsy

(A) Transatrial intrapericardial tricuspid annuloplasty (TRAIPTA) implant at 48 days of follow-up endothelialized and fused to the myocardium along the atrioventricular groove. **(B)** Right atrial appendage (RAA) puncture site at 7 days of follow-up, sealed with nitinol closure device. LAA = left atrial appendage; LV = left ventricle.

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TABLE 1

MRI Findings (N = 9)

	Baseline	Post-TRAIPTA	Follow-up (mean, 9.7 days)
Cardiac chambers			
RA area, cm^2	11.8 ± 1.6	12.0 ± 1.2	11.4 ± 2.1
RV end-diastolic volume, ml	84.5 ± 12.3	65.6 ± 20.9	73.9 ± 20.4
RV end-systolic volume, ml	42.5 ± 14.8	35.8 ± 16.9	36.8 ± 17.8
RV stroke volume, ml	42.0 ± 7.5	29.9 ± 5.9*	37.1 ± 6.5
RV ejection fraction, %	50.8 ± 11.6	47.6 ± 10.3	52.1 ± 10.4
LA area, cm^2	12.3 ± 2.6	11.1 ± 1.1	12.1 ± 1.9
LV end-diastolic volume, ml	93.3 ± 14.7	66.9 ± 13.3*	79.2 ± 8.7
LV end-systolic volume, ml	51.5 ± 13.9	36.6 ± 11.3*	41.9 ± 5.6
LV stroke volume, ml	41.8 ± 8.8	30.3 ± 5.6*	37.3 ± 8.0
LV ejection fraction, %	45.3 ± 9.6	46.1 ± 8.3	46.8 ± 6.5
TV annulus‡			
TV septal-lateral, mm	24.4 ± 2.1	12.6 ± 2.8†	13.9 ± 3.1†
TV anteroposterior, mm	32.8 ± 2.4	22.6 ± 4.0†	22.6 ± 3.6†
TV area, cm^2	11.8 ± 1.9	5.3 ± 1.8†	4.4 ± 1.5†
TV annular perimeter, cm	13.2 ± 1.1	9.6 ± 2.4†	9.8 ± 1.2†
TV leaflet coaptation length, mm	3.1 ± 0.9	6.8 ± 0.8†	6.1 ± 0.8†
MV annulus‡			
MV septal-lateral, mm	29.2 ± 3.7	24.8 ± 3.8*	25.2 ± 2.4
MV anteroposterior, mm	31.1 ± 2.5	26.3 ± 2.4*	26.1 ± 3.3*
MV area, cm^2	9.3 ± 2.0	6.0 ± 1.3†	5.6 ± 1.2†
MV annular perimeter, cm	11.9 ± 1.1	9.3 ± 1.2†	9.1 ± 0.9†
Pericardium			
Pericardial effusion, ml	0.0 ± 0.0	45.8 ± 43.7†	0.0 ± 0.0

Values are mean ± SD.

* $p < 0.05$.

† $p < 0.001$ compared with baseline.

‡ Annular measurements were performed in mid-systole.

LA = left atrium; LV = left ventricle; MRI = magnetic resonance imaging; MV = mitral valve; RA = right atrium; RV = right ventricle; TRAIPTA = transatrial intrapericardial tricuspid annuloplasty; TV = tricuspid valve.