

Tricuspid Annular Geometry and Strain After Suture Annuloplasty in Acute Ovine Right Heart Failure

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Background. Tricuspid valve repair using suture annuloplasty is thought to be more physiologic, but the effect of annular reduction on annular geometry and motion is unknown. We set out to investigate the effect of DeVega suture annuloplasty (DV) on tricuspid annular geometry and dynamics during acute right heart failure (RHF).

Methods. Ten adult sheep underwent implantation of sonomicrometry crystals around the tricuspid annulus and on the right ventricle; pressure transducers were placed in right ventricle, left ventricle, and right atrium. RHF was induced by a combination of 500 mL volume infusion, posterior descending artery occlusion, and pulmonary artery constriction. Hemodynamic, echocardiographic, and sonomicrometry data were acquired at baseline, with RHF, and after two progressive (8 to 10 mm) DV suture cinches (DV-1, DV-2) during RHF. Annular size, geometry, and dynamics were determined from crystal coordinates.

Results. Combination of volume infusion, ischemia, and pulmonary hypertension resulted in acute RHF and

significant functional tricuspid regurgitation grade (0.5 ± 0.5 versus 2.7 ± 0.8 , $p < 0.001$). Annular area increased with RHF from $700 \pm 98 \text{ mm}^2$ to $801 \pm 128 \text{ mm}^2$ ($p < 0.001$). DV-1 and DV-2 reduced annular area to $342 \pm 88 \text{ mm}^2$ and $180 \pm 57 \text{ mm}^2$ while reducing regurgitation grade to 1.2 ± 0.4 and 0.4 ± 0.5 , respectively (all $p < 0.001$ versus RHF). Tricuspid annular area contraction was $12\% \pm 7\%$, $10\% \pm 6\%$, and $12\% \pm 6\%$ for RHF, DV-1, and DV-2, respectively ($p = 0.25$) and annular height was $4.9 \pm 2.0 \text{ mm}$, $5.6 \pm 1.4 \text{ mm}$, and $5.5 \pm 1.7 \text{ mm}$ ($p = 0.43$). Mean transvalvular gradient was $1.3 \pm 0.7 \text{ mm Hg}$ and $2.0 \pm 1.0 \text{ mm Hg}$ with DV-1 and DV-2, respectively.

Conclusions. During acute ovine RHF, DeVega annuloplasty successfully treated tricuspid regurgitation and preserved normal tricuspid annular dynamics and geometry. These data may lead to more physiologic tricuspid reparative techniques.

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Functional tricuspid regurgitation (FTR) is a common clinical disease that predicts poor clinical outcomes and portends reduced long-term survival [1, 2]. The observed valvular insufficiency is considered functional [3] and thus not intrinsic to the tricuspid apparatus but a result of valvular/ventricular remodeling due to mostly left-sided valvular lesions, cardiomyopathy, pulmonary hypertension, or chronic atrial fibrillation. The mere clinical presence of FTR increases mortality in the general population [4] and in every subgroup of cardiac surgical candidates [5]. FTR is usually associated with tricuspid annular dilation, right ventricular failure, and leaflet tethering with surgical repair centered on annular reduction with prosthetic ring or suture annuloplasty [6].

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Because of inconsistent long-term results with suture annuloplasty, ring repair is currently favored [7]. A recent study from the Mayo Clinic [8], however, revealed equivalent long-term results and suggests that abandonment of suture annuloplasty may be premature. Suture annuloplasty offers lower cost of repair, no artificial implant, faster execution of the procedure, and potentially a more physiologic repair. The most commonly performed suture annuloplasty technique is the one initially proposed by DeVega [9] and later modified [10] to two layers of polypropylene suture placed circumferentially around the tricuspid annulus. Several further modifications of the original technique have been proposed, chiefly to decrease the incidence of suture-related tissue tearing [11]. The central role of any DeVega-like suture annuloplasty is focused on annular reduction, yet there is a paucity of data on how these techniques affect the geometry and dynamics of the tricuspid annulus. Such knowledge would bear fruit in designing more

physiologic reparative techniques, leading to potentially improved patient outcomes.

Although several animal models of TR have been described in the literature [12–14], concurrent right ventricle (RV) dysfunction that triggers tricuspid insufficiency is often omitted. Utilizing an acute ovine model of right heart failure and FTR [15], we set out to investigate the effect of the modified semicircular DeVega (DV) suture annuloplasty on valvular competence and tricuspid annular 3-dimensional (3D) geometry and dynamics. In addition to standard clinical metrics we also used continuous metrics to describe procedure-induced annular strains.

Material and Methods

All animals received humane care in compliance with the Principles of Laboratory Animal Care formulated by the National Society for Medical Research and the Guide for Care and Use of Laboratory Animals prepared by the National Academy of Science and published by the National Institutes of Health. The study protocol was approved by our local institutional animal care and use committee. The study utilized 14 animals. Two animals died before data acquisition (acute respiratory failure; left ventricle [LV] injury during sternotomy) and in an additional 2 animals the data were rendered unusable because of sonomicrometry crystal displacement. Data from the remaining 10 animals were analyzed and are presented here.

Surgical Preparation

Ten healthy Dorset castrated male sheep (52 ± 3 kg) were anesthetized with propofol (2 to 5 mg/kg intravenous [IV]), intubated, and mechanically ventilated. General anesthesia was maintained with inhalational isoflurane (1% to 2.5%) and fentanyl (5 to $20 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$). The operative procedure was performed through a sternotomy. On the beating heart the right atrium was opened, and a DeVega annuloplasty suture was placed around the tricuspid annulus. DV semicircular annuloplasty was performed with two 2-0 polypropylene sutures in 2 layers around the tricuspid valve orifice and anchored with pledgets at the anteroseptal commissure and the mid-septal annulus (Fig 1). The suture was externalized through the anteroposterior (A-P) annulus to a tourniquet. Six 2-mm sonomicrometry crystals (Sonometrics Corp, London, Ontario, Canada) were implanted with 5-0 polypropylene sutures around the tricuspid annulus (Fig 2). Crystal electrodes were exteriorized through the right atriotomy.

Four crystals were implanted on the right ventricular myocardium as shown in Figure 2. Silicone elastomer snare was placed around the posterior descending artery branch of the circumflex coronary artery close to the atrioventricular groove. Pressure transducers (PA4.5-X6; Konigsberg Instruments, Inc, Pasadena, CA) were placed in the LV, RV, and right atrium. A 24-mm pulmonary artery pneumatic occluder (In Vivo Metric, Healdsburg, CA) was placed around the main pulmonary artery. After

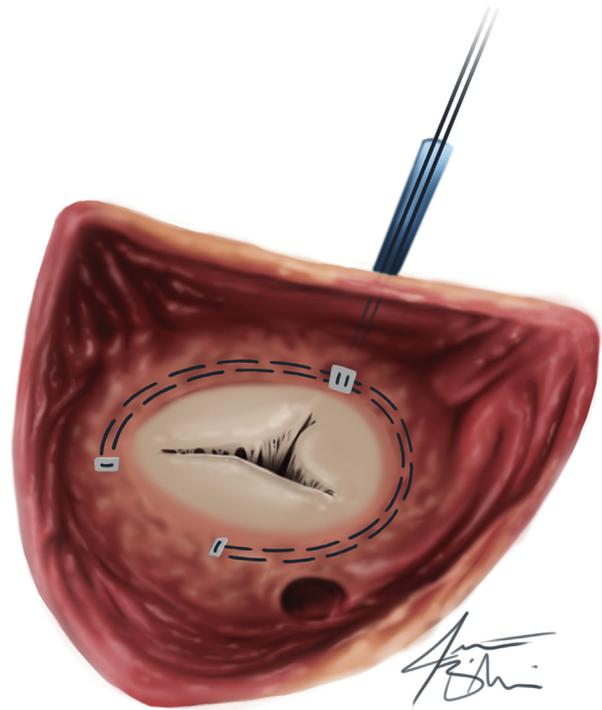


Fig 1. Schematic of the DeVega semicircular suture annuloplasty used.

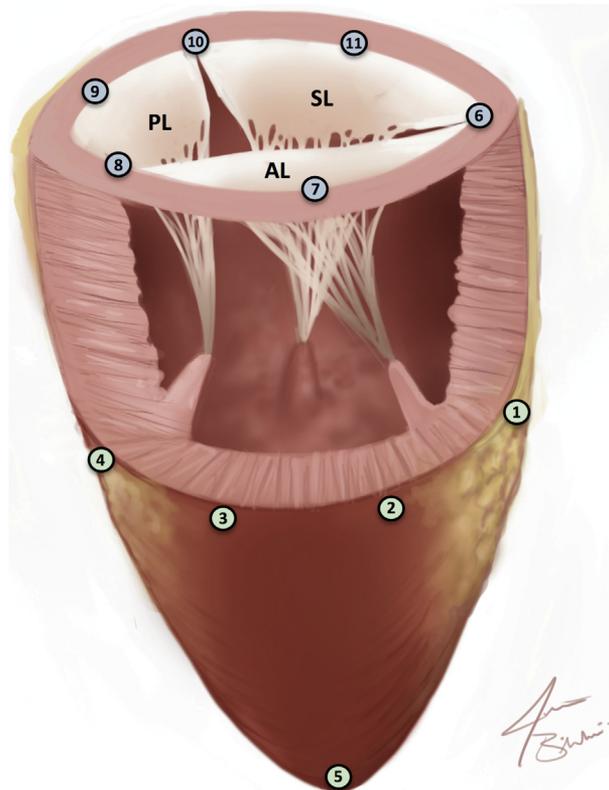


Fig 2. The location of the sonomicrometry crystals implanted around the tricuspid annulus and on the right ventricle. (AL = anterior leaflet; PL = posterior leaflet; SL = septal leaflet.)

weaning from cardiopulmonary bypass, animals were allowed to stabilize for 30 minutes to achieve steady state hemodynamics. Every animal received 300 mg of amiodarone IV and was maintained on lidocaine IV drip ($0.03 \text{ mg} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) to prevent ventricular ectopy. All animals were studied under open-chest experimental conditions.

Simultaneous hemodynamic, echocardiographic, and sonomicrometry data were acquired at baseline, after induction of acute right heart failure (RHF), and after 2 successive cinchings of the DeVega suture (DV-1 and DV-2) during RHF. The suture was cinched initially 8 to 10 mm by pulling against an epicardial pledget and clamped with a hemostat. During the second cinch, a second hemostat was used to mark the same 8 to 10 mm distance from the first while the suture was externalized further. At the conclusion of the experiments, the animals were euthanized by administering sodium pentothal (100 mg/IV) and potassium chloride bolus (80 mEq IV).

Induction of Acute Right Heart Failure

Acute RHF was induced as previously described [15] by a combination of 500 mL of crystalloid solution bolus infusion over 2 minutes followed by occlusion of posterior descending artery for 5 minutes and subsequent pulmonary artery constriction to increase maximal right ventricle pressure to at least 150% of its preocclusion value. Visually estimated degree of the pneumatic occlusion used did not obstruct more than 40% to 50% of the main pulmonary artery. Animals were permitted to achieve stable hemodynamics for 15 minutes under these combined interventions prior to DV annuloplasty suture cinchings. The model of acute RHF used provided consistent, stable conditions tolerated well up to 30 minutes.

The FTR was graded using epicardial echocardiography and included evaluation of color flow and continuous wave Doppler. TR was categorized as none or trace (0), mild (+1), moderate (+2), moderate to severe (+3), and severe (+4). The mean inflow gradient across the valve was assessed using continuous wave Doppler.

Data Acquisition and Analysis

Sonomicrometry data were acquired using a Sonometrics System DS3 (Sonometrics Corp) as previously described [16]. Data from 10 consecutive cardiac cycles acquired at 128 Hz during normal sinus rhythm were averaged for each animal. Sonomicrometry recordings were analyzed in CardioSOFT software ver. 3.4.73 (Sonometrics Corp). All values were calculated at their maximal and minimal throughout the cardiac cycle and at end-systole (ES) and end-diastole (ED). End-diastole was defined as the time of R wave in electrocardiography and ES was determined as the time of maximum negative dp/dt of left ventricular pressure.

Tricuspid Annular Geometry and Dynamics

Annular area and perimeter were calculated based on crystal positions [16]. Septolateral (S-L) annular dimension was calculated as the distance between crystals 7 and

11; A-P annular dimension as the distance between crystals 6 and 9 (Fig 1). Intercommissural annular dimensions were calculated as the distances between crystals 6 and 8, 8 and 10, and 6 and 10. Annular 3D geometry was expressed as annular height calculated as the maximal displacement of annular crystals from the annular plane.

Annular area contraction was characterized as the percentage difference between maximal and minimal annular area during the cardiac cycle. Regional annular contraction was defined as the percentage difference between maximal and minimal regional perimeter during the cardiac cycle. RV volume was calculated using the convex hull method based on crystal coordinates.

Strain Analysis

The strain analysis for both steps of suture annuloplasty was performed as previously described [17]. Strain, as a relative measure of displacement (and thus annular deformation), is calculated relative to a "reference configuration". Here, we chose RHF at ED and ES as the reference configurations for both DV-1 and DV-2 at ED and ES, respectively. Specifically, Green-Lagrange strain was calculated along the entire annulus for each animal and later displayed on a spline representation of the population-averaged annulus for each intervention. Global and regional average tricuspid annular strains were calculated for the entire annulus and the anterior, posterior, and septal annulus by averaging them along the respective regions. Negative or "compressive" strains imply that tissue is compressed, whereas positive or "tensile" strains imply that tissue is stretched.

Statistical Analysis

Data are presented as mean \pm standard deviation unless otherwise stated. The measured variables were compared between interventions with repeated-measures ANOVA, or Friedman repeated-measures ANOVA on ranks when the normality test failed, with DV-1 and DV-2 compared to RHF with Dunnett's post hoc test. SigmaPlot 12.5 software (SYSTAT, San Jose, CA) was used for all statistical analysis.

Results

Hemodynamics

The mean cardiopulmonary bypass time was 85 ± 12 minutes. Hemodynamic variables at baseline, with RHF, and at 2 successive steps of annuloplasty suture cinching (DV-1 and DV-2) are presented in Table 1. During induction of HF, RV dysfunction with increased central venous pressure (CVP) was observed and accompanied by stable heart rate and LV function. Suture annuloplasty cinching resulted in gradual decrease of RV volumes but did not perturb either LV or RV function. Interestingly, we did not observe the decrease in CVP with diminishing TR. This was an acute experiment, however, and there was limited time to appreciate the influence of reduced tricuspid insufficiency on CVP.

Table 1. Hemodynamics ($n = 10$)

Variable	Baseline	RHF	DV-1	DV-2	p Value
HR, min^{-1}	81 ± 13	78 ± 14	74 ± 14	76 ± 18	0.14
LV EDP, mm Hg	15 ± 8	17 ± 9	16 ± 11	14 ± 10	0.6
LV ESP, mm Hg	84 ± 9	72 ± 9	69 ± 24	71 ± 15	0.051
Maximal LV pressure, mm Hg	87 ± 8	77 ± 10	75 ± 24	75 ± 15	0.1
RV EDP, mm Hg	12 ± 14^a	20 ± 13	17 ± 14	16 ± 14^a	<0.001
RV ESP, mm Hg	30 ± 14^a	45 ± 12	40 ± 13	39 ± 14^a	<0.001
Maximal RV pressure, mm Hg	29 ± 14^a	47 ± 12	42 ± 13	41 ± 15^a	<0.001
CVP, mm Hg	11 ± 5^a	14 ± 5	14 ± 4	15 ± 4	<0.001
RV EDV, mL	68 ± 14^a	74 ± 18	63 ± 9^a	57 ± 13^a	<0.001

^a $p < 0.05$ versus right heart failure by Dunnett's test.

The p values were determined from repeated-measures ANOVA/repeated-measures ANOVA on ranks.

CVP = central venous pressure; DV-1 = first DeVega suture cinching; DV-2 = second DeVega suture cinching; EDP = end diastolic pressure; EDV = end diastolic volume; ESP = end systolic pressure; HR = heart rate; LV = left ventricle; RHF = right heart failure; RV = right ventricle.

Tricuspid Regurgitation

Before the experimental protocol, mean TR grade was 0.5 ± 0.5 . The combined interventions aimed at induction of acute RHF resulted in at least moderate TR in every animal, with a mean grade of 2.7 ± 0.8 ($p < 0.001$ versus baseline). With DV-1, FTR was reduced to 1.2 ± 0.4 ($p < 0.001$) and further suture tightening during DV-2 reduced mean FTR grade to 0.4 ± 0.5 ($p < 0.001$) (Figure 3). Mean pressure gradient across the tricuspid valve was 1.3 ± 0.7 mm Hg during DV-1 and 2.0 ± 1.0 mm Hg during DV-2.

Tricuspid Annular Geometry

The tricuspid annulus enlarged during RHF with both maximal area (Fig 3) and perimeter (Table 2) increasing. Stepwise suture annuloplasty tightening gradually decreased annular area, perimeter, and diameters. Annular height was 5.0 ± 1.9 mm at baseline and was unaffected by either induction of RHF (4.9 ± 2.0 mm), DV-1 (5.6 ± 1.4 mm), or DV-2 (5.5 ± 1.7 mm) suture cinchings

($p = 0.43$). Annular shape expressed as the orthogonal distance of each point along the annulus to the annular plane for baseline, RHF, and both annular reducing cinches is shown in Figure 4.

Tricuspid Annular Dynamics

Acute RHF did not change annular area contraction during the cardiac cycle, and, importantly, DV-1 and DV-2 maintained normal annular area dynamics even with significant area reduction well below the minimum area observed at baseline (Table 3). Similarly, dynamic change in regional annular length or commissural-commissural distances was not affected by DV-1 and DV-2 after induction of RHF.

Annular Strains

Figure 5 illustrates the absolute global and regional annular strains and Figure 6 presents the color maps of average annular strains with annular cinching during RHF. DV-1 and DV-2 caused progressive increase in compressive strain in all annular parts. Of note is the behavior of septal annulus not encompassed by the DV suture. This portion of the annulus showed little deformation in contrast to other regions with consistently compressive strain patterns.

Comment

Reductive tricuspid annuloplasty, whether with an annular suture or a prosthetic ring, represents the foundation of modern functional TR repair. In the current study, DeVega suture annuloplasty effectively eliminated FTR and preserved normal tricuspid annular dynamics and 3D geometry during acute right heart failure in sheep. DeVega suture annuloplasty abolished FTR by the reduction of S-L and anteroposterior diameters and all commissure-commissure distances. Even aggressive annular area reduction during DV-2 did not perturb normal annular dynamics or 3D geometry and was associated with low transvalvular pressure gradients. The

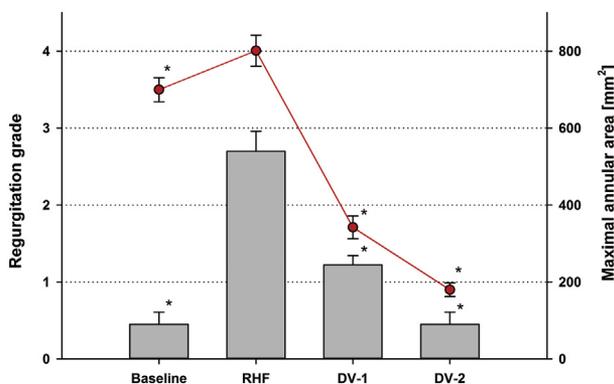


Fig 3. Tricuspid regurgitation grade (gray box plot + left y axis) and maximal tricuspid annular area (red line + right y axis). Error bars indicate 1 standard error of the mean. * $p < 0.05$ versus right heart failure by repeated-measures ANOVA with Dunnett's test. (DV-1 = first DeVega suture cinching; DV-2 = second DeVega suture cinching; RHF = right heart failure.)

Table 2. Tricuspid Annular Geometry ($n = 10$)

Variable	Baseline	RHF	DV-1	DV-2	<i>p</i> Value
Perimeter, mm					
max	102 ± 7 ^a	108 ± 8	74 ± 9 ^a	57 ± 9 ^a	<0.001
min	97 ± 8 ^a	103 ± 10	72 ± 9 ^a	54 ± 9 ^a	<0.001
A-P, mm					
max	31.6 ± 2.7 ^a	33.3 ± 2.2	20.9 ± 2.3 ^a	15.7 ± 2.5 ^a	<0.001
min	28.3 ± 3.4 ^a	30.0 ± 2.3	19.9 ± 2.4 ^a	14.9 ± 2.5 ^a	<0.001
S-L, mm					
max	27.4 ± 3.6	28.7 ± 2.7	19.8 ± 3.2 ^a	15.8 ± 2.5 ^a	<0.001
min	24.8 ± 3.8	26.5 ± 3.4	18.5 ± 3.2 ^a	14.7 ± 2.7 ^a	<0.001
C-C ₆₋₈ , mm					
max	24.1 ± 2.4 ^a	26.8 ± 1.8	14.4 ± 3.1 ^a	9.0 ± 2.8 ^a	<0.001
min	21.9 ± 1.9 ^a	24.5 ± 1.7	13.4 ± 3.1 ^a	8.3 ± 2.2 ^a	<0.001
C-C ₈₋₁₀ , mm					
max	27.5 ± 7.4 ^a	29.5 ± 7.6	19.2 ± 7.2 ^a	14.9 ± 6.8 ^a	<0.001
min	26.3 ± 7.2 ^a	28.3 ± 7.6	18.6 ± 7.3 ^a	14.4 ± 6.7 ^a	<0.001
C-C ₆₋₁₀ , mm					
max	35.2 ± 3.5 ^a	37.0 ± 3.1	27.6 ± 4.1 ^a	22.1 ± 5.2 ^a	<0.001
min	31.1 ± 3.6 ^a	34.6 ± 3.3	26.1 ± 4.6 ^a	21.1 ± 5.3 ^a	<0.001

^a $p < 0.05$ versus right heart failure by Dunnett's test.

The *p* values were determined from repeated-measures ANOVA. Number of crystals as shown in Figure 1.

A-P = anteroposterior; C-C = commissure-commissure; DV-1 = first DeVega suture cinching; DV-2 = second DeVega suture cinching; RHF = right heart failure; S-L = septolateral.

first step of our annuloplasty cinching (DV-1), with a 30% reduction of A-P dimension, reduced TR from moderate to severe to mild, and similar annular size reduction is usually achieved clinically [18]. Further

cinching with DV-2 resulted in roughly 50% size reduction—which can be considered as significant undersizing—yet such aggressive annular reduction when utilizing suture annuloplasty has been advocated

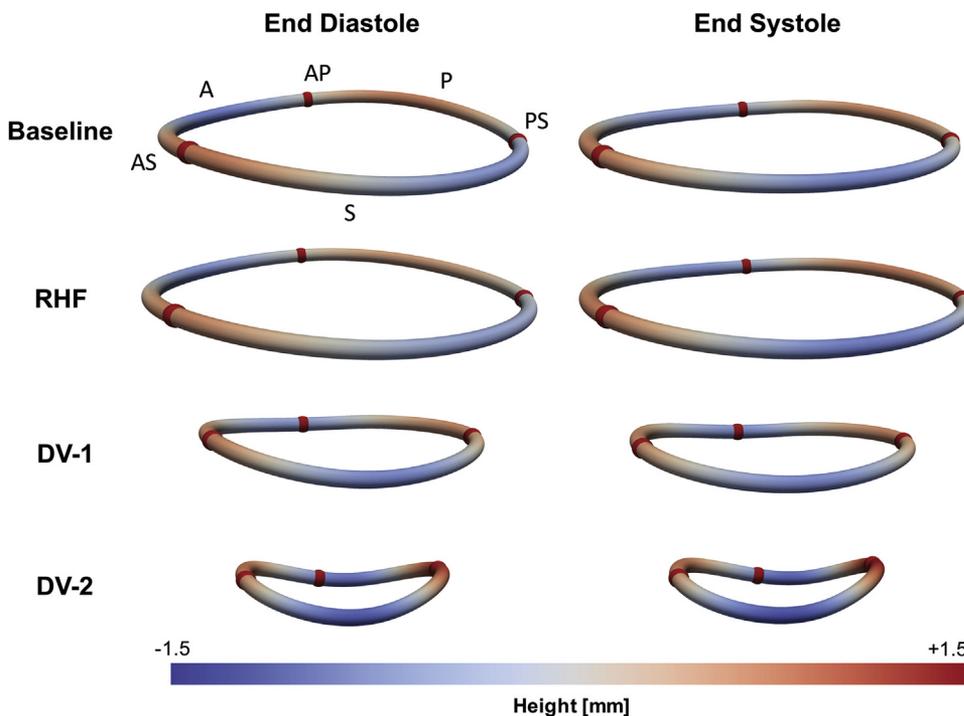


Fig 4. Tricuspid annular 3D geometry displayed as the out-of-plane displacement of each point along the annulus created using spline fit analysis. (A = anterior; AP = anteroposterior commissure; AS = anteroseptal commissure; DV-1 = first DeVega suture cinching; DV-2 = second DeVega suture cinching; P = posterior; PS = posteroseptal commissure; RHF = right heart failure; S = septal.)

Table 3. Tricuspid Annular Dynamics (n = 10)

Variable	Baseline	RHF	DV-1	DV-2	p Value
Annular contraction, %	12 ± 6	12 ± 7	9 ± 6	12 ± 6	0.6
Anterior annulus, %	11 ± 4	9 ± 4	8 ± 3	8 ± 3	0.21
Posterior annulus, %	7 ± 2	6 ± 3	6 ± 4	7 ± 4	0.18
Septal annulus, %	11 ± 5 ^a	8 ± 4	7 ± 3	10 ± 5	0.002
C-C ₆₋₈ shortening, %	9 ± 3	8 ± 4	7 ± 4	7 ± 3	0.25
C-C ₈₋₁₀ shortening, %	5 ± 1 ^a	4 ± 1	4 ± 3	4 ± 3	0.009
C-C ₆₋₁₀ shortening, %	10 ± 3 ^a	6 ± 3	5 ± 2	5 ± 2	<0.001

^a p < 0.05 versus right heart failure by Dunnett's test.

The p values were determined from repeated-measures ANOVA/repeated-measures ANOVA on ranks.

C-C = commissure-commissure; DV-1 = first DeVega suture cinching; DV-2 = second DeVega suture cinching; RHF = right heart failure.

by a recent study from the Mayo Clinic. Shinn and colleagues [8] reported the strategy of “overcorrection” with the DeVega annuloplasty for FTR repair. The investigators achieved good clinical results with routine reduction of the tricuspid annulus to a diameter of 2 cm, which is analogous to our annular diameter reduction of approximately 1.5 cm with DV-2 in smaller sheep hearts.

Changes in the 3D annular shape have been demonstrated to predict FTR [19], and annular deformation has been directly linked to geometrical changes observed in the RV [18]. Mechanistic insights from studies analyzing patients with chronic FTR suggest that restoration of bimodal annular shape and not annular reduction alone is critical to effective TR repair [19]. Similarly, reestablishing normal annular shape was found to decrease leaflet stress and annular strain after mitral repair [20]. Moreover, the preservation of annular height was found to optimize leaflet coaptation [21]. Currently available tricuspid annuloplasty prostheses do not restore the true physiological shape of the native annulus [16, 19]. DeVega suture annuloplasty in acute RHF, even with aggressive size reduction, preserved 3D annular geometry, which may in turn optimize leaflet stress distribution and potentially improve repair durability.

The variability in clinical results obtained with suture annuloplasty may be related to the lack of technique standardization as tricuspid suture annuloplasty is often freely modified by surgeons. The early “recommendation” suggested that suture annuloplasty should aim to reduce the annular orifice to admit 2 or 3 fingers [22], which underlines the lack of systematic approach towards restoration of annular size and geometry. Comparable to the results of Shinn and associates [8], the degree of initial annular cinching (DV-1) achieved 37% and 31% reduction in A-P and S-L diameters, respectively. With aggressive suture cinching (DV-2), we achieved a 52% and 45% reduction in the annular A-P and S-L diameters, respectively. The study of Min and coworkers [18], however, revealed that DeVega annuloplasty clinically reduces A-P and S-L diameters only by approximately 22% whereas flexible and rigid rings reduce these diameters by 32% and 38%, respectively. This degree of annular reduction

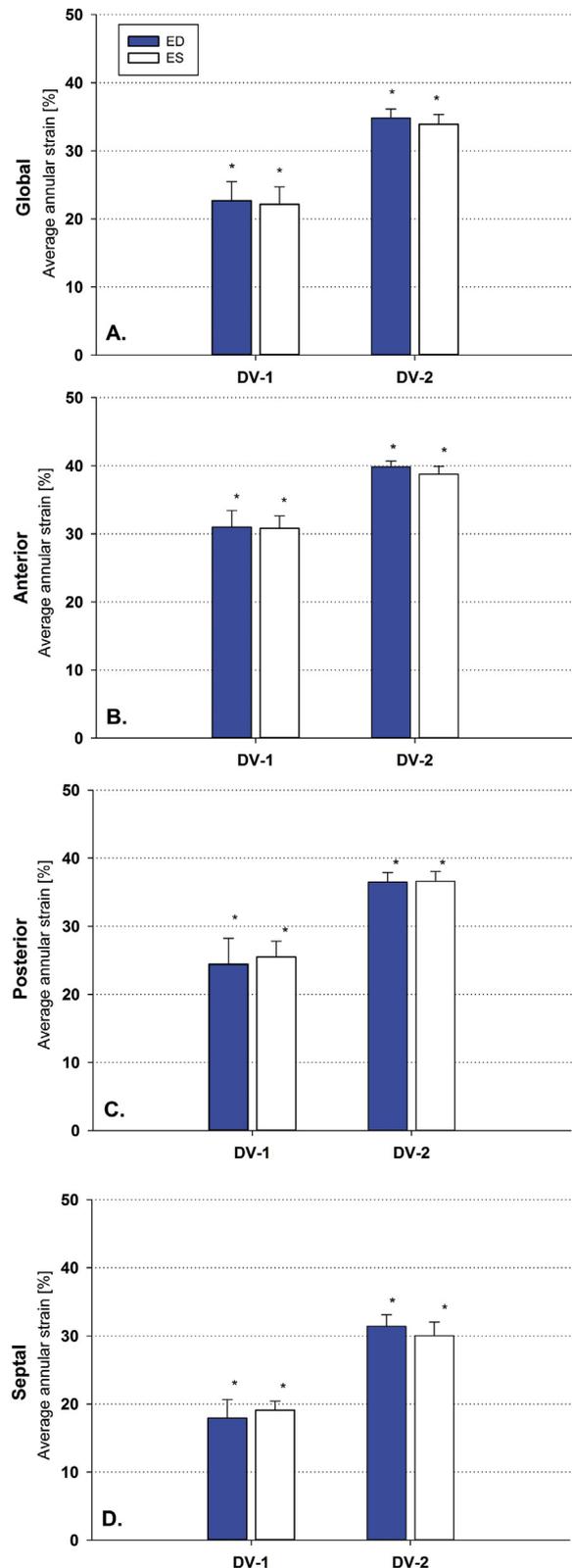


Fig 5. Absolute global (A) and regional (B–D) tricuspid annular strain. *p < 0.05 versus right heart failure (reference state) by repeated-measures ANOVA with Dunnett's test. (DV-1 = first DeVega suture cinching; DV-2 = second DeVega suture cinching; ED = end-diastole; ES = end-systole.)

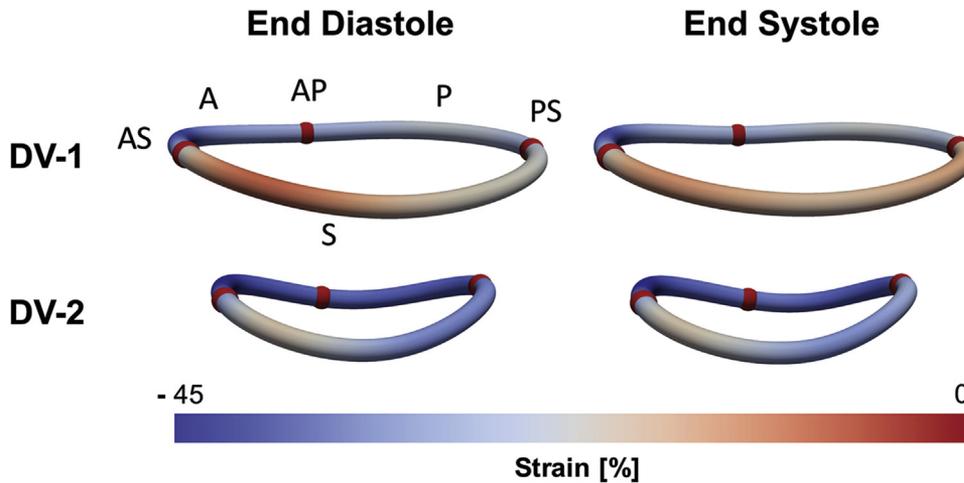


Fig 6. Maps of average tricuspid annular strain. (A = anterior; AP = anteroposterior commissure; AS = anteroseptal commissure; DV-1 = first DeVega suture cinching; DV-2 = second DeVega suture cinching; P = posterior; PS = posteroseptal commissure; RHF = right heart failure; S = septal.)

with suture annuloplasty may be inadequate as tight reduction promotes good long-term results [8], and, as shown in the current study, still maintains annular geometry and dynamics. Optimal annuloplasty dimension remains to be defined, however.

In the original technique described by DeVega [9], annuloplasty sutures were anchored at anteroseptal and posteroseptal commissures leaving the entire septal annulus unsupported. There is evidence, however, suggesting that the septal annulus dilates with FTR [19]. Moreover, *in vitro*, the septal annulus has been demonstrated to be under greatest tension that increases disproportionately with annular dilation [23]. The goal of any physiologic repair should be to minimize annular and leaflet stress, a goal best achieved through preservation of normal valve geometry [20]. The technique used in our study permitted partial support of the septal annulus, and this corroborates recommended clinical practice [24] and yields optimal surgical results as demonstrated by the Mayo Clinic group [8]. When the DeVega suture is anchored at septal commissures only, the force required to reduce the annulus in the septal-lateral direction increases. This, in turn, results in greater diameter decrease in the A-P rather than in the S-L direction with the same degree of suture cinching [25]. The technique used in our study achieved a more equal suture pull distribution around the annulus, and disproportion between A-P and S-L diameter reduction was not evident.

The A-P annular enlargement regardless of TR degree has been advocated as an indication for preventive annuloplasty [26, 27], yet other investigators support a more conservative approach [28]. Whether suture annuloplasty has any role as a preventive technique remains to be established but the results of studies on transplanted hearts are promising [29].

Strain analysis revealed an increase in the compressive strains in all annular parts subtended by the suture. Similar results of increased native annular strain with the reduction of valve circumference was observed in ovine studies of mitral annuloplasty rings [30]. Our findings of

larger compressive strains with greater constraint of the annular circumference, as seen in DV-2, are therefore not surprising in this context. Nevertheless, the interesting finding is the small strain change in the septal part of the annulus especially close to the anterior commissure. A similar strain pattern was reported in unprotected native mitral septal annular segments with mitral annuloplasty bands [30]. Conversely, the highest stress was found in the septal leaflet close to the annulus in the finite element model of the tricuspid valve [31]. This septal region of the tricuspid annulus was also previously shown to generate the greatest tension relative to other annular segments [23]. Moreover, we have previously demonstrated that the normal ovine tricuspid annulus has an asymmetric strain and curvature pattern during systole with the highest curvature in this particular region [17]. Strain analysis from our study might be helpful in gaining insight into the modes of valve repair failure after tricuspid annuloplasty. It can be conjectured that the bigger the deformation (higher absolute strain) within the annulus, the greater the chance of suture or ring dehiscence. The greatest strain observed in the anteroseptal commissure could in this context explain suture tearing in this region and the so-called bow-string effect responsible for the suboptimal outcomes of the original DeVega technique [32].

Study Limitations

The results of our study must be interpreted in the context of important limitations. This study utilized an ovine model of acute RHF that is substantially different from chronic clinical right heart failure. The utilized model was not designed to reflect clinical right heart dysfunction with associated tricuspid insufficiency, but to provide an experimental platform for physiologic evaluation of suture-based annular reduction in the setting of clinical determinants of right heart failure. Surgical intervention during acute RHF in healthy ovine hearts under open-chest experimental conditions limits the direct clinical extrapolation of the results. A chronic RHF

animal study is necessary to assess the long-term impact of the DeVega suture. Our study also did not examine the influence of reductive suture annuloplasty on the tricuspid leaflets and the subvalvular apparatus, key valvular components that contribute to the clinical pathophysiology of functional TR. Clinically, DeVega annuloplasty is currently seldom used, although there are still certain clinical situations, such as in transplanted hearts, when it may be a preferred technique.

Conclusion

During acute right heart failure in healthy adult sheep, modified DeVega suture annuloplasty effectively abolished functional tricuspid insufficiency while preserving normal annular geometry and dynamics, even with aggressive annular reduction. Chronic studies are needed to elucidate the potential advantage of preserved annular dynamic and geometry seen with suture annuloplasty in yielding improved clinical outcomes for patients with right heart dysfunction and functional tricuspid insufficiency.

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