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ORIGINAL ARTICLE

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The effect of pulmonary hypertension on ovine tricuspid annular dynamics[†]

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Abstract

OBJECTIVES: Pulmonary hypertension (PHT) is associated with tricuspid annular dilatation, but the effect of acute increase of pulmonary pressure on three-dimensional (3D) tricuspid annular dynamics and shape is unknown. Better understanding of tricuspid annular dynamics may lead to improved and more durable surgical reparative techniques.

METHODS: In nine open-chest anaesthetized sheep nine sonomicrometry crystals were implanted on the right ventricle while on cardiopulmonary bypass. Additional nine crystals were implanted around the tricuspid annulus (TA) with one crystal at each commissure defining three separate annular regions: anterior, posterior and septal. Two additional equidistant crystals were implanted between each commissure, creating three segments for every region. Pressure transducers were placed in the left ventricular (LV), right ventricular (RV) and right atrium. PHT was induced by acute pulmonary artery constriction with a pneumatic occluder. Sonomicrometry and echocardiographic data were collected before and after induction of PHT. TA area, regional and total perimeter, and 3D annular geometry were calculated from 3D crystal coordinates. Regional annular contraction was defined as the percentage difference between maximal and minimal region length during the cardiac cycle.

RESULTS: PHT increased RV pressure from 31 ± 9 mmHg to 46 ± 13 mmHg ($P = 0.001$) and decreased left ventricular (LV) pressure from 111 ± 24 mmHg to 78 ± 36 mmHg ($P = 0.018$). There was no significant tricuspid regurgitation observed with PHT. During PHT, the TA area increased by 12 ± 13% from 641 ± 139 mm^2 to 721 ± 177 mm^2 ($P = 0.037$). The total perimeter increased from 103 ± 11 mm to 109 ± 13 mm ($P = 0.02$). All annular regions dilated significantly with PHT with 8 ± 10, 5 ± 5 and 5 ± 5% increase in anterior, posterior and septal annular length, respectively ($P < 0.05$). PHT reduced regional annular contraction in the anterior region only (17 ± 7 vs 14 ± 8%; $P = 0.02$). The TA had a complex 3D saddle geometry and the shape of the annulus was altered during PHT only in the antero-posterior region.

CONCLUSIONS: The changes in tricuspid annular conformation, contractility and its 3D geometry observed during acute ovine PHT may help in the design of new pathology-specific tricuspid annular rings.

Keywords: Tricuspid valve • Pulmonary hypertension • Tricuspid annulus

INTRODUCTION

Tricuspid annulus (TA) is a central component of the tricuspid valve apparatus facilitating timely and efficient valve closure and maintaining valve competency. It is also the predominant target for surgical repair of tricuspid regurgitation (TR), which is most frequently functional in nature and arising from left heart pathology [1]. The TA has been shown to have a 'sphincteric' role in achieving valve closure [2], with more recent studies revealing complex saddle-shaped geometry [3] and the ability to reduce circumference and area by ∼20 and 30%, respectively [3, 4], during the cardiac cycle. Normal tricuspid annular shape and motion have been described using both sonomicrometry technology in large animals [5–7] and 2D [4, 8] and three-dimensional (3D)-echocardiography [8, 9] in humans. However, there is still paucity of data regarding its dynamics in many pathological conditions.

Almost 80% of TR observed clinically is functional in nature and a consequence of tricuspid annular dilation due to right ventricular (RV) dysfunction [10]. Pulmonary hypertension (PHT) of any aetiology is a known risk factor predisposing patients to functional TR [11]. It has been demonstrated that in functional TR, the TA becomes more planar and circular as it dilates [9]. Clinical

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experience suggests that the TA dilates only in the anterior and posterior region while the septal annulus remains fixed at the interventricular septum [12]. However, the relatively high incidence of recurrent TR after successful annuloplasty brings this into question and warrants further study of the geometry and motion of the TA [13, 14].

The aim of the present study was to evaluate in an ovine model the influence of acute pressure overload of the right ventricle on the dynamics and geometry of the TA. Better understanding of tricuspid annular dynamics may lead to improved and more durable surgical reparative techniques.

MATERIALS 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.

Surgical preparation

Nine healthy adult male sheep (65 ± 5 kg) were premedicated with ketamine (25 mg/kg im), anaesthetized with propofol (2–5 mg/kg IV), intubated and mechanically ventilated. General anaesthesia was maintained with inhalational isoflurane (1–2.5%). Fentanyl CRI (5–20 μg/kg/min) was infused as additional maintenance anaesthesia. Atropine (0.1 mg/kg IV) was given as needed for bradycardia defined as a heart rate <60. A micromanometer catheter (Millar Instruments, Inc., Houston TX, USA) was introduced through the left carotid artery for arterial blood pressure measurements and arterial blood gas analysis. The operative procedure was performed through a median sternotomy and the heart was exposed in the pericardial cradle.

Animals were fully heparinized and the aorta and both the superior and the inferior vena cava were cannulated in preparation for cardiopulmonary bypass. Caval snares were placed around the superior and the inferior vena cava. After activated clotting time exceeded 480, normothermic cardiopulmonary bypass was initiated. Subsequently, the aorta was cross-clamped and cold (4°C) crystalloid high-potassium cardioplegia (2670 ± 530 ml) was delivered in the aortic root to achieve diastolic cardiac arrest and myocardial protection during the procedure. After snaring both venae cavae, the right atrium was opened. Nine 2 mm sonomicrometry crystals (Sonometrics Corporation, London, ON, Canada) were implanted with 5-0 polypropylene suture around the TA. One crystal was implanted at each commissure defining three separate annular regions: anterior (A), posterior (P) and septal (S). Two additional equidistant crystals were implanted between each commissure creating three segments for every region. Crystal electrodes were exteriorized through the right atriotomy. Additional eight crystals were implanted in the right ventricular myocardium along four equally spaced longitudinal meridians with a ninth crystal at the right ventricular apex. Schematic representation of the complete crystal array is shown in Fig. 1. Micromanometer pressure transducers (PA4.5-X6; Konigsberg Instruments, Inc., Pasadena, CA, USA) were placed in the LV and RV through the apex. A pulmonary artery pneumatic occluder (Kent Scientific Co., Torrington, CT, USA) was placed around the main pulmonary artery and the tubing was externalized through the surgical incision.

After completion of crystal placements, the atriotomy was closed, cross-clamp removed, heart resuscitated and the animal was weaned from cardiopulmonary bypass. When satisfactory haemodynamics were achieved, heparin was reversed with protamine, and the animals were allowed to stabilize for 30 min to achieve steady-state haemodynamics prior to data acquisition.

Experimental protocol

Animals were studied as open chest, open pericardium acute experimental model. The animals were paralysed with cistatracurium (0.3 mg/kg) IV to prevent motion during data gathering and with additional fentanyl administered to assure animal comfort and analgesia. After haemodynamic stabilization, baseline sonomicrometry

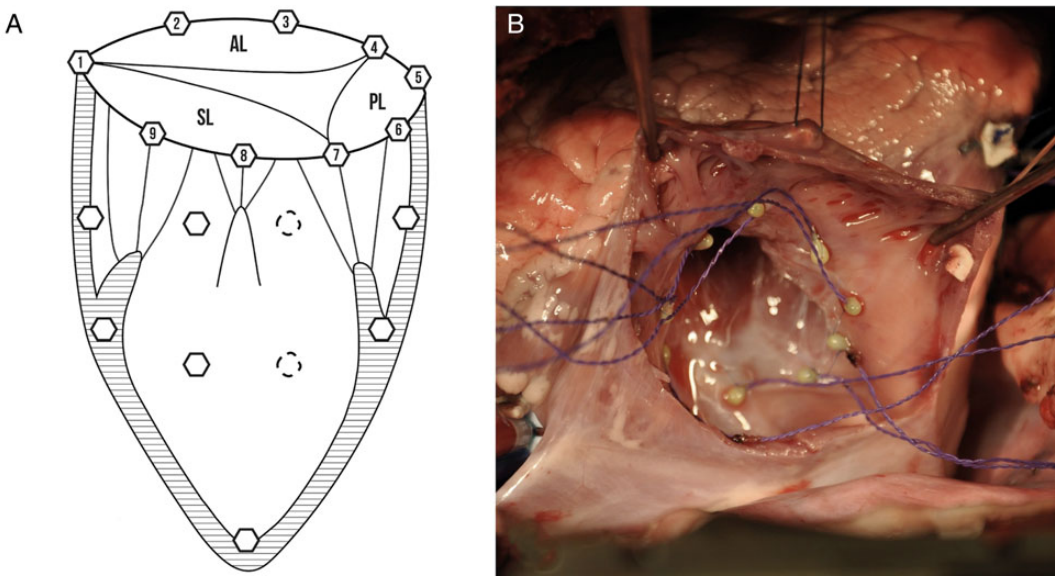

Figure 1: The locations of the sonomicrometry crystals around the tricuspid valve annulus and the right ventricle (**A**); intraoperative photography of annular crystals (**B**). AL: anterior leaflet; PL: posterior leaflet; SL: septal leaflet.

crystal data were acquired for at least ten consecutive cardiac cycles. Baseline (Control) epicardial echocardiography was obtained to assess function of the right ventricle and competence of the tricuspid valve.

Five minutes of stabilization was permitted between baseline and intervention data capture to assure steady-state haemodynamics as defined by unchanging blood pressure and heart rate. Subsequently, a pulmonary artery pneumatic occluder was tightened by inflating 10 ml of air through the externalized tubing to achieve acute PHT and increase in right ventricular pressure by at least 40%. When the RV pressure had increased adequately, crystal and echocardiographic data were acquired again (PHT).

At the conclusion of the experiment the animals were euthanized by administering sodium pentothal (1 g IV), followed (after 2 min) by an IV bolus of 80 mEq potassium chloride. The heart was excised and the proper placement of annular and ventricular crystals was confirmed.

Data acquisition and analysis

All sonomicromety data were acquired using a 32-channel Sonometrics Digital Ultrasonic Measurment System DS3 (Sonometrics Corporation, London, ON, Canada). This technique relies on measurements of distances within tissue by means of piezoelectric crystals and has been validated in prior large animal experiments [15]. Data in our study were acquired at 200 Hz with simultaneous left ventricular pressure, right ventricular pressure and electrocardiography (ECG) recordings.

All data recordings were analysed in CardioSOFT Software ver 3.4.60 (Sonomicrometrics Corporation). This post-processing software permits analysis of individual 3D crystal positions and subsequent distance measurements between crystals. To calculate the area and perimeter and assess the 3D geometry of TA, the centroid was calculated for the annular crystal vertices, and then a translation was applied to the *x*,*y*,*z* coordinates of the vertices so that the centroid became the origin. A subsequent rotation of these *x*,*y*,*z* coordinates was applied so that the normal vector of the plane was aligned parallel to the *z*-axis. The area was calculated by adding the nine triangular areas of each sequenced pair of vertices and the centroid utilizing only the *x*,*y* coordinates. Similarly, the perimeter prescribed by the vertices on the plane was calculated by adding the appropriate sequence of *x*,*y* distances between the selected vertices.

The distances between annular crystals and the best fit annular plane were used to determine the 3D shape of the TA. Annular height was calculated as the difference between maximal and minimal deviation from the annular plane.

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 the beginning of positive deflection in ECG voltage (*R* wave); end-systole (ES) was determined as the time of left ventricular end-systolic pressure.

Regional tricuspid annular contraction was calculated as the differences between the maximal and minimal perimeter throughout the cardiac cycle $[(P_{max} - P_{min})/P_{max} \times 100\%]$ for the particular region (anterior, posterior and septal) and its three segments. Similar measurements were done for the tricuspid annular area.

Right ventricular volume was calculated using a convex hull method based on the 3D coordinates of the nine ventricular and nine annular crystals.

Statistical analysis

Data are presented as mean ± standard deviation. The measured variables were compared between control and PHT conditions using Student's two-tailed *t*-test for dependent observations with a *P*-value of <0.05 considered significant.

RESULTS

Haemodynamics

Haemodynamic variables before (control) and after induction of PHT are presented in Table 1. There was no change in heart rate. Pulmonary artery constriction expectedly resulted in increased RV maximal pressure and decreased LV pressures. RV volume and central venous pressure also increased significantly, confirming the acute haemodynamic effect of pulmonary artery constriction. There was no significant TR observed with induction of acute RV pressure overload.

Tricuspid annular size and dynamics

PHT increased the end-diastolic tricuspid annular area significantly by 12 ± 13% from 641 ± 139 mm^2 to 721 ± 17 7 mm^2 ($P = 0.037$). End-systolic and maximal and minimal annular area throughout the cardiac cycle also increased significantly (Table 2). The total annular perimeter increased its end-diastolic value by 6 ± 6% from 103 ± 11 mm to 109 ± 13 mm. ($P = 0.02$). All annular regions enlarged significantly with PHT with an 8 ± 10, 5 ± 5 and 5 ± 5% increase in anterior, posterior and septal region length, respectively ($P < 0.05$). However, it was only the anterior region that decreased its contractility significantly with PHT (17 ± 7 vs 14 ± 8%; $P = 0.02$). The biggest contributor to anterior annular contraction was it mid-portion segment (between crystals 2–3). However, it was its pericommissural segment (near the antero-posterior commissure) that was most affected by PHT, decreasing its contractility from 15 ± 7 to 11 ± 7% ($P = 0.04$). There was also a trend for decreased contractility of the posterior region with PHT (14 ± 8 vs 11 ± 6%; $P = 0.058$) (Table 3). The most active part of the posterior region was its segment nearest to the postero-septal commissure. The lowest contractility was observed

Table 1: Haemodynamics

	Control	PHT	*P*-values
HR (min^{-1})	100 ± 17	104 ± 19	0.09
RVP maximal (mmHg)	31 ± 9	46 ± 13	0.001
RV EDP (mmHg)	12 ± 8	15 ± 8	0.078
RV EDV (ml)	56 ± 10	64 ± 14	0.002
RV ESV (ml)	46 ± 11	56 ± 14	0.001
LVP maximal (mmHg)	111 ± 24	78 ± 36	0.018
LV EDP (mmHg)	19 ± 14	8 ± 11	0.002
CVP (mmHg)	11 ± 3	14 ± 5	0.01

HR: heart rate; RVP: right ventricular pressure; RV EDP: right ventricular end-diastolic pressure; RV EDV: right ventricular end-diastolic volume; RV ESV: right ventricular end-systolic volume; LVP: left ventricular pressure; LV EDP: left ventricular end-diastolic pressure; SAP: systolic arterial pressure; DAP: diastolic arterial pressure; CVP: central venous pressure; PHT: pulmonary hypertension.

in the septal part of the annulus where its mid-portion segment contracted the least (Table 3).

Tricuspid annular shape

Least squares displacement of crystals from the annular plane revealed a 3D saddle-shaped geometry of the TA. Maximal elevation points ('saddle horns') were observed at the antero-septal and antero-posterior commissures (Fig. 2). The lowest points were located in the postero-septal commissure and in the mid portion of the anterior region. There was very little geometrical deformation of the annulus with PHT. Only the mid portions of both the anterior (around crystal 3) and the posterior (near crystal 6) region were significantly affected in response to pressure overload. No change in annular height at both ED and ES were observed between groups. (ED: 5.8 ± 4.2 vs 6.0 ± 4.6; $P = 0.7$; ES: 6.1 ± 4.7 vs 6.4 ± 5.1 $P = 0.26$; control versus PHT, respectively).

DISCUSSION

The current study represents the first attempt to evaluate tricuspid annular dynamics in the setting of acute PHT and right ventricular volume overload. Our control data confirmed previous findings of other investigators describing the 3D structure and geometric changes during the cardiac cycle of the TA [5, 6]. The annulus reconstructed from our study is a saddle-shaped structure with two 'horns'- the anterior one located at the antero-septal commissure and the posterior one located near the antero-posterior commissure. These geometric findings are in concordance with the sonomicrometry studies of Fawzy *et al.* [5] and Hiro *et al.* [6]. Similarly, this bimodal shape was also observed on 3D echocardiography in humans [16].

High pulmonary artery pressure is often associated with the development of functional TR [11]; however, development of TR seems to be multifactorial and is affected by the integrity of the tricuspid valve apparatus, RV dimensions and RV function, as in on study ~ 50% of patients with PAP > 70 mmHg still did not develop more than mild TR [17]. In our acute ovine model, we did not observe any significant increase in TR despite RV distension. The lack of TR may be the result of compensatory mechanisms of annular contractility and lack of chronic pressure overload and ventricular and annular remodelling. The decrease in annular contractility with PHT observed in our study was in the range of 3–4%, and perhaps insufficient to make the valve incompetent. Moreover, it may be that 15 mmHg (25%) increase in RV end-diastolic pressure in our model was inadequate to induce TR compared with the 66% RV volume increase observed by Liakopoulos *et al.* [17] that was associated with moderate/severe TR in 60% of the studied animals. We observed 12% annular area dilatation with PHT but in isolated porcine valves an increase in area of at least 40% was necessary to induce significant tricuspid insufficiency [18].

It is important to recognize that in our study the septal annulus significantly contributed to TA enlargement with PHT. Anterior and posterior annular region dilatation is thought to induce loss of leaflet coaptation and functional tricuspid insufficiency while the septal annulus remains fixed [12]. Subvalvular tethering may also contribute significantly by restricting leaflet apposition [3]. However, the 'forgotten' septal region of the TA in our model also contributed significantly to overall annular dilation. Its magnitude of enlargement during PHT was similar to that observed in the posterior region, and the septal annulus maintained an active role in annular dynamics. These findings are in contrast to the study of

Table 2: Tricuspid annular size throughout the cardiac cycle

	Area (mm^2)		Perimeter (mm)	
	Control	PHT	Control	PHT
ED	641 ± 139	721 ± 177*	103 ± 11	109 ± 13*
ES	623 ± 148	672 ± 190*	101 ± 12	105 ± 15*
Maximal	706 ± 139	766 ± 180*	108 ± 11	112 ± 13*
Minimal	579 ± 137	651 ± 194*	98 ± 11	103 ± 15*
%ΔMax–Min	18 ± 7	16 ± 8	10 ± 4	9 ± 5

PHT: pulmonary hypertension; ED: end-diastole; ES: end-systole.
*$P < 0.05$ by a paired *t*-test between control and PHT.

Table 3: Regional tricuspid annular dynamics during pulmonary hypertension

Length	Maximal (mm)		Minimal (mm)		Contraction (%)	
	Control	PHT	Control	PHT	Control	PHT
Anterior	32.5 ± 7.0	33.9 ± 7.6*	26.9 ± 6.4	29.3 ± 7.6*	17 ± 7	14 ± 8*
1–2	9.8 ± 2.6	10.9 ± 2.3*	8.1 ± 2.1	9.4 ± 1.7*	17 ± 11	13 ± 10
2–3	10.7 ± 3.5	10.8 ± 3.5	8.6 ± 3.4	8.9 ± 3.8	21 ± 10	20 ± 13
3–4	12 ± 4.4	12.3 ± 4.8	10.2 ± 3.6	10.9 ± 4.5	15 ± 7	11 ± 7*
Posterior	27.4 ± 5.4	28.0 ± 5.9*	23.7 ± 5.7	25.1 ± 6.3*	14 ± 8	11 ± 6
4–5	10.1 ± 2.4	10.4 ± 2.4*	8.9 ± 2.7	9.2 ± 2.8*	14 ± 9	12 ± 9
5–6	8.9 ± 2.4	9.0 ± 2.4	8.0 ± 2.5	8.4 ± 2.6	11 ± 7	9 ± 9
6–7	8.3 ± 2.2	8.6 ± 2.5	6.8 ± 2.1	7.5 ± 2.5	18 ± 14	13 ± 10
Septal	36.3 ± 8.3	37.4 ± 8.9*	32.9 ± 8.6	34.5 ± 9.6	10 ± 4	8 ± 4
7–8	10.1 ± 3.5	10.5 ± 3.6*	8.8 ± 3.5	9.3 ± 3.6	15 ± 7	13 ± 8
8–9	14 ± 3.4	14.2 ± 3.5	13.3 ± 3.3	13.6 ± 3.5	6 ± 4	4 ± 2
9–1	12.1 ± 3.1	12.7 ± 3.6	10.9 ± 3.0	11.6 ± 3.7*	11 ± 5	9 ± 6

The numbers (1–9) represent the consecutive annular crystals as shown in Fig. 1.
PHT: pulmonary hypertension.
$n = 9$; *$P < 0.05$ by a paired *t*-test between control and PHT.

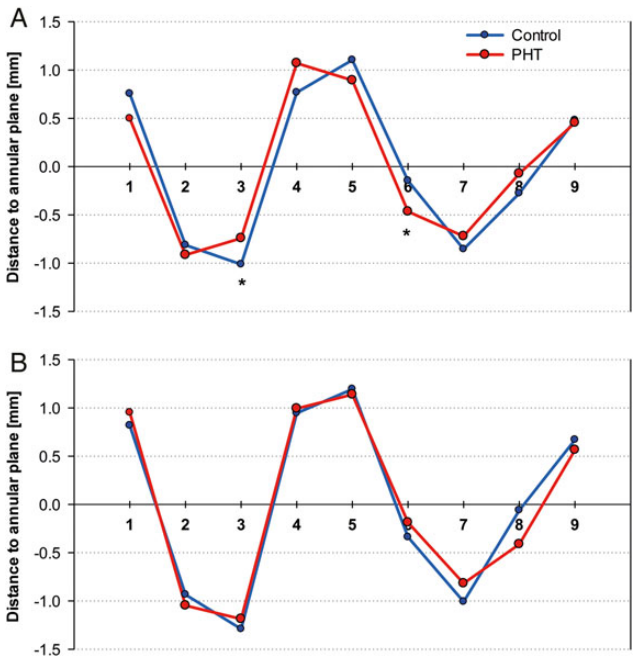

Figure 2: Geometry of tricuspid annulus based on the orthogonal distances of individual annular crystals (1–9) to the least squares plane of the tricuspid annulus. End-diastole (**A**), end-systole (**B**). *$P < 0.05$ by a paired *t*-test between control and PHT.

Dreyfus *et al.* [18] who suggested that annular enlargement involves only the anterior and posterior annulus, a concept partially supported by *in vitro* modelling. However, other ovine studies of tricuspid dynamics confirm our baseline findings of septal contractility and dynamic motion [6]. Furthermore, the septal portion of the mitral annulus has traditionally been considered a static portion of the mitral apparatus that did not remodel in states of pathology. Recent data have questioned this surgical dogma, revealing that fibrous portion of the mitral annulus dilates in patients with cardiomyopathy [19] and in animal models of biventricular failure [20]. As such, it is not unreasonable to expect that the septal region of the TA would dilate under either pressure or volume overload of the RV. These findings have significant clinical implications, as the most commonly utilized surgical repair of functional TR is ring annuloplasty. The currently available prostheses starting from Carpentier-Edwards Classic through St.Jude Medical Tailor Band till the newest Edwards MC3, Medtronic Contour 3D or Tri-Ad Adams tricuspid rings do not fully support the septal annulus due to concerns over conduction pathway injury; however, annular dilatation in this region could contribute to recurrent TR. We thus preliminarily propose a closed 3D annuloplasty ring as one of the possible solutions to avoid recurrent TR. In similar fashion, complete ring annuloplasty has replaced partial band annuloplasty for the treatment of functional mitral regurgitation.

Non-planarity and saddle-shaped annular geometry were first described in animal studies of the mitral valve [21]. This 3D conformation of the annulus was shown to decrease leaflet stress and aid valvular competency [22]. Owing to similar embryonic development and functional importance, it is not surprising that the TA has a similar bimodal shape that we observed under control conditions. We did not observe any important alterations in this tricuspid annular geometry with acute PHT although one may expect flattening and other shape deformations with PHT, but these alterations were seen only with the development of TR [16]. Since significant TR was not seen in our study with PHT, these results are difficult to extrapolate. On the other hand, maintained annular geometry as represented by TA height was also reported by Ring *et al.* [9] in 3D echocardiographic study of TA dynamics in dilated hearts. Interestingly, in their study, the analysed subjects had no more than mild TR despite an enlarged RV, similar to our experimental conditions.

Limitations of the study

The results of our study must be interpreted in the context of important limitations. This was an acute open-chest experimental model with inherent effects of anaesthesia and an open thorax on RV dynamics. However, acute ovine cardiac valvular studies have yielded important data that have subsequently been confirmed in chronic closed-chest models [23, 24]. The acute occlusion and increase in pulmonary pressure may not resemble the most common clinical scenario of chronic RV pressure overload resulting in PHT leading in many cases to functional TR. In addition, this study did not investigate other factors (such as papillary muscles and RV function) that may play a role in the mechanics of TA. This study included only healthy animals, which do not represent the chronic human condition and as such extrapolation of results into everyday clinical practice needs appropriate caution.

CONCLUSION

In our acute ovine study, we found that the TA is a saddle-shaped non-planar structure with two horns in the antero-septal and posterior regions. All three regions of the annulus maintained dynamic motion throughout the cardiac cycle at baseline. During acute PHT and LV pressure overload, the TA dilated along all annular regions while 3D annular geometry was essentially preserved. The length change of the anterior annulus, which was most contractile at baseline, was significantly depressed with PHT. These data provide new insight into tricuspid annular dynamics during states of haemodynamic pathophysiology and may aid in design of better annular prostheses with more predictable and durable clinical results.

Funding

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Conflict of interest: none declared.

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APPENDIX. CONFERENCE DISCUSSION

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Dr T. Wahlers *(Cologne, Germany):* This study in my eyes has clinical relevance because the knowledge about the pathophysiology of tricuspid regurgitation is still limited. Despite the fact that isolated tricuspid reconstruction is a rare operation, various rings and techniques are available addressing this problem, but all of us have experienced recurrent regurgitation after initial success. To relate your nice model to the clinical scenario, I've got three short questions.

In clinical pulmonary hypertension you very often observed severe tricuspid regurgitation. Don't you think we perhaps would have to modify your model in a way that we have to interfere with the contractility in a way that we have to initiate cardiomyopathy to more accurately mimic the clinical scenario?

Dr Malinowski: You're absolutely right, but we have to bear in mind the limitation of this study. It's an acute animal model in which we were not able to induce tricuspid regurgitation; and in fact, we did not study the remodelling of the right ventricle.

The fact that in the clinical condition we usually have a problem with the right ventricle and the solution with the annuloplasty is a solution to the ventricular problem similar to the mitral regurgitation. So in fact, it may be the case that more changes in the right ventricle will result in more changes in the tricuspid annulus. It's difficult to study this in an acute condition because we were not even able to produce tricuspid regurgitation, although we found that the right ventricle dilated. It is a proper model, but still it is an acute condition and truly does not resemble the chronic clinical setting.

Dr Wahlers: The second point might be perhaps your recommendation: Do you think that current ring concepts have to be changed from perhaps open to closed rings? Can you comment on this, on the clinical scenario, whether this is necessary based on the importance of the septal region you highlighted.

Dr Malinowski: You're absolutely right, I think one of the most important conclusions from our research is that all annular regions are dynamic and that the previous concept of the stable, fixed septal leaflet probably is not true any longer because all parts contract. So I think that we should redefine our thinking and maybe go into the closed rings.

Of course, we are all afraid as surgeons of the conductance system there; but maybe it will let us do better and avoid the situation of more and more patients coming back with a recurrent tricuspid regurgitation. That's a really, really bad condition and we should do everything to avoid this. And maybe that's the first step we took to redefine our thinking about the annulus.

Dr Wahlers: The final point is we learned from mitral valves that if you put a patient under anaesthesia, mitral regurgitation decreases from 3 to 1. Have you varied, for example, your narcotics in order to investigate that?

The second point is I've seen that your CVP only increased, I think, from 11 to 14. So perhaps your model could be stressed even more if you preload the patients more or vary the anaesthetics.

Dr Malinowski: But still that is coming back to your first question, there is an obvious limitation of that kind of acute model in which we cannot exclude the influence of the anaesthesia and in general the open chest conditions. Although the CVP increase was small, it was still significant. We induced that kind of pulmonary hypertension, and right ventricle volume overload to let the animal survive and to continue the experiments. It is quite difficult in the animal model; we have to balance between something that is clinically relevant but still allows us to carry on the experiment.

Dr H. Fawzy *(Tanta, Egypt):* I have a question for the technique you have been using, those 3-D crystals. How did you route them out from the heart? Did you get it from the right atrium? I'm talking about the wires, as the wires are attached to the leaflets and go out of the right atrium; this can cause tricuspid regurgitation.

Dr Malinowski: The wires were externalized through the right atriotomy.

Dr Fawzy: So did you think that might be affecting the dynamics of the tricuspid if we have the pacemaker wire fixed to the right ventricle and going through the tricuspid and routing out of the heart, this can cause tricuspid regurgitation.

Dr Malinowski: I think not, there are loose wires that stay there. This kind of setup, with sonomicrometry crystals, has been validated in many, large animal models previously both on the mitral and tricuspid valves. I think this is the kind of model we have. I haven't heard of any influence of the crystal wires on the dynamics of whatever was studied. I am pretty sure that it shouldn't have any impact on that.

Dr Fawzy: And for the heights that you have it for the different points like the anteroseptal and the anteroposterior crystals, did you have any reference crystal you put it on the RV or somewhere that that can be referred to that?

Dr Malinowski: Yes, nine additional crystals were placed on the right ventricle to study the dimensions and the volume increase and to validate our model. For RV crystals, yes, most of them were placed epicardially and the ones placed inside were externalized.

Dr Fawzy: So you route out the one in the annulus through the right atrium and the one in the RV through the RV. The reference one, where did you route them out, from the RV? I'm talking about the wires again.

Dr Malinowski: We did not analyse the right ventricle function, it was just to validate our model to see if the right ventricle dilates due to volume overload we produced by increasing the pressure, and they were placed epicardial on the right ventricle and on the septum and externalized there. So that was our setup.