Conclusions—This study shows that although annular dilatation alone leads to TR, isolated PM displacement can also cause TR; annular remodeling strategies should be tailored in the setting of severe PM displacement. (Circulation. 2011;124:920-929.)

Key Words: annular geometry ■ papillary muscles ■ tricuspid regurgitation ■ valvular regurgitation

Functional tricuspid valve regurgitation (TR) is increasingly recognized as a source of morbidity in patients with severe left-sided valvular heart disease, particularly those with chronic mitral valve regurgitation. Early investigators believed that functional TR would resolve after surgical correction of left-sided valve disease and did not advocate concomitant tricuspid surgery. Subsequently, because it was recognized that severe TR often persisted despite corrective left-sided valve surgery and that reoperation for severe functional TR was perilous, interest grew in simultaneously repairing the tricuspid valve. Because tricuspid valve dilatation was recognized as the primary mechanism of functional TR, remodeling annuloplasty with a prosthetic ring became the treatment of choice of most surgeons. More recent studies have further clarified the role of annular dilatation centered around the right ventricular (RV) free wall portion of the tricuspid annulus as the primary lesion and thus target of remodeling strategies, and American and European guidelines recommend concomitant annular remodeling for severe annular dilatation or for moderate to severe TR in patients undergoing left-sided valve surgery.

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In reality, however, the mechanisms of functional TR remain somewhat obscure. Interestingly, functional mitral regurgitation has been much more carefully studied on the bench and by imaging and is recognized to involve primarily posterior papillary muscle (PM) displacement, leading to leaflet tethering and a compromised zone of leaflet coaptation; annular dilatation is an associated lesion. It is generally accepted that with annular dilatation, TR will occur, but the mechanism by which this occurs has yet to be demonstrated. As in functional mitral regurgitation, another possible mechanism for TR is PM displacement, which may result from RV dilatation. The role of PM displacement is much less well defined in functional TR, and this may have important implications for its surgical treatment.
clinical implications. It is possible that one of the mechanisms of recurrent TR after tricuspid valve repair with an annuloplasty ring, which occurs in up to 30% of patients undergoing surgery, is failure to address the leaflet tethering due to PM displacement. Understanding the mechanisms of TR will lead to improved diagnosis and treatment. The objective of this study is to develop methodologies to investigate and understand tricuspid valve mechanics in vitro, specifically, the effects of annular dilatation and PM displacement, both isolated and combined, and their relation to TR. This study also seeks to identify the mechanisms of functional TR through investigating individual changes in leaflet mechanics.

Methods

In Vitro Pulsatile Flow Loop

Experiments were conducted in an in vitro right heart simulator. The right heart simulator was designed on the basis of the Georgia Institute of Technology left heart simulator, which has been widely used to conduct studies on native mitral valves. Adjustments were made to accommodate for the lower pressures on the right side of the heart and the difference in valve anatomy. The main components of the simulator consisted of a clear acrylic ventricular and atrial chamber, an outer pump chamber with a flexible bulb pump connected to solenoid valves and a compressor, a pulmonary reservoir, an adjustable annulus plate, adjustable PM holding rods, and a rigid pulmonic outflow tract that houses a mechanical valve (Figure 1). The triggered solenoid valves controlled inflow of compressed air to expand and deflate the bulb, altering ventricular pressure and controlling the pulsatility of the system. Physiological conditions were maintained throughout experimentation, as follows: 5 L/min, 70 bpm, 40 mm Hg peak transvalvular pressure. Atrial and pulmonary pressures were maintained through the reservoir height. Cardiac output was controlled through resistances upstream of the tricuspid valve and downstream of the pulmonary valve (Omniscience, tilting disc mechanical valve). Saline solution 0.9% was used as a blood analog in the system.

Adjustable Annulus Plate

Physiological dilatation was achieved with a rigid septal segment and an adjustable anterior and posterior section, simulating reported physiological dilatation of the free wall section. Physiological shape change from a triangle, in a normal state, to an

Figure 1. Georgia Institute of Technology right heart simulator, used to simulate physiological flow and pressure. Valve was sutured to an annulus plate that was placed between the atrial and ventricular chambers with the papillary muscles attached to the papillary muscle rods.

Figure 2. An annulus plate was used to simulate physiological dilatation with a wire fed through a spring of the anterior and posterior segments and a rigid tube for the septal section. Thus, the anterior and posterior segments dilated while the septal section was maintained, with dilatation ranging from a normal annulus area (6 cm²) to 100% dilatation (12 cm²) with increments of 20%.
Valve Selection and Preparation
Because of the difficulty of obtaining numerous human tricuspid valves, porcine valves were used because they have been reported to have anatomy similar to that of humans.25 Porcine hearts were acquired from a local abattoir (Holifield Farms, Covington, GA) and transported to the laboratory in a cooler. Valves of appropriate size with an annulus area of 6 cm$^2$ were selected by measuring the annulus of the pressurized heart. The intact leaflet height and width were then measured with the use of a flexible ruler to confirm annulus of the pressurized heart. The intact leaflet height and width were then measured with the use of a flexible ruler to confirm appropriate size, as reported previously for human valves.13,24,26 Further selection criteria were based on chordae insertions and PMs in 3 groups (anterior, posterior, and septal) to allow for fixation in the simulator. Selected valves were excised with the annulus, leaflets, chordae, and PMs intact.

Valves were then sutured to the adjustable annulus plate with attachment points on the 3 commissures corresponding to annulus segments. The PMs were wrapped with Dacron cloth to prevent tearing of the PMs and to ensure that there was no restriction of the chordae. A button was then attached to the cloth to allow for attachment of the PMs to the adjustable PM rods in the simulator.

Papillary Muscle Positioning

Once the valves were placed in the simulator, the PMs were placed in a normal position with the septal PM (SPM) at the septal and anterior commissure, the anterior PM (APM) at the anterior and posterior commissure, and the posterior PM (PPM) at the septal and posterior commissure. PMs were positioned with the APM farthest from the annulus plane and the SPM closest, as reported for normal subjects with the use of magnetic resonance imaging.27 This was achieved with 3-dimensional adjustable rods with the use of a system of gears, as used in previous studies17,28 (Figure 3). The PMs were then adjusted apically to ensure that the chordae were neither taut nor slack. Slight adjustments were made once the simulator was running to account for valve-to-valve variability and to ensure proper leaflet coaptation. Displacement of the PMs was achieved by displacing the PMs on the septal wall simultaneously (PPM/SPM), simulating left ventricular (LV) dilatation,26 and by the sole displacement of the APM to simulate RV dilatation.30,31 Combined RV and LV dilatation was simulated by displacing all PMs simultaneously. All PMs were displaced by 10 mm. Specific displacement directions for each condition are shown in Figure 4.

Measurement and Data Collection
Markers were placed in a 2×2-mm grid on the entire surface of each leaflet with tissue-marking dye (Thermo Scientific, Pittsburgh, PA), and an image was taken before placement in the simulator to serve as a baseline and determine the original length of the leaflets along the line of interest. Video (DCR-TRV30, Sony, Japan) was recorded of the marked leaflets from the atrial side, perpendicular to the leaflets, throughout the cardiac cycle for all conditions.

Proper coaptation and no regurgitation were verified in the normal position before experimentation and served as control with normal annulus area and PM position. The following disease conditions were then simulated: isolated annular dilatation, isolated PM displacement, and combined annular dilatation and PM displacement. See Figure 4 for more details.

Bulk flow through the tricuspid valve was measured with an electromagnetic flow probe and was monitored with a flow meter (600 series, Carolina Medical Electronics, East Bend, NC). Atrial and ventricular pressures were measured with a differential pressure transducer (DP15–30, Validyne, Northridge, CA). Online flow and pressure signals were monitored with a LABVIEW-based (version 8.2, National Instruments, Inc, Austin, TX) in-house data acquisition system.

Data and Statistical Analysis
Flow was averaged over 15 cardiac cycles. TR was calculated by subtracting the closing volume as determined from the control condition. An Anderson Darling test was first performed to assess data normality. Each valve served as its own control, whereas measurements of disease conditions were compared with those made on the same valve for the normal position. A general linear model, with valve as a random factor, was used to investigate the effect of individual and combined annular dilatation and PM displacement on TR. Significance was determined at $P<0.05$ with a Dunnett post hoc test to determine which groups were significantly different from control.

Matlab (Matlab R2007a) was used to process all images and to convert visible leaflet markers to matrices. Residual leaflet length (RLL) was determined at the central coaptation point, where all 3 leaflets joined. RLL was calculated by subtracting the visible markers on each leaflet along the central line at peak systole from the number of markers at baseline and multiplied by 2 mm. Detailed RLL methodology can be found in the online-only Data Supplement.

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A general linear model was used to assess the effect of leaflet and disease on RLL, with significance at $P<0.05$. A Dunnett post hoc test was used to determine which groups were significantly different from control. Results have been divided into sections in which isolated annular dilatation, isolated PM displacement, and combined annular dilatation and PM displacement were investigated. Hemodynamic data as well as RLL data are presented for all cases. Absolute RLL measurements are reported for isolated annular dilatation demonstrating incremental changes with
dilatation, with percent change in RLL shown for annular dilatation and PM displacement, both isolated and combined, to demonstrate relative changes from control.

**Isolated Annular Dilatation**

Malcoaptation, as visualized by a hole, first appeared at 40% dilatation of the normal annulus area and increased in size with further dilatation. In all cases, the orifice was in the central region of the 3 leaflets, toward the septum.

Annular dilatation had a significant effect ($P=0.000$) on TR. A significant increase ($P=0.05$) in regurgitation fraction could be seen with 40% annular dilatation compared with the control ($7.9\pm3.4$ mL; $n=8$). Further dilatation $>40\%$ resulted in a significant ($P=0.05$) increase in TR from control (Figure 5A).

Leaflet ($P=0.000$) and annular dilatation ($P=0.000$) had a significant effect on RLL. RLL decreased with increasing annular dilatation for all leaflets ($n=8$) (Figure 5B). A significant difference ($P=0.05$) was seen in RLL of the posterior leaflet compared with both the anterior and septal leaflets, with the posterior having the largest RLL throughout. There was no difference in RLL between the anterior and septal leaflets. The anterior leaflet RLL was most affected by annular dilatation as it decreased from an initial RLL of $1.1\pm0.4$ to $0.2\pm0.2$ cm, with the largest decrease in percent change of RLL. The loss of valve competence and significant TR coincided with a minimum RLL of $0.5\pm0.2$ cm, as measured on the anterior leaflet. A negative correlation could be seen between RLL and TR for the posterior ($r=0.303$) and anterior ($r=0.685$) leaflets but not with the septal leaflet ($r=-0.423$), demonstrating that as RLL decreases, the amount of TR increases.

**Isolated Papillary Muscle Displacement**

A defect with absence of leaflet coaptation was visibly present in the center of the 3 leaflets with PM conditions,
which resulted in significant TR. The area of absent coaptation was located toward the center of the annulus. Coaptation of the leaflets with lateral displacement, away from the septum, of the SPM/PPMs was highly variable, with coaptation either closer to the septum or farther toward the center of the annulus. TR was significantly affected by PM displacement when annular size remained normal. Specifically, displacement of the APM, SPM/PPM, and all PMs in all directions created significant TR compared with normal PM position (Figure 6A). High variability in TR with lateral displacement of the S/PPMs resulted in a nonnormal distribution of the data, with TR of $11.4\pm5.4$ mL ($n=3$) and $1.7\pm0.3$ mL ($n=5$).

PM displacement caused an increase in the percent change in RLL for the posterior and anterior leaflets, whereas the septal leaflet was decreased for all conditions ($n=7$) (Figure 6B). RLL varied significantly by leaflet ($P=0.000$) but not with PM displacement ($P=0.967$). Septal leaflet RLL was significantly different ($P\leq0.05$) from posterior and anterior RLL, with the septal leaflet having a greater decrease in percent change. When compared within PM displacements with significant TR and across leaflets, only APM ($P=0.002$) and all PM leaflet displacement in all directions ($P=0.043$) had a significant effect on percent change in RLL. In both conditions, the septal leaflet was significantly ($P\leq0.05$) different from the anterior and posterior leaflets, with the posterior and anterior leaflets also being different from all PMs displaced. The highest decreases in RLL corresponded to the highest levels of TR and were $\geq27\%$.

![Figure 5](image-url) Hemodynamic and residual leaflet length (RLL) measurements for isolated annular dilatation. A, Tricuspid regurgitation fraction as an effect of annulus area times normal with significance (*$P<0.05$) compared with control (1); $n=8$. B, RLL as an effect of annulus area times normal, as shown for each individual leaflet: posterior (black), anterior (gray), and septal (light gray). SE is shown for RLL.

![Figure 6](image-url) Hemodynamic and residual leaflet length (RLL) measurements for isolated papillary muscle (PM) displacement. A, Tricuspid regurgitation fraction as an effect of PM displacement with significance (*$P<0.05$) compared with control (Cont); $n=8$). Bar colors correspond to PM that was displaced: black (no displacement), dark gray (anterior papillary muscle [APM]), gray (posterior papillary muscle/septal papillary muscle [PPM/SPM]), and light gray (all PMs). Lat. indicates lateral; Apil., apical, Ant., anterior, Post., posterior; and Sept., septal. B, Percent change in RLL from control as an effect of PM displacement as shown for each individual leaflet: posterior (black), anterior (gray), and septal (light gray). Threshold for significant regurgitation is shown with dashed line. SE is shown for RLL.
leaflet mobility (Figure 7B).

When the effect of annular dilatation and PM displacement was investigated for each leaflet, only the anterior leaflet RLL was recorded for all leaflets for all conditions (n=7). When the effect of annular dilatation and PM displacement was investigated for each leaflet, only the anterior leaflet RLL was significantly affected by PM displacement at dilations of both 40% (P=0.035) and 100% (P=0.049). Displacement of all PMs in all directions resulted in significant differences (P<0.05) in TR from annular dilatation at all levels measured. The largest levels of TR were reached at 100% dilatation, with all PMs displaced in all directions (19.6 ± 5.7 mL; n=8) (Figure 7A).

RLL was recorded for all leaflets for all conditions (n=7). When the effect of annular dilatation and PM displacement was investigated for each leaflet, only the anterior leaflet RLL was significantly affected by PM displacement at dilations of both 40% (P=0.035) and 100% (P=0.049). Displacement of all PMs in all directions resulted in significant differences (P<0.05) for anterior leaflet RLL from control for both levels of displacement. These changes resulted in an increase in anterior leaflet mobility (Figure 7B).

Combined Annular Dilatation and Papillary Muscle Displacement

Malcoaptation occurred at the junction of the 3 leaflets for all disease conditions because annular dilatation of 40% alone resulted in malcoaptation and remained with added PM displacement. With the largest levels of TR, in which the annulus was dilated by 100% and all PMs were displaced in all directions, the area of no coaptation was located more toward the anterior and posterior annulus segments.

When combined, annular dilatation (P<0.000) and PM displacement (P=0.000) had a significant effect on TR. Severe PM displacement, with all PMs displaced in all directions, was the only displacement that resulted in further increases (P<0.05) in TR from annular dilatation at all levels measured. The largest levels of TR were reached at 100% dilatation, with all PMs displaced in all directions (19.6 ± 5.7 mL; n=8) (Figure 7A).

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Discussion

In this study, we sought to understand the potential contributions of annular dilatation and PM displacement to functional TR. Thus, we will first discuss how annular dilatation alone plays a role in TR, then explain PM displacement, and finally explain how these 2 combine to significantly affect TR.

First, with our investigation of annular dilatation, we found that TR was highly dependent on annular dilatation, with significant TR occurring with only 40% dilatation, whereas it was seen at 75% dilatation in in vitro mitral valve studies.

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First, with our investigation of annular dilatation, we found that TR was highly dependent on annular dilatation, with significant TR occurring with only 40% dilatation, whereas it was seen at 75% dilatation in in vitro mitral valve studies. It may be the increased complexity in the mechanics of bringing 3 flexible surfaces together, compared with 2 with the mitral valve, that causes the tricuspid valve to leak earlier. A central zone of leaflet malcoaptation was consistent with annular dilatation, demonstrating the dependence on the 3 leaflets coapting simultaneously. Previous studies of TR repair have determined varying thresholds for determining when to clinically repair the annulus. In a study by Dreyfus et al,5 any tricuspid valve that was >70 mm in diameter by direct measurement was repaired, and Carpenter et al4 repaired the valve if the surgeon could insert 3 fingers into the annulus. In this study, annular dilatation of 40% corresponded to an annulus area of 8.8 ± 0.2 cm² and septal lateral diameter of 2.3 ± 0.05 cm, which is less than half the diameter recommended by Dreyfus et al for repair.

Although annular dilatation may cause TR, it is important to understand the mechanism behind it to gain insight in regard to why some patients with annular dilatation have TR and others do not. As the annulus dilates, the anterior and posterior sections are displaced, causing the posterior and anterior leaflets to be pulled away from the central coaptation line. This results in the reduction of RLL at the point of coaptation. When the RLL is sufficiently reduced, 3 leaflets cannot properly coapt, resulting in gaps, wrinkles, and leaflet mismatch (Figure 8A). A significant increase in TR coincided with a visible gap and an RLL of 0.5 cm. Minimum RLL of 0.5 cm of any leaflet may be a predictor of TR. Although the leaflets have RLL available for coaptation, the complexity of coaptation may require >0.5 cm of tissue from each leaflet to form a seal and prevent regurgitation. This study found
anterior leaflet RLL to be most affected by annular dilatation. We believe that this is because the anterior leaflet attempts to compensate for the increase in the orifice area created by dilatation of the annulus by reaching to cover it. Multiple studies have reported the anterior leaflet to be the longest and largest in area compared with the posterior and septal leaflets, which may be the body’s attempt to compensate for annular dilatation up to a certain level. In contrast to the anterior leaflet, the septal leaflet experienced little change in RLL as an effect of dilatation. This may be explained by previous studies that have reported the large number of chordae insertions to the septal leaflet from the septum and the possibility for restricted motion. Although it is accepted that annular dilatation may result in TR, it is not the only mechanism.

The effect of PM displacement on valve function has been investigated on the left side of the heart, both in vivo and in vitro, but has only recently been investigated on the right side. Although studies have yet to correlate PM displacement with TR, studies have correlated changes in RV geometry with increases in TR. It is believed that as the RV dilates, the PMs become displaced, resulting in leaflet tethering.

A recent study conducted by our group found that dilatation of the LV can affect RV PM position, with displacement of the septal PM toward the center of the RV in patients with a dilated LV. Although the previous study was unable to specifically correlate the presence of TR in these patients with the PM displacement, the results presented in this study may give us an idea of how the lateral displacement reported in patients may cause TR.

Next, through our investigation of PM displacement, we demonstrated that PM displacement alone may lead to significant TR. As with the case of isolated PM displacement, we found significant levels of TR in the presence of displacement of the APM and SPM/PPM, both individually and combined. Although isolated displacement of the PMs is rare, its relevance is important in understanding what may happen if the annulus is repaired and the PMs are left displaced.

In this study, proper coaptation of the leaflets was most affected by multidirectional PM displacement, with the changes in coaptation altered most with all PMs displaced in all directions, corresponding to changes in the mobility of the septal leaflet. With this increased mobility, the coaptation line of the 3 leaflets was more toward the center of the annulus, thus, the septal leaflet covered more of the orifice but was not long enough to do so because it is the shortest leaflet, and this resulted in a central zone of malcoaptation (Figure 8B). Although numerous PM displacements were investigated, severe displacement, with the APM, S/PPM, and all PMs displaced in multiple directions simultaneously, was the only condition that resulted in significant TR. Although lateral

Figure 8. A, Mechanism of regurgitation as seen with 100% dilatation. Anterior and posterior segments of annulus move away from center, creating a central hole toward the septum. With anterior leaflet extending to cover enlarged orifice and pulled away with dilatation, residual leaflet length is insufficient. APM indicates anterior papillary muscle; SPM, septal papillary muscle; and PPM, posterior papillary muscle. B, Mechanism of regurgitation as seen with displacement of the SPM and PPM. Mobility of septal leaflet is increased, thus changing the location of the coaptation line, resulting in a central malcoaptation in the center of the annulus because the septal leaflet is too short. This results in an insufficient residual leaflet length of the septal leaflet. C, Mechanism of regurgitation as seen with 100% dilatation and all papillary muscles (PMs) displaced in all directions. Anterior and posterior segments of annulus move away from center, and displacement of PMs alters coaptation line, resulting in a central coaptation, located toward the free wall. With anterior leaflet extending to cover enlarged orifice and pulled away with dilatation, along with an altered coaptation line, the residual leaflet length is insufficient to prevent regurgitation.
displacement of the SPM/PPM resulted in significant TR in some of the cases, the high variability did not allow for statistical comparison of a control group. This variability may be a result of the high variability in chordae insertion into the septal leaflet. In addition, septal leaflet RLL was highly affected by all PM changes because it experienced a reduction for all PM displacements. We believe that the septal leaflet was most affected by changes in PM displacement because it has many chordae insertions, with those coming from the SPM being the shortest.24

Finally, we found that when we combined the conditions of annular dilatation ≥40% and all PMs displaced in all directions, we achieved the highest levels of TR. A combination of multiple mechanisms results in the increased levels of TR that we observed. With the anterior and posterior leaflets pulled away from the central line of coaptation because of annular dilatation, increased leaflet mobility of the septal leaflet with SPM and PPM displacement, as discussed previously, and restriction of the anterior leaflet mobility with displacement of the APM, the highest levels of regurgitation were achieved (Figure 8C).

Clinical Implications

Although our study found that, in general, the valves failed to prevent regurgitation with 40% annular dilatation by area, the valves were of relatively similar size. Clinically, not all patients with annular dilation have regurgitation, and we believe that this may be due to variations in leaflet and subvalvular anatomy. We found that the anterior leaflet is responsible for compensating for an increase in orifice area as a result of annular dilatation. Thus, it may be important for the clinician to look not only at annulus size but at RLL to determine the potential for regurgitation. Although it may be difficult with current echocardiographic techniques to measure RLL in vivo, leaflet length, as measured just before coaptation, and coaptation length may be used to assess the valve. Future studies should be conducted to determine whether, in fact, patients with annular dilatation but no TR have longer leaflets, specifically the anterior leaflet.

As knowledge and experience with the tricuspid valve increase, changes are being made to clinical standards to incorporate this information. For example, American Heart Association/American College of Cardiology standards recommend repair for patients with tricuspid annular dilatation, specifically recommending annuloplasty to correct annular dilatation at the time of left-side repair when mild TR is present.10 This decision only requires the consensus of the clinicians and does not state the size at which the annulus is considered dilated. Current repairs for functional TR focus mainly on reducing the size of the annulus to correct annular dilatation. This study shows that it may also be important to restore the available leaflet length for coaptation, providing as much overlap as possible, specifically in the central region. In the presence of PM displacement, repairing the annulus to its true size with a ring annuloplasty may not be enough if the PMs are displaced enough to severely affect the mechanics of proper leaflet closure. With evidence that PM displacement alone can lead to TR, it may be important to investigate TR and ventricular size, both RV and LV, in addition to annulus area when a physician decides to treat a patient. Potential adjuncts in the setting of leaflet tethering and PM displacement could include downsizing ring annuloplasty (a mainstay repair strategy in functional mitral regurgitation) or leaflet augmentation19 to increase the amount of leaflet area available for coaptation. More specifically, evidence-based standards are ultimately needed to ensure that those who need treatment are treated appropriately. As more research is conducted to understand TR, these thresholds and possibly even predictors may be discovered.

Limitations

Measurement of RLL reported here is an estimate of the leaflet available for coaptation because it does not take into account any curvature, stretching, or compression that may occur when the leaflet surfaces join and thus may underestimate the actual length. The common technique that is practiced currently is to measure coaptation length, a surrogate of RLL, which provides a global measurement for the coapting leaflet length but cannot distinguish individual leaflets. The method used here allowed us to determine the contribution of each individual leaflet to coaptation. The technique cannot be applied directly to clinical applications but can be used only as a method to provide further information about leaflet mechanics.

Although a saddle-shaped annulus was used, similar to that reported in humans,8,19 in the control conditions for the PM displacement and combined cases, isolated annular dilatation studies had a flat annulus. We compared the closing volume between valves (n=8) with and without saddle with normal annulus size and PM positions and found no significant difference (P≤0.05). The role of the 3-dimensional annulus shape in proper valve function has yet to be determined. We do not believe that the presence or absence of a saddle in our study affected our results.

Although the rigidity of the RV is not physiological, we believe that it does not significantly affect the results of this study because our analysis is focused on the valve itself. Interactions between the RV and valve apparatus are simulated by displacement of the PMs themselves. Many previous mitral studies have been published with the use of a rigid ventricle box and have provided a mechanistic understanding of the valve under both physiological and pathological conditions.

Conclusions

This study demonstrates that whereas annular dilatation alone leads to TR, isolated PM displacement can also cause TR, with the most severe TR achieved with a combination of these. Alterations in either the annulus size or PM position result in the distortion of proper coaptation mechanics. With annular dilatation, the anterior leaflet attempts to compensate for the dilatation because it is the section that is being dilated. With isolated PM displacement, the septal leaflet
has the largest reduction in RLL, which may be attributed to numerous chordae insertions into the leaflet. In conclusion, addressing annular size and PM location may be important for effectiveness and durability of tricuspid valve repairs.

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Disclosures
None.

References
CLINICAL PERSPECTIVE

Current repairs for functional tricuspid regurgitation (TR) focus mainly on reducing the size of the annulus to correct annular dilatation and often result in recurrent TR, requiring additional treatment. We believe that understanding the mechanisms responsible for TR will lead to improved diagnosis and treatment. Although it is accepted that annuloplasty is beneficial in the correction of TR, this study shows that it may also be important to restore the available leaflet length for coaptation, providing as much overlap as possible, specifically in the central region. In the presence of papillary muscle displacement, reducing the annulus to its normal size with a ring annuloplasty may not be enough if the papillary muscles are displaced enough to severely affect the mechanics of proper leaflet closure. With evidence that papillary muscle displacement alone can lead to TR, it may be important to investigate papillary muscle position and ventricular size, both right and left, in addition to annulus area when the physician decides on the technique for tricuspid valve repair. More specifically, evidence-based standards are ultimately needed to ensure that those who need treatment are treated appropriately. As more research is conducted to understand the mechanisms of TR, these thresholds and possibly even predictors may be discovered.
In Vitro Characterization of the Mechanisms Responsible for Functional Tricuspid Regurgitation
Erin M. Spinner, Patrick Shannon, Dana Buice, Jorge H. Jimenez, Emir Veledar, Pedro J. del Nido, David H. Adams and Ajit P. Yoganathan

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SUPPLEMENTAL MATERIAL:
Detailed Residual Leaflet Length Methodology

PART ONE: Extract Images from Video

1. Extract the static image.

2. Extract the peak systolic image from the video file. This is determined as the frame when the valve is fully closed and does not move.

3. Save each systolic images and label accordingly.
4. This step is repeated for all conditions, for all valves.

PART TWO: Convert dots to Matrix

Note: The following steps are completed using Matlab.

1. Count the dots in each row and the number of rows.
   a. Static Image: All dots are counted.
   b. Systolic Image: Only visible dots are counted.
2. With the information create a matrix.
3. Determine the row and column in which the references are located and identify this within the matrix. Shown as an x in the below image.
4. Repeat this for all conditions.

5. Overlay the static and each condition and plot.

6. Repeat for each leaflet, condition and valve.

PART THREE: Measuring RLL

1. Determine the line of interest to measure RLL, central coaptation.

2. Locate the line of interest on the leaflet plot. This is aided by using the reference as a land mark.

3. Determine RLL. RLL = Static # of markers - # of visible markers at condition
   For the case shown: RLL = 16-7=9 markers, each marker is spaced 0.2 cm, thus RLL=1.8 cm
4. Repeat for each leaflet, condition and valve.