Altered right ventricular papillary muscle position and orientation in patients with a dilated left ventricle

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Objective: To investigate the impact of left ventricular dilatation on right ventricular papillary muscle displacement.

Methods: Thirteen patients underwent high-resolution cardiac magnetic resonance imaging at Emory University Hospital: Seven patients with congestive heart failure and a dilated left ventricle composed the dilated left ventricular group, and 6 normal subjects were used as a control. A total of 120 cardiac magnetic resonance imaging slices were acquired in a short-axis view at end diastole for each subject. Cardiac magnetic resonance imaging slices were used to identify the papillary muscle tip position in 3-dimensional coordinates for the septal, posterior, and anterior papillary muscles. The centroid of the papillary muscle coordinates was used as the reference point for comparison between subjects. The relative orientation between the right ventricular papillary muscles was evaluated and compared between the dilated left ventricular group and normal subjects.

Results: Dilatation of the left ventricle resulted in a significant (P = .05) displacement of the septal right ventricular papillary muscle toward the centroid: normal group, 0.0285 ± 0.036 mm/mm versus dilated left ventricular group, 0.1437 ± 0.026 mm/mm. More specifically, the septal papillary muscle significantly (P = .03) moved away from the septal wall (normal group: 0.61 ± 0.09 mm/mm, dilated left ventricular group: 0.379 ± 0.037 mm/mm). Specific locations of all 3 right ventricular papillary muscles were reported for normal subjects and patients with a dilated left ventricle.

Conclusions: Patients with a dilated left ventricle have significantly increased displacement of the septal right ventricular papillary muscle away from the septum when compared with normal controls. This demonstrates pathophysiologic contribution of the left ventricle to specific papillary muscle alterations within the right ventricle.

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Left-sided heart disease is traditionally thought to affect the right side of the heart through pulmonary hypertension1,2 and increased afterload on the right ventricle (RV). Left ventricle (LV) dilatation and dysfunction, even in the absence of pulmonary hypertension, can have a detrimental impact on the RV and may be a potential mechanism for high occurrence of tricuspid regurgitation (TR) in patients with mitral regurgitation. In this study, it is hypothesized that pulmonary hypertension geometric changes on the left side may also directly affect the right side because the RV and LV are connected through the interventricular septum.3 The volume in which the LV and RV can fill is restricted by the pericardial sack; thus, a dilated LV may enlarge not only into the pericardial space but also into the RV because it is a lower pressure chamber. Changes in RV geometry induced from LV geometric changes may contribute to secondary TR through papillary muscle (PM) displacement, improper muscle contraction, or other complications. Changes in RV geometry may lead to alterations in the positions of the PMs of the tricuspid valve (TV) because they are directly connected to the ventricle. Jouan and colleagues4 and Hiro and colleagues5 investigated the RV PM position in sheep over the cardiac cycle, but they did not report how these positions may change in a diseased state. Studies have implicated RV dilatation as a cause of anterior PM displacement.6,7 The speculated mechanism for pathologic PM displacement is that as the free wall of the RV dilates, the anterior PM, located on the free wall of the ventricle and attached to the valve leaflets through chordae tendineae, moves outwardly, thus tethering the TV leaflets.8 In addition to the proposed motion of the anterior PM due to RV dilatation, it is of interest to determine whether the PMs on the septal wall, the posterior and septal PMs, are displaced as the result of LV dilatation. Although it is probable that the septal and posterior PMs are affected by LV dilatation through the interventricular septal motion, it has yet to be demonstrated in...
Abbreviations and Acronyms

- ARP = annulus reference plane
- LV = left ventricle
- MRI = magnetic resonance imaging
- PM = papillary muscle
- RV = right ventricle
- TR = tricuspid regurgitation
- TV = tricuspid valve

human subjects. The specifics of which PMs are displaced with specific causes, the direction of displacement, and how much they are displaced remain unknown.

If in fact there is an effect on the PMs in the RV as the result of LV dilatation, this may further support the belief that the dynamics on the left side of the heart affect the right side of the heart. We hypothesize that geometric changes in the LV, as a result of LV dilatation, may also alter RV geometry and displace the PMs located on the septal wall.

This study identifies and compares the position of RV PMs in normal subjects and patients with LV dilatation to quantitatively establish the RV PM positions and the influence of LV dilatation on RV PM displacement. Magnetic resonance imaging (MRI) was the imaging modality chosen for this study because it has the potential to image cardiac structures in high resolution with no radiation dosage.

MATERIALS AND METHODS

Patient Selection

A total of 13 subjects were scanned as part of a high-resolution geometric protocol at Emory University Hospital Midtown, Atlanta, Georgia. Of these, 7 were patients with congestive heart failure (dilated LV group) with the following implications: mild (n = 2) to severe (n = 5) mitral valve regurgitation, a dilated LV (LV end-diastolic volume 234.7 ± 70.8 mL), mean age of 65 ± 14 years, New York Heart Association class 2 (n = 2), 3 (n = 4), 4 (n = 1), pulmonary regurgitation (n = 3), pulmonary hypertension mild (n = 3) to moderate (n = 3), normal systolic function (n = 6), and mild TR (n = 5). The remaining 6 were normal subjects used as controls for the study (normal group), with no known heart conditions and a mean age of 25 ± 2.5 years (n = 5). The study was approved by the Emory University Institutional Review Board for human subjects, patient privacy was protected according to Health Insurance Portability and Accountability regulations, and all patient identifiers were removed from data used in this study. Informed consent was obtained from all participants in the study.

Image Acquisition

All scans were acquired on a Philips Intera CV 1.5 T (Andover, Mass) system using a cardiac coil. High-resolution, electrocardiogram, and navigator echo-gated ‘‘whole heart’’ gradient echo sequence was used in the short-axis orientation spanning both the RV and LV during end diastole. Pixel size ranged from 0.53 to 0.70 mm, slice thickness ranged from 1.5 to 2.0 mm, and echo time ranged from 1.3 to 2.1 ms. Approximately 120 slices were acquired at end diastole, as confirmed with cine images, to cover the entire LV and RV.

Data Processing

XnView 1.92 (Pierre-Emmanuel Gougele, France) was used for visualization and post-processing. References in the x, y, and z direction were identified to compare the PMs in 3-dimensional space. An annulus reference slice was identified to calculate the z distance relative to the annulus reference plane (ARP). The annulus plane was selected as a reference plane so the relative proximity and change could be related to the annulus plane. The ARP was identified as the slice in which the right coronary artery and aortic leaflets were visible. The diameter of the ascending aorta was measured and recorded in the ARP. PM tip position was manually identified in the various images by identifying the regions of low intensity (the PM) among regions of high intensity (blood) in the RV (Figure 1). The first slice in which the PM was visible was recorded to obtain the z distance with respect to the ARP. The x and y coordinates of the center of the PM tip were recorded to give the full 3-dimensional position.

Three-dimensional coordinates were recorded for all 3 PMs: septal, posterior, and anterior in all subjects. All images were registered to the MRI coordinate system for purposes of position and orientation.

The centroid of the 3 PM coordinates was calculated for the 3 PMs for each patient and used as a reference point. All distances were normalized by ascending aortic diameter to minimize patient variability. X, y, and z distances from the centroid (Figure 2, A) were calculated for each PM and the distance from each PM to the centroid (Figure 2, C). In addition, the triangle formed by the 3 PMs was used to investigate the relative position of the PMs. The distance of each triangle side length (Figure 2, B) and area of the triangle formed by the 3 PMs (Figure 2, D) was reported.

PMs were classified into groups on the basis of their location; (i) anterior: located on the free wall; (ii) septal: located on the anterior portion of the septum; and (iii) posterior: located on the posterior portion of the septum.

Statistical Methods

An Anderson Darling test was used to test for normality. A 2-sample Student t test was performed to determine statistically significant differences between normal patients and patients with heart disease. Values are expressed as mean ± standard error. A post hoc power analysis was conducted on groups in which a significant difference was noted to confirm appropriate power (≥0.7).

RESULTS

Normal Group

All measurements for normal PM position can be found in Table 1. It is notable that the sides of the triangle formed by the PMs were of similar length. The septal PM was the furthest from the centroid with a normalized distance of 1.07 ± 0.08 mm/mm. The anterior PM was the most apical and furthest from the ARP of the 3 PMs (0.20 ± 0.04 mm/mm), with the septal PM the closest to the ARP (0.85 ± 0.07 mm/mm). A 3-dimensional depiction of normal PMs and their position in the heart can be seen in Figure 3.

Dilated Left Ventricular Group

All measurements for diseased PM position are shown in Table 1. The same relative position and orientation of all 3 PMs was noted in both groups. The septal PM was furthest from the centroid (0.76 ± 0.11 mm/mm). The anterior PM was furthest away from the ARP (0.15 ± 0.03 mm/mm), and the septal PM was the closest (0.74 ± 0.05 mm/mm).

Differences

Significant differences between normal patients and patients with heart disease were only detectable with the septal PM. The differences were observed in the y distance
(P = .03) of the septal PM (diseased 0.38 ± 0.04 mm/mm; normal 0.61 ± 0.09 mm/mm) and thus displaced in a posterior direction. In addition, the septal PM was also displaced toward the centroid (P = .05) in patients with heart disease (dilated 0.029 ± 0.04 mm/mm; normal 0.144 ± 0.03 mm/mm), and therefore away from the septum. Significant differences of PM displacement are shown in Figure 4.

**DISCUSSION**

To our knowledge this is the first report describing the specific locations of all 3 RV PMs in normal patients and patients with a dilated LV using cardiac MRI. Comparison between RV PM position in normal patients and patients with a dilated LV revealed a significant difference in the relative position of the septal PM. This study found that LV dilatation resulted in the inward displacement of the septal PM toward the centroid of the 3 PMs, which may be due to the displacement of the septal wall into the RV with the increased LV volume. LV dysfunction has typically been related to RV dysfunction through pulmonary hypertension, but changes in the LV geometry may be transferred to the RV though the interventricular septum. Anyanwu and colleagues proposed LV dysfunction or dilatation as a mechanism of TR with normal pulmonary hypertension. Weber and colleagues discuss in detail interventricular septum effects and the displacement of the septum into the RV with acute
LV distention. Our results clearly show the interplay of ventricle volume and the effect of the septum displacement into the RV on the PMs. In addition, we believe that motion of the septal wall into the RV may translate to the septal leaflet as it is attached to the septum through chordae tendineae and the septal and posterior PMs. Specific identification of the effects of the septal PM displacement on the leaflets was outside the scope of this study, and therefore we cannot conclude whether this displacement leads to TV tethering or prolapse. However, there is evidence that prolapse and tethering of the leaflets result in TR. As the PMs are displaced, the tension in the chordae may be decreased, but this study investigated only the PM motion. We speculate that the PM displacement observed in the group with heart disease may have contributed to the TR present in 5 of the 7 patients. Our analysis is a first step in demonstrating that left-sided heart disease has a direct impact on right-sided anatomic structures, resulting in a configuration that may lead to TV leaflet prolapse and tethering. Further studies are needed to investigate the direct impact of PM displacement on valve function. Such studies are currently under way to confirm these phenomena.

LV, Left ventricle; SE, standard error; PM, papillary muscle. *P ≤ .05 compared with normal.

FIGURE 3. Anatomic representation of PM position in the RV. PM, Papillary muscle.
most common surgical intervention for non-rheumatic TV focuses on reducing the dilated annulus to its original size with the placement of an annuloplasty ring. Studies have found that restoring the annulus to a normal size may not eliminate TR completely, and in many cases it returns.\(^\text{16,17}\) Thus, solely reducing the annulus may be insufficient to permanently correct TR.\(^\text{16,18}\) Although PM displacement may be one contributor to this recurrence of TR, this study also demonstrates that it may be important to assess the impact of the LV size and septal motion on RV function, even before surgery is attempted. These outcomes suggest that the underlying pathophysiology associated with TR is perhaps not well understood. Previous studies have attempted to correlate RV PM displacement to TR and found TV tethering and restriction of leaflet mobility caused by PM displacement to be an independent predictor of TR after TV annuloplasty.\(^\text{7,9}\) Although the relation of LV dilatation to durability of TV repairs has yet to be proven, this study demonstrates the geometric relation between the 2 ventricles. Futures studies may aim to determine pre- and postoperative PM position and determine the relation to patient outcome. This study demonstrates a useful tool that can be implemented to measure PM position in both normal patients and patients with heart disease.

**Limitations**

This study was limited to the effects of LV dilatation on the posterior and septal PMs, because they are located on the septal wall. Because of the retrospective nature of this study, only PM positions at end diastole were acquired. Future studies of the dynamic position of the PMs through the cardiac cycle are required. Although the images used for this study were at end diastole, after careful observation of the ventricle throughout the cardiac cycle we found the position of the PMs did not move from the time of analysis until the beginning of systole when the ventricle began to contract. It is of importance to note that although TR is a systolic phenomenon, the displacement of the PMs elucidated in this study is expected to have an effect on the TV apparatus during systole and may even be further exacerbated. This can be determined by future studies that investigate the PMs not only at peak systole but also throughout the entire cardiac cycle.

**CONCLUSIONS**

Dilatation of the LV resulted in a significant displacement of the septal PM position. This change in PM position is due to change in geometry of the LV with dilatation and movement of the septal wall into the RV. Implications of the displacement of septal and posterior PMs in the role of TV function are yet to be determined.

**References**


