# Assessment of Papillary Muscle Function Using MRI Tissue Tagging

**R. M. Setser[1], M. S. Kotys[2], T. Boonyasirinant[1], and S. D. Flamm[1]**

[1]Imaging Institute, Cleveland Clinic, Cleveland, Ohio, United States, [2]Philips Healthcare, Cleveland, Ohio, United States

## Introduction

The papillary muscles (PM) are an integral component of the mitral valve apparatus and their function is intrinsically linked with left ventriclular (LV) function [1]. Previous studies have shown that PM function can be impaired in patients with myocardial infarction [2-3], with potential implications for mitral regurgitation. Tagged MRI can be used to quantify LV strain [4], but to date has not been used to quantify PM strain. However, MRI might be advantageous when imaging mobile structures such as PMs, as images can be acquired in any orientation and the tag lines can be specified in any direction. Thus, the purpose of this study was to develop a technique for quantifying PM function using tagged MRI and to establish a range of normal values for subsequent comparison to patient data.

## Methods

Healthy volunteers (n=6, 4M/2F, age 37±10 years (mean±std dev)), and patients without previous myocardial infarction (n=2, 1M/1F, age 19/38 years), were imaged using a clinical 1.5T MRI scanner (Achieva XR, Philips, Best, The Netherlands) with 16 element phased-array coil (SENSE Torso XL, Philips). Scout images were acquired to define LV geometry, followed by short axis bSSFP cine images from LV base to apex. A pseudo-long axis image orientation was defined in which both PMs were visible throughout the cardiac cycle. Line tagged images (TR/TE 6/2 ms, 1x2mm pixels, 8mm thick, 6mm tag spacing) were acquired in this orientation with the tag lines oriented perpendicular to the PMs (Figure 1).

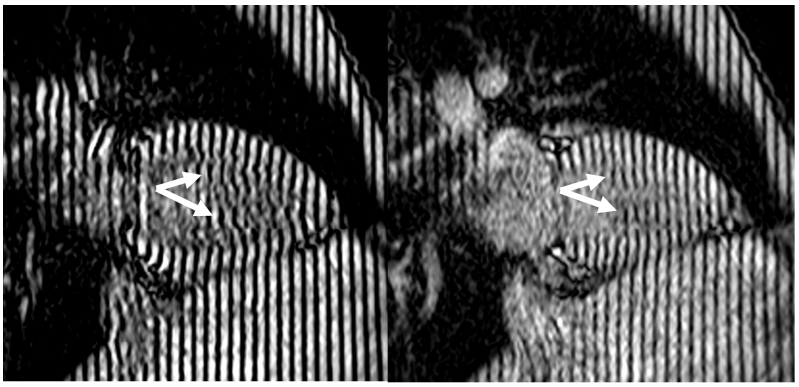

Figure 1. Line tagged images at end diastole (left) and end systole (right). Papillary muscles are denoted by arrows.

In each image frame, a line profile was defined along each PM and in LV myocardium adjacent to each PM. Signal intensity along each line was used to semi-automatically identify tag line spacing in each image frame. End systolic PM strain and LV longitudinal strain were computed as $L_{ES}$-$L_{ED}$ / $L_{ED}$, where L denotes average tag spacing at end systole (ES) end diastole (ED), respectively.

## Results

Strain in the anterior PM (APM) and posterior PM (PPM), as well in the anterior and inferior LV wall, are shown in the Table at right (negative values indicate shortening). Overall, APM strain was significantly greater than ANT LV (p=0.02); However, the difference between PPM strain INF LV only trended towards significance (p=0.07). In addition, there was no significant difference between APM and PPM strain (p=0.55), or between ANT LV and INF LV strain (p=0.64).

| Study | End systolic Strain (%) | | | |
|---|---|---|---|---|
| Subject | APM | PPM | Ant LV | Inf LV |
| V1 | -20 | -15 | -16 | -12 |
| V2 | -24 | -24 | -18 | -19 |
| V3 | -27 | -22 | -17 | -26 |
| V4 | -23 | -13 | -16 | -11 |
| V5 | -23 | -19 | -18 | -19 |
| V6 | -15 | -26 | -18 | -20 |
| P1 | -21 | -18 | -13 | -16 |
| P2 | -21 | -25 | -17 | -16 |
| Avg (SD) | -22 (3) | -20 (5) | -17 (2) | -17 (5) |

## Discussion / Conclusions

Our results are consistent with previous reports. Using tissue Doppler ultrasound, Kang et al. reported -24±5% PPM strain and -17±3% INF LV strain [3]. In addition, Petitjean et al. reviewed the MRI literature and reported normal LV longitudinal strains ranging from -15% to -18% (data from 5 separate studies) [4].

PM function is intrinsicaly linked with global LV function and serves to maintain LV shape [1]. It has been shown that inferior wall infarcts often involve the PPM, which impairs its function accordingly [2]. However, further investigation is needed to ascertain the relationship between PM infarct status and function and mitral regurgitation.

## References

**1**. Carabello BA. J Am Coll Cardiol 2008;52:319-26. **2**. Rayhill SC, et al. Circ Res 1994;74:1179-87.
**3**. Kang SJ, et al. J Am Soc Echocardiogr 2005;18:815-20. **4**. Petitjean J, et al. J Cardiovasc Magn Reson 2005;7:501-16.