

Technologist presentation

## Is infarct location a prediction of valvular enhancement?

Ronald B Williams\*, Mark Doyle, Vikas K Rathi, June A Yamrozik and Robert WW Biederman

Address: The Center for Cardiovascular Magnetic Resonance Imaging, The Gerald McGinnis Cardiovascular Institute at Allegheny General Hospital, Pittsburgh, PA, USA

\* Corresponding author

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### Introduction

Every day in CMR, delayed hyperenhancement (DHE) is utilized for evaluation of the patient's extent of myocardial damage from an infarction. In the past, following an MI, we have shown that DHE signal may be seen in the post-MI valvular apparatus, not limited to the myocardium. Also, dilation of the mitral annulus was noted over time in some patients. However, it is not known if the infarction location influences which valves enhance, in particular, whether there is a local or a global influence on valvular enhancement.

### Purpose

We hypothesize that the location of the infarct does not influence which valves enhance.

### Methods

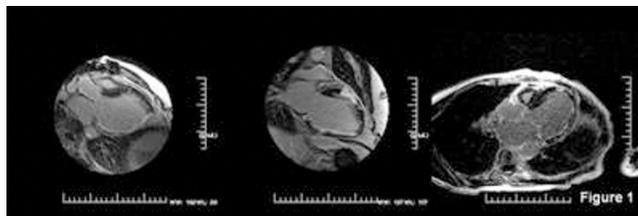
One hundred-one (101) post-MI CMR examinations (chronic MI: 97% >6wks; 3% acute <6 wks) were retrospectively reviewed for their infarct location, and whether mitral valves (MV), tricuspid valves (TV), and aortic valves (AV) exhibited any DHE signal after the administration of intravenous gadolinium contrast agents, either 0.2 mmol/kg Magnevist (Berlex, Wayne, NJ.) or 0.15 mmol/kg MultiHance (Bracco, Princeton, NJ). The areas observed were anterior, anterior septum, septum, inferior, inferior septum, lateral, anterior lateral, inferior lateral, and apical areas of the myocardium. The location of the infarct was then assigned anatomically to LAD, RCA, and the LCX coronary distributions.

### Results

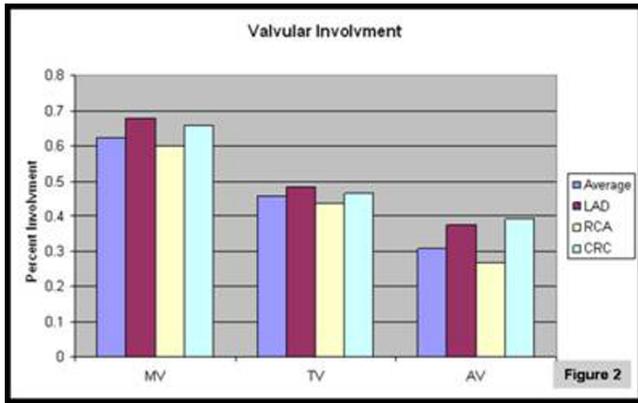
CMR images in 101 patients (72 males,  $64.5 \pm 29.77$  years; 29 females,  $62.9 \pm 12.87$  years) were retrospectively reviewed for infarction location. See Figure 1. Globally, the valves affected were MV (66%), TV (45%) and AV (30%). When analyzed by infarct territory (LAD, RCA and LCX) there was not a trend present showing any significant difference from the global distribution, See Figure 2

### Conclusions

The reason for valvular enhancement status post MI is not clearly understood. Using CMR DHE, and correlating the location of the areas of DHE signal, it suggests that the distribution of valvular enhancement is not influenced by the coronary artery involved. This points to a global rather than regional involvement of the valvular apparatus.



**Figure 1**



**Figure 2**

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