Myocardial Perfusion Study of Heart Failure Swine with Semi-quantitative Analysis

T. Song[1,2], M. N. Hood[2,3], J. A. Stainsby[4], and V. B. Ho[2,3]

[1]Global Applied Science Laboratory, GE Healthcare, Bethesda, MD, United States, [2]Radiology, Uniformed Services University of the Health Sciences, Bethesda, MD, United States, [3]Radiology, National Naval Medical Center, Bethesda, MD, United States, [4]Global Applied Science Laboratory, GE Healthcare, Toronto, ON, Canada

Introduction

Cardiac perfusion imaging is a recognized method for non-invasive evaluation for myocardial ischemia [1]. However, it is unclear how global heart failure affects myocardial perfusion. In this study, we explore semi-quantitative perfusion in a Yorkshire swine heart failure study.

Methods

In this study, five Yorkshire swine were implanted with pacemakers with the intent of producing tachycardia-induced heart failure. Each pig was scanned on a 1.5T MR scanner (GE Medical System, Waukesha, WI, USA) at baseline and at time of heart failure. For MR acquisition, we used a standard fast gradient echo perfusion acquisition with an 8-channel cardiac coil, 40 phases, 128x128, 7mm slice, 2RR, and with free breathing. 0.2mmol/kg dose of Gadoteridol was injected intravenously at 1ml/sec. Signal intensity (SI) time courses were evaluated for the myocardium and the blood pool. Ejection fraction, arrival time, peak time, slope, maximum upslope, and contrast enhancement ratio (CER) were calculated using Cine Tool (GE Medical System, Waukesha, WI, USA). Indexes are defined as the value of myocardium weighted by the value of the blood pool. Specifically, semi-quantitative analysis of slope index, maximum upslope index, and CER index were calculated. A paired t-test was evaluated in each measured parameter group to identify the statistical difference between baseline and heart failure scans.

Results

Average left ventricular ejection fraction was $45\% \pm 4\%$ at baseline and was $16\% \pm 7\%$ at heart failure, with negative myocardial delayed enhancement (MDE) showing no infarcts. Example signal intensity curves for both cases are shown in Fig. 1. The signal intensity (SI) curves of baseline myocardial perfusion were sharper and narrower than those at heart failure. In comparison, the myocardial perfusion curves at heart failure were slower and wider. The semi-quantitative parameter results are listed in Table 1. The box plot of the data for each pair of quantitative metrics is shown in Fig. 2. Statistically significant differences ($p<0.03$) were seen in arrival time, peak time, and the slope only and for maximum upslope, and contrast enhancement ratio (CER) ($p<0.07$). On the contrary, indexes did not yield significant differences between normal and heart failure cases. This is similar to the findings in [1] for ischemia cases. In addition, correlations between all parameters with ejection fractions were also computed as shown in Table 1.In all parameters with statistical significance, peak time was highly correlated with ejection fraction (EF: 85%) and maximum slope was least correlated with EF (42%); the remaining three parameters were moderately correlated with EF (63%-66%). This suggests that decreases in cardiac output accounts for a large portion of the changes in cardiac perfusion dynamics, contrary to ischemia cases. However, changes in EF cannot completely explain all changes observed in the data. Remodeling and physiological changes other than cardiac output during global heart failure may also affect the dynamics of the contrast agent.

Conclusions

Currently, the relationship between myocardial perfusion and global heart failure has not been well explored. In this study of tachycardia-induced heart failure in swine, perfusion showed delayed arrival time, delayed peak time, and reduced slope of enhancement in myocardium ($p<0.03$). With $p<0.07$, it showed decreased maximum upslope and increased CER. It was also found that changes in cardiac output were a major but not the sole source of changes in the bolus dynamics. Future work is needed to explore the potential role of myocardial perfusion imaging applied to heart failure patients, as the ability to identify coexistent myocardial ischemia may be problematic in this population.

References

[1] Li-Yueh Hsu, et al. JMRI 23:315–322, 2006

Table I: Parameters with mean and standard deviation values from perfusion analysis.

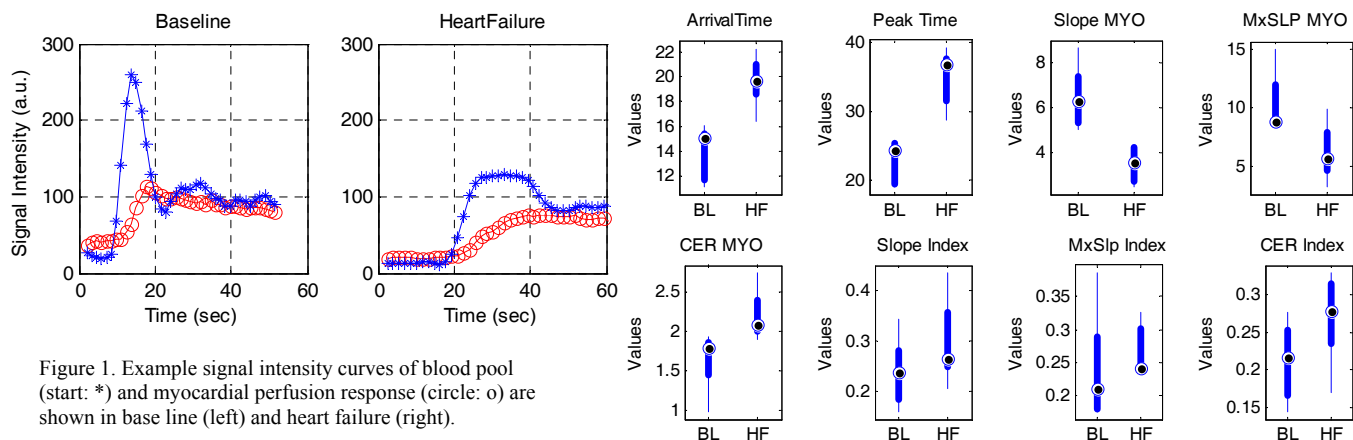|	Arrival Time (sec)	Peak Time (sec)	Slope (a.u.)	MxSlope (a.u.)	CER (a.u.)	SlopeIndex (a.u.)	MxSlopeIndex (a.u.)	CERIndex (a.u.)
Baseline	13.9 ± 2.2	22.7 ± 3.2	6.5 ± 1.4	10.4 ± 2.8	1.7 ± 0.4	0.24 ± 0.07	0.24 ± 0.09	0.21 ± 0.05
Heart Failure	19.6 ± 2.1	34.9 ± 4.2	3.4 ± 0.8	6.3 ± 2.5	2.2 ± 0.3	0.30 ± 0.09	0.27 ± 0.04	0.27 ± 0.06
p-Value	0.012	0.016	0.025	0.068	0.067	0.330	0.624	0.246
EF Correlation	63%	85%	66%	42%	64%	26%	9%	38%

Figure 1. Example signal intensity curves of blood pool (start: *) and myocardial perfusion response (circle: o) are shown in base line (left) and heart failure (right).

Figure 2. Box plot of the baseline and heart failure groups: arrival time, peak time, slope, maximum upslope, CER, slope index, maximum upslope index (MxSlp), and CER index from left to right. The box has lines at median (circle dot), the lower and upper quartile values (wider box bar end). Whiskers extend from each end of the box to the adjacent values in the data.