Noninvasive Assessment of Ventricular Contractility During Cardiac MRI Examinations

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Introduction

Ventricular contractility defines the innate blood pumping ability of the left ventricle (LV), and its quantification, without the confounding effects of preload and afterload, is important in assessing left ventricular performance. However, traditional measures of ventricular function, such as ejection fraction, are influenced by preload and afterload, and are therefore poor indicators of contractility. Several load-insensitive measures of contractility have been derived, but their calculation requires continuous measurements of central pressure. Central pressures are not currently monitored during cardiac MRI exams, so development of a method to do so could significantly enhance its diagnostic value.

We have implemented techniques to estimate central pressures and ventricular contractility noninvasively during cardiac MRI exams. We used arterial tonometry to noninvasively record radial artery blood pressure waveforms. Aortic pressure was then calculated using a generalized frequency domain transfer function [1]. Aortic pressure was combined with MRI-derived LV volume to compute normalized maximal ventricular power ($nPWR_{max}$), defined as the maximum product of instantaneous left ventricular pressure and aortic outflow, divided by end diastolic volume squared, which has been shown previously to be a load independent index of contractility [2]. To validate the contractility and afterload sensitivity of $nPWR_{max}$, we performed a study on healthy volunteers in which contractility was increased by dobutamine and afterload was increased using methoxamine during a cardiac MRI exam.

Methods

Ten healthy volunteers, with no history of cardiovascular disease, were recruited (8M/2F, age 27.5 ± 5 years). Tonometer radial artery pressure and MR images were first acquired at baseline contractility (BL1). Intravenous dobutamine was then administered at two doses, 10 mg/kg/min (DB1_10) and 20 mg/kg/min (DB1_20), with pressure and image acquisition at each dose. Wall motion was assessed before advancing to the next dose. After the stress test, volunteers were allowed to recover and contractility was assessed again (BL2). Intravenous methoxamine was then administered at 15 mg/kg/min until systolic blood pressure had increased by 30-60 mmHg, and data was acquired (METH). This was followed by a second dobutamine stress test, identical the first (DB2_10, DB2_20). Subjects were allowed to recover and data was acquired a last time (BL3). Sphygmomanometer (cuff) pressure was recorded at each stage of the study for calibration of the tonometer waveform.

Imaging was performed on a 1.5T MR scanner (Philips Gyroscan ACS-NT, PT6000) using an ECG triggered, breath-hold, segmented gradient echo-echo planar pulse sequence: FOV 350 mm, RFOV 80%, TR 1 R-R interval, TE 5.5 ms, 128x128 matrix, flip angle 35, EPI factor 15. 12 short axis, 9 mm thick slices were acquired with no gap. Breath-hold duration was under 20 seconds.LV volume was computed by manually tracing endocardial borders in systolic frames of each slice. Approximately 10 consecutive, steady state beats were selected from each tonometer data set. Segments were parsed into individual beats, resampled to 60 Hz to remove heart rate variations and filtered to reduce noise. Individual beats were then calibrated using cuff systolic and diastolic pressures, and averaged point by point. Aortic pressure was computed using a generalized frequency domain transfer function relating radial artery and aortic pressure, which we generated in a separate study using data from patients undergoing routine cardiac catheterization. Ventricular contractility was then computed as $nPWR_{max} = (dV_{LV}/dt*P_{Ao})_{max}/V_{ed}^2$, with aortic pressure used as a surrogate to LV systolic pressure. Statistical differences between groups were assessed by analysis of variance with Scheffe subtesting.

Results

No significant difference in ejection fraction was seen at any of the study stages (compared to BL1). Results for $nPWR_{max}$ are shown in Figure 1. A detectable difference in $nPWR_{max}$ was seen between the 20 mg/kg/min dobutamine stages (DB1_20, DB2_20) and baseline (BL1) with no significant differences between BL1 and the other stages.

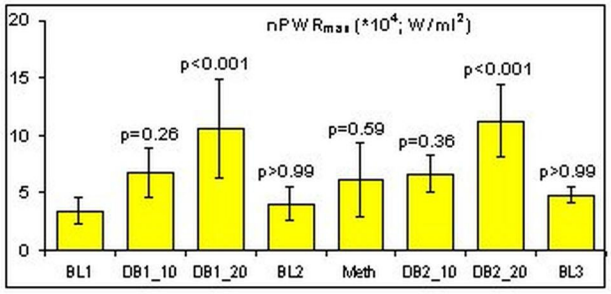

Figure 1. Normalized maximal ventricular power as a function of study stage. P-values shown are versus BL1. See Methods section for stage descriptions.

Discussion

We have used arterial tonometry and a frequency domain transfer function to noninvasively measure aortic pressure during cardiac MRI examinations, and have combined these central pressure measurements with MRI-derived LV volume to quantify contractility using normalized maximal ventricular power ($nPWR_{max}$), a load independent index of contractility [2]. We have shown sensitivity of $nPWR_{max}$ to contractility at an intermediate dose of dobutamine and insensitivity of $nPWR_{max}$ to increased afterload with methoxamine. This technique of noninvasive contractility assessment may have application in a variety of patient groups, including patients with heart failure to follow response to therapy.

References

1. Karamanoglu et al., *Eur Heart J*., 1993;14:160.
2. Kass et al., *Circulation*, 1991;84:1698.