Integrated Assessment of Diastolic and Systolic Ventricular Function Using Diagnostic Cardiac Magnetic Resonance Catheterization

Validation in Pigs and Application in a Clinical Pilot Study

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OBJECTIVES This study sought to develop and validate a method for the integrated analysis of systolic and diastolic ventricular function.

BACKGROUND An integrated approach to assess ventricular pump function, myocontractility (end-systolic pressure–volume relationship [ESPVR]), and diastolic compliance (end-diastolic pressure–volume relation [EDPVR]) is of high clinical value. Cardiac magnetic resonance (CMR) is well established for measuring global pump function, and catheterization-combined CMR was previously shown to accurately measure ESPVR, but not yet the EDPVR.

METHODS In 8 pigs, the CMR technique was compared with conductance catheter methods (gold standard) for measuring the EDPVR in the left and right ventricle. Measurements were performed at rest and during dobutamine administration. For CMR, the ESPVR was estimated with a single-beat approach by synchronizing invasive ventricular pressures with cine CMR–derived ventricular volumes. The EDPVR was determined during pre-load reduction from additional volume data that were obtained from real-time velocity-encoded CMR pulmonary/aortic blood flow measurements. Pre-load reduction was achieved by transient balloon occlusion of the inferior vena cava. The stiffness coefficient $\beta$ was calculated by an exponential fit from the EDPVR. After validation in the animal experiments, the EDPVR was assessed in a pilot study of 3 patients with a single ventricle using identical CMR and conductance catheter techniques.

RESULTS Bland-Altman tests showed good agreement between conductance catheter–derived and CMR-derived EDPVR. In both ventricles of the pigs, dobutamine enhanced myocontractility ($p < 0.01$), increased stroke volume ($p < 0.01$), and improved diastolic function. The latter was evidenced by shorter early relaxation ($p < 0.05$), a downward shift of the EDPVR, and a decreased stiffness coefficient $\beta$ ($p < 0.05$). In contrast, in the patients, early relaxation was inconspicuous but the EDPVR shifted left-upward and the stiffness constant remained unchanged. The observed changes in diastolic function were not significantly different when measured with conductance catheter and CMR.

CONCLUSIONS This novel CMR method provides differential information about diastolic function in conjunction with parameters of systolic contractility and global pump function. (J Am Coll Cardiol Img 2009;2:1271–81) © 2009 by the American College of Cardiology Foundation
Heart failure is a common cause of mortality and death. In pathophysiology, different forms of systolic and diastolic heart failure can be defined (1–3). In many patients, combined forms of systolic and diastolic dysfunction coexist and are difficult to differentiate. Substantiated knowledge about the predominant form of heart failure is essential for optimizing treatments. Thus, an integrated approach for evaluating systolic contractility in conjunction with diastolic relaxation and compliance would be of high clinical value.

Recent innovations in imaging provided a variety of new methods for the noninvasive assessment of cardiac function. These methods allow determination of stress–strain relation or inflow profiles of the ventricles, for example. However, many of these parameters are pre-load-dependent and/or afterload-dependent or only reflect regional myocardial function. Therefore, analysis of pressure–volume relations is still regarded as the most reliable way to obtain load independent parameters of contractility and diastolic compliance (1,2).

Advances in fast imaging techniques made diagnostic cardiac magnetic resonance (CMR) catheterization a realistic option (4–8). It was shown to provide accurate estimates of the end-systolic pressure–volume relationship (EDPVR) by combining invasive ventricular pressures with cine CMR-derived ventricular volumes. The EDPVR is widely considered the optimal quantification of systolic contractile function (6,8).

In the current study, we propose a novel CMR approach that combines real-time derived ventricular volumes with invasively measured ventricular pressures for assessment of the end-diastolic pressure–volume relationship (EDPVR). The EDPVR characterizes ventricular chamber stiffness in a relatively load-independent fashion and showed practical importance for the assessment of patients with diastolic dysfunction (1,2,9,10).

The aim of this experimental study was to develop a CMR method for assessment of the EDPVR and to validate this method in the pig right and left ventricle using conductance catheter techniques as a gold standard reference. Subsequently, the applicability of this method in a clinical context was evaluated in a pilot study involving patients with a single ventricle after Fontan operation. In these patients, diastolic dysfunction has been reported by several investigators (11,12).

METHODS AND STUDY DESIGN

Animal experiments. The animal experiments were authorized by the responsible animal care authorities. The validation study was conducted in 8 pigs (31 ± 5 kg). The animals were pre-medicated with 5 mg/kg azaperone and 10 mg/kg ketamine intramuscularly. Anesthesia was maintained with 1.5% isoflurane inhalation. All CMR and conductance catheter measurements were performed at end expiratory breath-hold and during muscle relaxation with 0.01 mg/kg vecuronium bromide intravenously. After completion of measurements, animals were euthanized.

The timeline and brief description of the protocol are shown in Figure 1. Right and left ventricular pressure–volume relations were first assessed by conductance catheter (gold standard). Thereafter, the animals were transferred to the neighboring CMR laboratory. All measurements were performed at rest and repeated during continuous infusion of dobutamine at 10 μg/kg/min with at least a 10-min interval between inotropic stimulation and repeated measurements at rest.

Measured parameters. Parameters of ventricular global, myocontractile, and diastolic function were obtained using conductance catheter and/or CMR techniques.

GLOBAL PUMP FUNCTION (BY CINE CMR). This parameter is composed of ventricular end-diastolic, end-systolic, and stroke volume assessed by cine CMR.

MYOCONTRACTILE FUNCTION (BY CONDUCTANCE AND CMR CATHETERIZATION). The slope of the ESPVR (E_{max}) was defined as a measure of contractility and was derived from the pressure–volume loops as determined with conductance catheter and CMR techniques. The E_{max} was indexed to 100 mg myocardial muscle mass (E_{max,i}).

DIASTOLIC FUNCTION (BY CONDUCTANCE AND CMR CATHETERIZATION). From pressure measurements, we derived τ as a parameter of early diastolic relaxation. The stiffness constant (β) was determined from a set of EDPVR and was defined as a measure of diastolic compliance. The β value was
calculated from the EDPVR with an exponential regression: \( EDP = Ae^{-\beta \cdot EDV} \) where \( EDP \) = end-diastolic pressure, \( EDV \) = end-diastolic volume, and \( A \) = curve-fitting constant. The \( \beta \) value was indexed to ventricular volumes for creating a dimensionless index where appropriate.

**Concept of CMR-derived EDPVR.** Because of its inherent nonlinearity, the assessment of the EDPVR ideally requires pressure–volume data acquired at multiple loading conditions. Therefore, ventricular loading was gradually altered by vena cava balloon occlusion. Beat-to-beat alteration in volume load and pressure were measured simultaneously with real-time CMR and liquid-filled catheters. The concept for measuring EDPVR using CMR-catheterization is based on working steps that are shown in Figures 2 to 4.

**STEP A: CINE CMR.** Biventricular phasic absolute volumes were acquired over several cardiac cycles with multislice-multiphase cine CMR. During CMR, ventricular pressures were measured continuously, averaged, and synchronized with the cine CMR derived volumes to construct a baseline pressure–volume loop under steady-state conditions. Synchronization of pressures and volumes was achieved by a trigger signal (Fig. 2, left). The ESPVR was estimated from the baseline loop using a single-beat approach as previously described (7,13).

**STEP B: REAL-TIME CMR.** Instantaneous blood flows were measured using real-time velocity-encoded cine (VEC) CMR in the pulmonary trunk and ascending aorta. Recording of ventricular pressures was started with the beginning of CMR and synchronized with the flow using the trigger signal. At steady state and in the absence of atrioventricular valve insufficiency, effective right or left ventricular stroke volumes are considered being equivalent to the effective antegrade pulmonary or aortic blood flow volumes. Therefore, ventricular chamber volumes can be computed by subtracting the effective stroke volumes from the end-diastolic volume of the cine CMR measurements (marked baseline EDPV in Fig. 3). After 3 to 4 heartbeats, pre-load was lowered by transient balloon occlusion of the vena cava. Importantly, for these unloaded beats...
ventricular filling is unknown, thus the absolute volume (horizontal position of the pressure–volume loop) is undetermined. Initially, end-diastolic volumes of all unloaded loops were arbitrarily arranged to show diminishment in stroke volume.

**STEP C: POST-PROCESSING.** To calibrate the absolute volume of the flow-derived real-time pressure–volume loops, we matched the end-systolic volume of each unloaded beat with the ESPVR volume intercept at the measured corresponding end-systolic pressure. The resulting end-diastolic pressure–volume points were used to determine the EDPVR and to calculate \( \beta \).

**Conductance catheter.** The conductance catheter study was performed using a Leycom signal processor (CD-Leycom, Zoetermeer, the Netherlands) and 6-F to 7-F dual-field catheters (Millar, Houston, Texas). Catheter tips were positioned in the left or right ventricular apex, and measurements were performed during transient pre-load reduction by vena cava balloon occlusion using a 36-mm sizing balloon inflated with isotonic saline solution (AGA Medical, Plymouth, Minnesota). Parallel conductance was determined by the saline dilution method (14). The calibration factor alpha was calculated from CMR-derived stroke volumes.
Data were post-processed using Conduct NT-V2.0.1 (CD-Leycom) and a MATLAB platform (The MathWorks, Natick, Massachusetts).

CMR catheterization. CMR ACQUISITION OF VENTRICULAR PRESSURES AND VISUALIZATION OF CATHETERS. After completion of conductance catheter measurements, catheters were replaced under X-ray angiography by 4-F fluid-filled pigtail diagnostic catheters (Cordis, Warren, New Jersey). The balloon catheter for vena cava occlusions was kept unchanged in the inferior vena cava. The pressure catheters were connected to a pressure transducer (Becton-Dickinson, Franklin Lakes, New Jersey) amplified, recorded, and analyzed with Ponemah software (all DSI, St. Paul, Minnesota). Initial pressure recordings were obtained in the catheter laboratory. Thereafter the animals were moved to the CMR laboratory, where steady state hemodynamic conditions were confirmed. In the CMR laboratory, an additional pressure transducer was placed together with the animal within the bore. Radiofrequency pulses induced pressure signal artifacts on this transducer at the beginning of each VEC CMR measurement. These artifacts were used as a trigger signal for synchronizing measured pressures with acquired volume and flow signals (Figs. 2 and 3). Correct catheter position during CMR was confirmed on interactive real-time CMR as previously described (15).

Ventricular volumes and myocardial mass. All CMR studies were performed on a 1.5-T scanner (release 2.6.1, Philips Intera, Best, the Netherlands). Ventricular chamber volumes and myocardial mass were determined from a stack of multislice-multiphase steady-state free-precession cine CMRs covering the entire heart (16). Sequence parameters were: repetition time/echo time 3.4/1.7 ms, slice thickness 6 mm, no gap, in-plane resolution 1.9 × 1.3 mm, 45 phases per cardiac cycle, number of averages 1, sensitivity encoding reduction factor 2. Analysis was performed using View Forum software (Release 6.1, Philips). Biventricular endocardial and epicardial borders were manually traced for computing ventricular volumes and myocardial mass where the septum was accounted left ventricular. Papillary muscles and prominent right ventricular trabeculation were excluded for volume measurements. Stroke volume was calculated as the difference between the diastolic and systolic volumes. Ejection fraction was calculated as the ratio of stroke volume to end-diastolic volumes.

Pulmonary and aortic blood flow. Quantitative blood flow was measured using real-time VEC CMR (17) orthogonal to the dominating flow direction in the pulmonary trunk and the ascending aorta. For the measurements, we corrected for potential phase errors arising from the concomitant magnetic field. Sequence parameters were: repetition time/echo time 23/6.5 ms, matrix 128 × 256, field of view 400 mm, slice thickness 8 mm, encoding velocity 150 cm/s, sensitivity encoding reduction factor 3, half-scan factor 0.6, echo planar imaging factor 41. This resulted in the dynamic scan time of 31 ms for the acquisi-
Integrated Myocardial Function Analysis by CMR

Table 1. Animal Experiments, Parameters of Cardiac Function by Cine CMR and VEC CMR (Where Indicated)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>At Rest</th>
<th>During Dobutamine Administration</th>
</tr>
</thead>
<tbody>
<tr>
<td>General characteristics</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>30.3 ± 6.8</td>
<td>NA</td>
</tr>
<tr>
<td>RV muscle mass (g)</td>
<td>27.8 ± 6.3</td>
<td>NA</td>
</tr>
<tr>
<td>LV muscle mass (g)</td>
<td>83.9 ± 7.2</td>
<td>NA</td>
</tr>
<tr>
<td>Global ventricular pump function</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RV end-diastolic volume (ml)</td>
<td>45.6 ± 9.3</td>
<td>43.6 ± 8.4</td>
</tr>
<tr>
<td>RV end-systolic volume (ml)</td>
<td>15.1 ± 4.1</td>
<td>8.6 ± 3.2*</td>
</tr>
<tr>
<td>RV stroke volume (ml)</td>
<td>30.6 ± 4.5</td>
<td>35.3 ± 6.3*</td>
</tr>
<tr>
<td>RV stroke volume by VEC CMR (ml)</td>
<td>29.6 ± 3.1</td>
<td>34.9 ± 6.5*</td>
</tr>
<tr>
<td>LV end-diastolic volume (ml)</td>
<td>46.5 ± 9.6</td>
<td>45.8 ± 7.4</td>
</tr>
<tr>
<td>LV end-systolic volume (ml)</td>
<td>16.7 ± 6.3</td>
<td>10.1 ± 4.9*</td>
</tr>
<tr>
<td>LV stroke volume (ml)</td>
<td>29.7 ± 4.1</td>
<td>35.7 ± 5.7*</td>
</tr>
<tr>
<td>LV stroke volume by VEC CMR (ml)</td>
<td>29.1 ± 4.4</td>
<td>36.4 ± 5.3*</td>
</tr>
<tr>
<td>Cardiac output (l/min)</td>
<td>4.0 ± 1.2</td>
<td>6.4 ± 1.4*</td>
</tr>
</tbody>
</table>

*Significant differences (p < 0.05) between measurements at rest versus during dobutamine administration. LV = left ventricle; NA = not assessed; RV = right ventricle; VEC CMR = velocity-encoded cine cardiac magnetic resonance.

RESULTS

Validation study. COMPARISON OF CINE CMR AND VEC CMR STROKE VOLUMES. The data are shown in Table 1. At rest and during dobutamine administration, there was no significant difference between these methods.

CMR versus conductance catheter–derived diastolic compliance. Sequential conductance catheter and CMR measurements were realized at similar hemodynamic conditions, evidenced by the fact that right and left ventricular pressures as well as heart rates were not significantly different between the 2 experimental stages (Table 2). The hemodynamic responses to dobutamine also were similar (Table 2).

The Bland-Altman test showed good agreement between β values determined with the 2 methods at rest and during dobutamine administration (Fig. 5). Importantly, conductance catheter–derived and CMR-derived pressure–volume loops showed parallel changes for measurements of β at rest and during stress (Table 2, Fig. 6). The relative changes of Emax,i were also at similar levels (Table 2).

Response to dobutamine. GLOBAL PUMP FUNCTION (BASED ON CINE CMR). As expected, in response to dobutamine, the right and left ventricle showed a significant increase in stroke volume and cardiac output (p < 0.01) (Table 1).

MYOCONTRACTILITY (BASED ON CONDUCTANCE AND CMR CATHETERIZATION). Inotropic stimulation with dobutamine increased Emax,i significantly in both ventricles (p < 0.01). The response to dobutamine was more pronounced in the left compared with the right ventricle (Table 2). The noted changes of Emax,i were similar for the conductance catheter and CMR measurements (Table 2).

DIASTOLIC FUNCTION (BASED ON CONDUCTANCE AND CMR CATHETERIZATION). Active early relaxation, as indicated by smaller τ, improved significantly during dobutamine administration in both ventricles (p < 0.01) (Table 1). In addition, in all animals and both ventricles, the EDPRV shifted toward the bottom right of the pressure–volume diagram (Fig. 6). There was also a slight but significant decrease of β (p < 0.05) (Table 2, Fig. 5). The noted changes were again similar for the conductance catheter and CMR measurements.
Clinical experiments. Oxygen saturation was above 95%, also unaltered during dobutamine administration. All Fontan circuits were free from obstructions. Concordantly during dobutamine administration there was a decrease of end-diastolic and -systolic volumes (Table 3). Contractility, stroke
volumes, and heart rate increased slightly, resulting in elevated cardiac outputs (Table 3). Active early relaxation was slightly enhanced. In contrast, the EDPVR shifted toward the left in the pressure–volume diagram and β remained nearly unchanged (Table 3, Figs. 6 and 7). Similar observations were made with conductance catheter and CMR techniques (Fig. 6).

**DISCUSSION**

We developed and validated a novel method for estimating the EDPVR, an index of ventricular chamber stiffness, by combining invasive pressure measurements with noninvasive real-time CMR volume and flow data. Our results indicate that the proposed method is suited to determine left and right ventricular chamber stiffness in conjunction with parameters of contractility and global pump function.

**Technical consideration.** The ESPVR and EDPVR are widely used in physiological studies because they reflect intrinsic systolic and diastolic myocardial function in a relatively load-independent fashion. Clinical application is still limited, mainly because of technical difficulties in performing the required measurements. The proposed CMR method might potentially expand clinical application because it is technically straightforward, and once the small and user-friendly catheters are in place, a complete set of right and left ventricular pressure–volume acquisitions can be obtained in <10 min.

Ideally, ESPVR and the EDPVR are determined from a family of pressure–volume loops during transient pre-load reduction (9,10). Previously, methods were introduced to estimate the ESPVR by single-
beats approaches (7,8,13,19). However, similar simplified approaches to estimate the EDPVR are complicated by its inherent nonlinearity. Therefore, load interventions, which are commonly performed by balloon occlusion of the vena cava, are needed for determining profiles of ventricular chamber stiffness.

The conductance catheterization is an established method for measuring ventricular pressure–volume relations in animal experiments and human studies. However, by theory it requires a symmetrically shaped ventricle, and derived volumes need calibration with volumes obtained by a valid reference method, such as CMR (20). Consequently, conductance measurements in the nonsymmetrically shaped right ventricle or in a variety of congenitally malformed hearts can be problematic. Particularly these patients often need a differential analysis of ventricular systolic and diastolic function to determine optimal treatments.

To date, CMR is considered the gold standard in terms of accuracy and reproducibility for quantification of ventricular volumes (4,5). As mentioned, this measurement accuracy is crucial for the assessment of pressure–volume relations, particularly in asymmetrically shaped ventricles. Measurement errors by calibrating issues or by geometric assumptions are avoided (14,21). Similar to the conductance catheter, the CMR method proposed in this study requires invasive measurement of pressures. The fluid-filled CMR catheters used were, however, substantially smaller in size (4-F vs. 7-F) and easily positionable. To avoid susceptibility artifacts or radiofrequency pulse-induced heating during CMR, the application of metallic high-fidelity pressure-tipped catheters was excluded, and consequently materials without metallic components were used (15). A potential down side of fluid-filled catheters is the attenuation of pressure amplitudes. To minimize this effect, we limited the total length of the catheter and the connecting pressure line to H1 m.

In the current study, we determined the ESPVR using a single-beat approach as previously reported (7,8,13,19). This approach implies that ventricular volumes are measured by cine CMR over several beats and are synchronized with pressures averaged for the same time period. To minimize beat-to-beat variability, keep the hemodynamic condition and state of ventilation at identical levels during data acquisition.

Physiologic aspects. In the animal study, during dobutamine administration, diastolic function improved by faster early relaxation in conjunction with a slight bottom-right shift of the EDPVR. Similar to observations in previous studies (20,22), we also

Figure 7. Ventricular Stiffness in the Healthy Animal Heart and in Fontan Patients
Representative CMR-derived pressure–volume loops of a pig right and left ventricle and a patient with a single ventricle. The end-systolic pressure–volume relation (ESPVR) and end-diastolic pressure–volume relation (EDPVR) at rest and during dobutamine administration are shown (left, dashed lines). The EDPVRs are shown more detailed (right). Note the left shift of the relation in the patient as opposed to the right-bottom shift in the pig’s right and left ventricle during dobutamine administration. Abbreviations as in Figure 1.
noted a decrease of $\beta$. In contrast, in the patients, the EDPVR shifted during dobutamine administration toward the upper left in the pressure–volume diagram. Early relaxation improved slightly, and $\beta$ did not change substantially.

Several reasons can account for the noted abnormal diastolic function in Fontan patients. The single ventricle, no matter of left/right type, is mostly of abnormal geometric shape with a direct impact on the mechanical properties of the ventricle and thus on systolic and diastolic function (12). Moreover, a recent pathohistological study showed abnormal myoarchitecture of the connective tissue matrix (23). During infancy these ventricles are exposed to prolonged cyanosis and volume load, which may induce fibrosis and thus have an impact on diastolic stiffness (24).

Accordance exists that the EDPVR reflects chamber capacity and compliance. Similar to the ESPVR, this relation is influenced by ventricular configuration, including size, and heart rate. Therefore, the impact of volume and heart rate changes during dobutamine administration must be considered when interpreting these data. The tendency toward a smaller $\beta$ in the animal study and an invariable $\beta$ in the patients was, however, also noted when $\beta$ was indexed to ventricular chamber size (Tables 2 and 3).

In our study, parameters of contractility and chamber stiffness were measured with conductance catheter and CMR techniques nonsimultaneously. As mentioned earlier, alteration in volume load and heart rate, which can be induced among other factors by prolonged sedation, may have an impact on the ESPVR and EDPVR. We were able to keep baseline hemodynamic parameters, including heart rate, stable with changes of <10% between the sequential measurements (Table 2). We recommend measuring the EDPVR with the proposed CMR catheterization technique at heart rates of no more than 150 beats/min. This is for purely physiological reasons and for allowing reasonable temporal resolution in the real-time CMR flow measurements. The development of faster real-time CMR applications should be subject to future research.

Normalization of contractile and stiffness indexes may be required when comparing different study populations. Concerning the diastolic $\beta$, Burkhoff et al. (9) suggested the use of a dimensionless index by multiplying $\beta$ by the myocardial wall volume, which can be directly obtained from the acquired cine CMR scans. The variability of wall volume measurements, particularly for the right ventricle, is known to be substantial (25). As an alternative, $\beta$ might be normalized to chamber volume. This important issue must be systematically investigated in future research.

**Study limitations.** The CMR and conductance catheter measurements cannot be performed simultaneously. For comparison of measured data, we aimed for keeping the animals’ physiological conditions, such as heart rate and blood pressure constant. Care must be taken when comparing physical exercise with dobutamine stress (26). Therefore, it would be inappropriate to directly translate our findings regarding chamber stiffness during dobutamine administration to exercise conditions. Some technical limitations should be mentioned. In the CMR setting, we measured ventricular pressures with fluid-filled catheters to avoid metallic components, in contrast with the high-fidelity, solid-state sensors incorporated in the conductance catheter. A fluid-filled catheter manometer system acts as a low-pass filter, thus high-frequency components are attenuated. However, by using relatively stiff and short catheters and carefully removing trapped air bubbles, adequate recordings of cardiac and arterial pressure signals can be obtained and the impact on derived indexes is expected to be very limited (27). For indexes that require high-frequency components, such as relaxation time constants, CMR-compatible pressure catheters or sophisticated signal processing may need to be considered (28,29). VEC CMR measurements are susceptible to several potential sources of error, which include turbulent flow and moving valve planes. The CMR method was introduced to the clinical scenario in a small, well-controlled pilot study. Future studies must include a much larger number of patients.

**CONCLUSIONS**

This work presents an CMR catheterization method for the assessment of diastolic and systolic pressure–volume relation in the left and right ventricle. The applied method combines cine CMR ventricular volumes, real-time VEC CMR blood flow, and invasive ventricular pressure measurements. Our results indicate that the proposed CMR method provides, in addition to parameters of systolic contractility and global pump function, accurate load-independent indexes of biventricular diastolic function. The proposed CMR method might potentially expand the application of pressure–volume relation in the clinical con-
text. Because ventricular volumes are measured by CMR without geometric assumptions, the technique might also be suitable for assessment of right ventricular disorders or congenitally malformed hearts.

**REFERENCES**


**Key Words:** cardiac magnetic resonance • diagnostic catheterization • pressure–volume loops • diastolic function.