Noninvasive quantification of left ventricular elastance and ventricular-arterial coupling using three-dimensional echocardiography and arterial tonometry

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Gayat E, Mor-Avi V, Weinert L, Yodwut C, Lang RM. Noninvasive quantification of left ventricular elastance and ventricular-arterial coupling using three-dimensional echocardiography and arterial tonometry. Am J Physiol Heart Circ Physiol 301: H1916–H1923, 2011. First published September 9, 2011; doi:10.1152/ajpheart.00760.2011.—Most techniques previously used to assess left ventricular (LV) end-systolic elastance (Ees) and ventricular-arterial coupling (C LV-A) relied on invasive measurements and data acquisition over a wide range of loading conditions. Our goals were to 1) assess the feasibility of noninvasive assessment of Ees and CLV-A using real-time three-dimensional echocardiography (RT3DE) and arterial tonometry; 2) test the ability of this approach to detect changes in LV contractility; and 3) study its reproducibility. We studied pharmacologically induced changes in inotropic state (5 and 10 μg·kg−1·min−1 dobutamine) in normal volunteers (N = 8) and compared 10 normal volunteers with 10 patients with dilated cardiomyopathy (DCM; ejection fraction <35%). RT3DE LV images, calibrated carotid artery tonometry, and Doppler tracings were obtained to noninvasively estimate Ees and CLV-A, using two alternative calculations. Dobutamine caused a significant stepwise increase in blood pressure, heart rate, ejection fraction, and Ees, and a decreased CLV-A. In patients with DCM, Ees was significantly reduced and CLV-A elevated, compared with controls. Both inter- and intraobserver variability were good for all measured parameters, as reflected by intraclass correlation coefficients (>0.8) and coefficients of variation (<20%). While both Ees estimates showed significant differences between DCM patients and controls, one estimate resulted in no overlap and better reproducibility (interobserver intraclass correlation coefficient: 0.83 vs. 0.47, coefficients of variation: 20 vs. 29%). This is the first study to test the feasibility of using RT3DE-derived LV volumes in conjunction with arterial tonometry to noninvasively quantify LV elastance and CLV-A. This approach was found to be sensitive enough to detect expected differences in LV contractility and reproducible. Due to its noninvasive nature, this methodology may have clinical implications in various disease states.

left ventricular contractility

LEFT VENTRICULAR (LV) pressure-volume loops provide valuable information on LV systolic and diastolic function, as well as on LV-arterial coupling (CLV-A) (8). End-systolic elastance (Ees), the slope of the end-systolic pressure (ESP)-volume relationship, has been used as a load-independent index of myocardial contractility (2). In the past, to obtain pressure-volume loops, invasive pressure and volume measurements were required to be obtained under a wide range of loading conditions, thus limiting the clinical applicability of this technique. To avoid these limitations, several investigators have proposed invasive solutions to estimate Ees from a single cardiac cycle (23, 24, 27), and, more recently, the accuracy of noninvasive estimates of Ees has been demonstrated (4). However, the feasibility of this noninvasive methodology was tested in only few clinical studies using two-dimensional (2D) echocardiography, combined with noninvasive estimates of end-systolic LV pressure. In addition, the reproducibility of this methodology is not well established (1, 6, 22). Moreover, there is a second theoretical approach, based on bilinear, rather than linear, approximation of the time-varying elastance curve, to calculate Ees (24). This latter approach has not been tested in the clinical setting.

It was established that real-time three-dimensional (3D) echocardiography (RT3DE) allows more accurate quantification of LV volumes than 2D echocardiography (7, 9, 14, 25). In addition, it has been shown that end-systolic LV pressure can be accurately estimated noninvasively by tonometry from the carotid artery (5). Accordingly, we hypothesized that RT3DE, in combination with arterial tonometry, could be used for accurate noninvasive measurements of the LV ESP (LVESP)-volume relationship, arterial elastance, and CLV-A. The goal of the present study was to assess this approach, using the two alternative calculations of Ees, by testing its ability to detect changes in systolic elastance and CLV-A, and by studying its reproducibility.

METHODS

Study design. The study included two protocols. The first protocol involved pharmacologically induced changes in inotropic state in normal healthy volunteers who were studied at rest and during infusion of dobutamine (5 and 10 μg·kg−1·min−1). The goal of the second protocol was to determine whether this technique is sensitive enough to detect abnormal LV elastance. To achieve this goal, we compared normal volunteers and patients with LV systolic dysfunction. In both protocols, RT3DE, Doppler echocardiography, and tonometry of the carotid artery were performed, and peripheral blood pressure was measured in each patient to obtain parameters needed to calculate Ees and CLV-A.

Participants. We prospectively studied a total of 25 subjects, including 15 normal volunteers and 10 consecutive patients with dilated cardiomyopathy (DCM) and severe LV systolic dysfunction [LV ejection fraction (EF) <35%], after 2 patients were excluded because of inadequate image quality. Patients with aortic stenosis or cerebrovascular disease were excluded. The protocol was approved by the Institutional Review Board, and each subject provided informed consent.

Data acquisition. Echocardiographic imaging was performed by a single experienced sonographer using an iE33 ultrasound system with the X5 transducer (Philips Healthcare, Andover, MA). Pulmonary venous flow, used to measure diastolic deceleration time (dpPVFD), was acquired from the apical four-chamber view by positioning the pulsed-wave Doppler sample volume in the right upper pulmonary vein. The apical five-chamber view was then acquired to depict both
the mitral and aortic flows in the same pulsed-wave Doppler tracing. Isovolumic contraction time (ICT) was determined by positioning the sample volume at a depth at which distinct mitral valve closure and aortic valve opening were best visualized (Fig. 1). Additionally, aortic continuous Doppler was recorded to measure LV ejection time (ET), defined as the time interval between the opening and the closure of the aortic valve.

RT3DE imaging was performed using a wide-angled “full-volume” acquisition mode, in which four wedge-shaped subvolumes were obtained over consecutive cardiac cycles during a single breath hold. Special care was taken to include the entire LV cavity within the pyramidal scan volume. After gain settings were optimized for endocardial visualization, three to four data sets were acquired and stored digitally for offline analysis.

Carotid artery applanation tonometry was recorded simultaneously with the echocardiographic imaging using SphygmoCor device (AtCor Medical, Sydney, Australia). The tonometer was calibrated using oscillometric arm-cuff blood pressure recordings (Dinamap Pro 1000V3, GE Medical Systems, Milwaukee, WI).

Data acquisition, including echocardiographic imaging and arterial tonometry, was performed in <10 min in each of the study participants.

Data analysis. RT3DE images were analyzed offline using commercial software (3DQ-Advanced QLab, Philips Healthcare, Andover, MA). Initially, two- and four-chamber views with the largest long-axis dimensions were selected from the pyramidal data set in the first time frame of the data set, i.e., end diastole, as described previously (18). In these two planes, five points, including four points on the mitral annulus (two in each plane), and the apex in either plane were manually initialized to define the endocardial surface. Then the endocardial surface was manually adjusted in multiple apical planes, while including the papillary muscles in the LV cavity, and its position was corrected as necessary in multiple arbitrary cut-planes until the best match was visually verified. The software was used to automatically track the endocardial boundary in 3D space throughout the cardiac cycle and generate a time curve of LV volume (Fig. 2, top), from which end-systolic (ESV) and end-diastolic LV volumes (EDV) were obtained. Thereafter, stroke volume (SV) was calculated as EDV – ESV and EF as SV/EDV. Analysis of RT3DE data sets to obtain LV volumes required <2 min in most patients.

Calibrated carotid artery applanation tonometry time curves were used to estimate central aortic waveform, from which LVESP, defined as the pressure measured at the end of LV ejection, was obtained. The ejection duration was defined as the period of time from the start of the pulse corresponding to the aortic valve opening to the incisura indicating the closure of the aortic valve (Fig. 2, bottom).

Doppler dpPVFd was used to determine LV end-diastolic pressure (EDP) using the previously described equation: EDP = 36.7651 – 0.10299 × dpPVFd (21).

*Estimation of the LV elastance. The LV elastance (Ees) was estimated using two different algorithms, referred to below as technique 1 and technique 2. Technique 1 involved an algorithm adapted from the previously described noninvasive single-beat technique (4). This algorithm assumes a linear relationship and a constant volume intercept (Vo), which was validated against invasive measurements. Briefly, the single-beat elastance was calculated as:

\[
E_{es} = \left\{ \frac{P_d}{\text{ESP}} \times \text{SV} \times E_{N\text{d}(\text{est})} \right\}
\]

where \( P_d \) is the diastolic blood pressure; ESP is the LVESP; and \( E_{N\text{d}(\text{est})} \) is the noninvasive estimated normalized left ventricular elastance at the onset of ejection. \( E_{N\text{d}(\text{est})} = 0.0275 - 0.165 \times \text{ejection fraction} + 0.3656 \times (P_d/\text{ESP}) + 0.515 \times (E_{N\text{d}(\text{avg})}) \), with \( E_{N\text{d}(\text{avg})} \) (group-averaged normalized left ventricular elastance at the onset of ejection) calculated using a previously described seven-term polynomial function,
dtPVFd, Ees, and CLV-A, all measurements in the 20 subjects studied. invasive measurements used in this study, including ICT, ET, EDV, ESV, the LV becomes less efficient (15, 16).

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DEFINED AS CLV-A ZERo VOLUME IS ASSOCIATED WITH ZERO PRESSURE, EA CAN BE CALCULATED

Systolic and diastolic time intervals (26) (Fig. 3). Assuming that Ea exceeds Ees (i.e., CLV-A is zero), then stroke work falls, and the LV becomes less efficient (15, 16).

Reproducibility analysis. To assess the reproducibility of noninvasive measurements used in this study, including ICT, ET, EDV, ESV, dtpVFD, Ees, and CLV-A, all measurements in the 20 subjects studied in protocol 2 were repeated by the same observer at least 1 mo later and by a second independent observer, both blinded to all prior measurements.

Fig. 2. Aortic pressure time-curve obtained from tonometry of the carotid artery (top) and left ventricular (LV) volume time-curve obtained by real-time three-dimensional echocardiography (bottom).

\[ E_{Na(gav)} = \sum_{i=0}^{\infty} a_i \times t_{Na} \]

where \( a_i \) are empirically determined coefficients (4). The value of \( t_{Na} \) (normalized time) was determined by the ratio of pre-ejection period to total systolic period, thus \( t_{Na} = ICT/(ICT + ET) \). Different from the method of Chen et al. (4), ESP was not approximated by the product of brachial systolic pressure, \( P_s \) x 0.9, but directly obtained from the central aortic waveforms estimated by calibrated arterial tonometry (Fig. 2).

Technique 2, originally proposed by Shishido et al. to estimate elastance invasively from a single beat (24), was applied to our noninvasive data to calculate \( E_{es} \) as:

\[ E_{es} = \frac{P_d + (P_d - EDP)/ICT \times ET \times \alpha - ESP}{SV} \]

where \( \alpha = -0.210 + 1.348 \times EF + 0.682 \times ICT/(ICT + ET) \).

Estimation of the \( C_{LV-A} \). The arterial system can be described by the relation between SV and ESP. The slope of this relationship represents the effective arterial \( E_{es} \) (\( E_{es} \)), which incorporates the principal elements of vascular load, including peripheral resistance, total vascular compliance, characteristic impedance, and systolic and diastolic time intervals (26) (Fig. 3). Assuming that zero volume is associated with zero pressure, \( E_{es} \) can be calculated as the ratio of ESP to SV (\( E_{es} = ESP/SV \)) (26). The \( C_{LV-A} \) is thus defined as \( C_{LV-A} = E_{es}/E_{es} \). Of note, the LV and arterial system are optimally coupled to produce stroke work when \( C_{LV-A} = 1 \), and when \( E_{es} \) exceeds \( E_{es} \) (i.e., \( C_{LV-A} > 1 \)), then stroke work falls, and the LV becomes less efficient (15, 16).

Statistical analysis. Results are expressed as median (with interquartile range) for continuous variables and as counts (percentage) for categorical variables. Continuous variables were compared using nonparametric Wilcoxon and Kruskal-Wallis tests. \( P \) values < 0.05 were considered significant. Inter- and intraobserver variability of each measured parameter was expressed in terms of intraclass correlation coefficients with its 95% confidence interval and absolute difference in percentage of the mean of the corresponding pairs of repeated measurements, i.e., coefficient of variation (CV). All analyses were performed using the R statistical software (The R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

Study subjects. In protocol 1, all eight healthy volunteers were men, age of 36 yr (32–48 yr), body mass index 23.4 kg/m² (22.6–27.1 kg/m²), and body surface area 1.9 m² (1.9–2.0 m²). The basic demographics and hemodynamic data of the 10 normal control subjects and 10 patients with DCM in protocol 2 are shown in Table 1. Three of the normal volunteers were used in both protocols.

Effects of dobutamine on LV elastance and \( C_{LV-A} \). As expected, dobutamine caused a significant increase in blood pressures, heart rate, and EF (Table 2). We found that our approach was able to detect a significant increase in LV elastance (Fig. 4, top) and a significant decrease in \( C_{LV-A} \) (Fig. 4, bottom), when using both techniques 1 (Fig. 4, left) and 2 (Fig. 4, right) to calculate \( E_{es} \), even with the lower dose of dobutamine.

Effects of DCM on LV elastance and \( C_{LV-A} \). The 10 patients with DCM enrolled in protocol 2 had severely dilated ventricles with reduced LV function, as reflected by EDV of 244 ml (211–254 ml) and EF of 24% (18–27%). In addition, LVESP and systolic blood pressure (SBP) were significantly reduced in patients with DCM compared with the normal controls (Table 1). Along with these expected differences between these patients and the control subjects, a significant decrease in LV elastance and an increase in \( C_{LV-A} \) were also
noted, when calculating Ees using both techniques 1 (Fig. 5, left) and 2 (Fig. 5, right). Of note, unlike technique 1 that showed a considerable overlap between the groups in both Ees and CLV-A, technique 2 resulted in no overlap.

**Reproducibility.** The results of the reproducibility analysis are summarized in Table 3. Both inter- and intraobserver variability were good, as reflected by low percent variability values and high intraclass correlation coefficients, with the exception of ICT and Ees, when calculated using technique 1. Of note, the reproducibility of Ees measurements was better when using technique 2 (Table 3).

Table 2. Dobutamine-induced hemodynamic changes in the normal volunteers in protocol 2

<table>
<thead>
<tr>
<th>Dobutamine, µg·kg⁻¹·min⁻¹</th>
<th>Baseline</th>
<th>5</th>
<th>10</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR, beats/min</td>
<td>69 (64–71)</td>
<td>75 (60–78)</td>
<td>89 (75–96)</td>
<td>0.029</td>
</tr>
<tr>
<td>SBP, mmHg</td>
<td>130 (117–134)</td>
<td>140 (134–149)</td>
<td>157 (150–164)</td>
<td>0.0026</td>
</tr>
<tr>
<td>DBP, mmHg</td>
<td>77 (73–80)</td>
<td>70 (67–81)</td>
<td>73 (71–75)</td>
<td>0.6</td>
</tr>
<tr>
<td>ESP, mmHg</td>
<td>115 (102–118)</td>
<td>117 (112–119)</td>
<td>113.5 (109–123)</td>
<td>0.63</td>
</tr>
<tr>
<td>EDP, mmHg</td>
<td>21 (18–22)</td>
<td>19 (16–20)</td>
<td>21 (18–26)</td>
<td>0.36</td>
</tr>
<tr>
<td>EDV, ml</td>
<td>143 (129–154)</td>
<td>134 (113–159)</td>
<td>122 (108–141)</td>
<td>0.46</td>
</tr>
<tr>
<td>ESP, mmHg</td>
<td>54 (50–61)</td>
<td>42 (34–46)</td>
<td>26 (21–33)</td>
<td>0.00034</td>
</tr>
<tr>
<td>EF, %</td>
<td>61 (61–63)</td>
<td>71 (68–74)</td>
<td>79 (78–81)</td>
<td>0.0009</td>
</tr>
<tr>
<td>Ees, mmHg/ml</td>
<td>1.2 (1.1–1.4)</td>
<td>1.2 (1.1–1.5)</td>
<td>1.2 (1.1–1.3)</td>
<td>0.75</td>
</tr>
</tbody>
</table>

Technique 1

| Ees, mmHg/ml                | 1.8 (1.7–1.9) | 2.4 (2.1–2.9) | 3.4 (2.9–4.4) | 0.0005 |
| CLV-A, units                | 0.7 (0.6–0.7) | 0.5 (0.5–0.6) | 0.3 (0.3–0.3) | 0.0005 |

Technique 2

| Ees, mmHg/ml                | 2.18 (2.05–2.56) | 2.76 (2.29–3.36) | 3.24 (2.79–4.20) | 0.0005 |
| CLV-A, units                | 0.60 (0.55–0.61) | 0.45 (0.40–0.48) | 0.35 (0.33–0.37) | 0.0005 |

Values are medians with interquartile ranges in parentheses. P values derived using the Kruskal-Wallis test.

**DISCUSSION**

Most techniques previously used to assess LV contractility and CLV-A are affected by loading conditions and relied on invasive measurements (2). To date, the clinical need for load-independent noninvasive solutions to estimate LV contractility has remained largely unanswered. This is the first study to test the feasibility of using RT3DE-derived LV volumes in conjunction with arterial tonometry-derived LV pressures to noninvasively quantify LV elastance and CLV-A. The theoretical advantages of this novel approach are as follows: 1) RT3DE-derived LV volumes are more accurate and more reproducible than those obtained using 2D echocardiography, because the former technique is not affected by foreshortened apical views and does not rely on geometric assumptions (17); and 2) tonometry-based estimates of end-systolic LV pressure are more direct and thus potentially more accurate than the extrapolation from arm cuff pressure. Our study was designed to evaluate the ability of this combination of techniques to detect changes in LV contractility, induced both by pharmacological means (use of dobutamine in normal volunteers) and by disease (comparison of patients with DCM to normal subjects).

One additional novel aspect of our study was the use of an alternative technique to calculate LV elastance, which was initially developed as an invasive single-beat solution, but has never been used with noninvasive hemodynamic measurements (24). This alternative technique requires the knowledge of LV EDP, which can be noninvasively obtained using several previously validated methods (3, 19, 21). In this study, we used the technique described by Olariu et al. based on dtPVF’d, which was validated against invasive measurements in a large group of patients (21). Accordingly, our choice was motivated by the high accuracy and relative simplicity of this methodology, potentially leading to high reproducibility.

Today, most investigators use arm cuff blood pressure to estimate LVEF by multiplying noninvasive SBP by 0.9. This approximation was originally described in a study involving 10 subjects using invasive measurements of SBP (12). Nevertheless, several studies used this approximation with noninvasive arm cuff blood pressure (1, 4, 6, 20, 22). Since the use of the...
same approximation across a wide range of patients and loading conditions might not be optimal, in the present study, we used instead tonometry of the carotid artery. Arterial tonometry is a valuable tool not only for waveform analysis of the carotid artery, but also as a surrogate for central aortic waveform analysis, as it allows measurement of actual carotid blood pressures, which closely resemble the pressure measured in the ascending aorta (10, 11).

We found that this combination of noninvasive techniques provides a tool for estimation of LV elastance, and CLV-A, which is sufficiently sensitive to detect an increase in LV ejection fraction induced by doses of dobutamine as low as 5 μg·kg⁻¹·min⁻¹, as well as a decrease in contractility seen in patients with DCM. While both techniques used to estimate LV elastance similarly showed significant differences in the two protocols, the results of these protocols did not allow us to definitively determine which of the two techniques is superior in its discriminative power.

Also importantly, our repeated analyses demonstrated good reproducibility of the noninvasive measurements used to estimate LV elastance and CLV-A (Table 3). Thus, noninvasively, the reproducibility of our approach to estimate Ees was also good, especially when Ees was estimated using the second technique. The difference in the reproducibility of the two techniques can be explained by the relative weight of ICT in the two calculations of Ees, since ICT had the highest variability among all measured parameters. This parameter is used in the first order of approximation in technique 1, while technique 2 uses only its first order of approximation.

Limitations. A limitation of the approach tested in this study is that it does not truly represent a single-beat technique, because 3D echocardiographic data were obtained over four consecutive cardiac cycles, and also because the other measurements were not obtained during the same beat. This could have potentially resulted in temporal discordance and noise. Another limitation is associated with the use of carotid tonometry to estimate LVESP, which would be inaccurate in patients with aortic stenosis or occlusive carotid vascular disease. Furthermore, it is difficult to rule out the effects of elevated heart rates on the echocardiographic measurements used to estimate Ees in our study. This is because heart rate on the echocardiographic measurements used in both protocols in our study is a validated tool not only for waveform analysis of the carotid artery, but also a surrogate for central aortic waveform analysis, as it allows measurement of actual carotid blood pressures, which closely resemble the pressure measured in the ascending aorta (10, 11).
flow profiles; and 2) RT3DE-based measurement of LV ESV may be affected by the relatively low temporal resolution.

One limitation of our study is the small size of the study groups in both protocols. However, these samples were sufficiently large to reach a high level of significance, when testing differences in LV elastance and CLV-A, between baseline and low dose of dobutamine, as well as between normal controls and patients with DCM. Increasing the number of patients would be highly unlikely to affect the results.

Another limitation of this study is that it does not include validation against an independent reference technique. However, there is no accepted noninvasive “gold standard” to estimate LV elastance, which is why this study was designed to test the ability of the proposed approach to detect pharmaco-

Table 3. Intra- and interobserver reproducibility of measured and calculated parameters

<table>
<thead>
<tr>
<th></th>
<th>Intraobserver Reproducibility</th>
<th>Interobserver Reproducibility</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CV, %</td>
<td>ICC (95% CI)</td>
</tr>
<tr>
<td>ICT, ms</td>
<td>8 ± 14</td>
<td>0.66 (−0.04; 0.87)</td>
</tr>
<tr>
<td>ET, ms</td>
<td>5 ± 9</td>
<td>0.87 (0.72; 0.94)</td>
</tr>
<tr>
<td>dtPVFD, ms</td>
<td>14 ± 11</td>
<td>0.79 (0.56; 0.91)</td>
</tr>
<tr>
<td>EDV, ml</td>
<td>12 ± 10</td>
<td>0.96 (0.81; 0.98)</td>
</tr>
<tr>
<td>ESV, ml</td>
<td>13 ± 9</td>
<td>0.98 (0.91; 0.99)</td>
</tr>
<tr>
<td>$E_a$, mmHg/ml</td>
<td>16 ± 17</td>
<td>0.46 (0.28; 0.66)</td>
</tr>
<tr>
<td>Technique 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$E_{es}$, mmHg/ml</td>
<td>22 ± 17</td>
<td>0.51 (0.28; 0.80)</td>
</tr>
<tr>
<td>CLV-A</td>
<td>13 ± 10</td>
<td>0.88 (0.73; 0.94)</td>
</tr>
<tr>
<td>Technique 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$E_{es}$, mmHg/ml</td>
<td>19 ± 17</td>
<td>0.75 (0.59; 0.95)</td>
</tr>
<tr>
<td>CLV-A</td>
<td>21 ± 14</td>
<td>0.89 (0.76; 0.96)</td>
</tr>
</tbody>
</table>

CV, coefficient of variation; ICC, intraclass correlation; CI, confidence interval; ICT, isovolumic contraction time; ET, ejection time; dtPVFD, pulmonary venous flow used to measure diastolic deceleration time.
logically induced changes in contractility and known differences between normal subjects and patients with cardiomyopathy, rather than to directly assess its accuracy.

One might also see as a limitation the fact that we used patients with severe LV systolic dysfunction to test our methodology. While this initial study was designed to test this noninvasive approach under relatively favorable conditions, its ability to detect less extreme changes in contractility needs to be addressed.

Clinical implications. Pressure-volume loops generated using invasive techniques have provided important insights into LV mechanics (2). While RT3DE allows continuous measurement of LV volume throughout the cardiac cycle, carotid arterial tonometry provides LV pressure-time curves throughout systole. Thus the combination of these two curves allows noninvasive construction of the systolic portion of the pressure-volume loop (Fig. 3, from point C to point D). To complete the noninvasive reconstruction of the pressure-volume loop, the diastolic portion (Fig. 3, from point A to point B) could be obtained by using the single-beat solution, recently tested with invasive measurements (13). Although the clinical implications of this approach remain to be determined in future studies, its fully noninvasive nature may have significant potential in terms of assessing both myocardial contractility and relaxation properties in a variety of disease states.

The development of a reliable and reproducible, fully noninvasive technique to estimate LV elastance could be of interest in several settings. For example, assessment of the effects of new drugs on contractility and $C_{LV-A}$ could prove useful as complementary information to the standard end points of LV performance (i.e., EF) routinely used in clinical trials. Another potential clinical use of the noninvasive assessment of the ventricular-arterial coupling is its predictive value for the outcomes of cardiovascular disease, as recently described in the context of myocardial infarction (1).

Conclusion. This is the first study to test the feasibility of using RT3DE-derived LV volumes in conjunction with arterial tonometry to noninvasively quantify LV elastance and $C_{LV-A}$. This approach was found to be reproducible and sensitive enough to detect expected differences in LV contractility. Due to its noninvasive nature, this methodology may have clinical implications in various disease states.

GRANTS

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS


REFERENCES


