Right ventricular outflow tract systolic excursion: a novel echocardiographic parameter of right ventricular function

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Received 6 November 2011; accepted after revision 24 February 2012; online publish-ahead-of-print 19 March 2012

Aims
Right ventricular (RV) function has important prognostic and therapeutic implications. Assessment of RV function using echocardiography is challenging. The aim of this study was to evaluate a new parameter of RV function, right ventricular outflow tract systolic excursion (RVOT_SE).

Methods and results
RVOT_SE was measured using M-Mode echocardiography from the parasternal short-axis view at the level of the aortic valve, and was defined as the systolic excursion of the RVOT anterior wall. RVOT_SE was measured in 50 patients (age 64 ± 18 years, 28 males) with normal RV function [RV fractional area change (FAC) ≥ 35% and tricuspid annular plane systolic excursion (TAPSE) ≥ 1.6 cm] and 40 patients (age 68 ± 12 years, 35 males) with reduced RV function (RV FAC < 35% and TAPSE < 1.6 cm). RV FAC was 46 ± 7% in the normal RV group and 22 ± 5% in the reduced RV group (P < 0.0001). TAPSE was 2.2 ± 0.4 cm in the normal RV group and 1.0 ± 0.2 cm in the reduced RV group (P < 0.0001). RVOT_SE was 9.6 ± 1.5 mm in the normal RV group and 1.7 ± 1.1 mm in the reduced RV group (P < 0.0001). RVOT_SE < 6 mm identified patients with reduced RV function with 100% sensitivity and 100% specificity. Survival at 1 year was 63% in patients with RVOT_SE < 6 mm and 84% in patients with RVOT_SE ≥ 6 mm, P = 0.004.

Conclusion
RVOT_SE is a novel, simple, and promising parameter for assessing RV function, and it is associated with poor survival.

Keywords
Right ventricle • Echocardiography • Myocardial contraction • M-Mode echocardiography

Introduction
Right ventricular (RV) function has important prognostic and therapeutic implications. Reduced RV contraction predicts poor survival in patients with left heart failure and in patients with tricuspid regurgitation (TR).1–3 RV function is important for selecting patients for left ventricular (LV)-assist device implantation and for referral to surgery of patients with isolated severe TR.3–5 Assessment of RV function using echocardiography can be challenging. Current quantitative methods such as 2D fractional area change (FAC), tricuspid annular plane systolic excursion (TAPSE), tissue Doppler S' and isovolumic myocardial acceleration (IVA), and 3D echocardiography all have limitations.6 FAC does not necessarily represent the ejection fraction (EF) of the entire RV, whereas the RV has a complex 3D structure, tissue Doppler parameters are angle dependent and there is limited normative data, and 3D echocardiography is limited by currently available imaging quality of RV borders.6–9

An additional tool for evaluating RV function in a simple and easily reproducible way is therefore needed. The right ventricular outflow tract (RVOT) has superficial circumferential muscle fibres, which causes radial RVOT contraction during systole.10 Surgeons frequently estimate RV function during surgery by looking at the RVOT contraction.

The aim of this study was to evaluate a new parameter of RV function, right ventricular outflow tract systolic excursion (RVOT_SE) using M-Mode echocardiography.

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Methods

Patient population
We studied 90 patients referred for echocardiography, who had adequate parasternal views.

Group 1 consisted of 40 consecutive patients with reduced RV function (FAC < 35% and TAPSE < 1.6 cm).

Group 2 consisted of 50 patients undergoing echocardiography during the same period as group 1 patients, with normal RV function (FAC ≥ 35% and TAPSE ≥ 1.6 cm).

Demographic and clinical data were collected for all patients, and vital status and cause of death were determined at a median follow-up of 463 days (range 1–882 days). The study protocol was approved by the local ethics committee.

Echocardiography
Transthoracic echocardiography was performed using a commercially available echocardiographic system (Philips IE33, Eindhoven, Netherland, with an S3 1–3 MHz broadband transducer, or General Electric Vivid q or S6, Horten, Norway, with an M4S-RS 1.5–3.6 MHz broadband transducer). Both system types were used similarly in the two groups and we found no difference in M-mode or tissue Doppler measurements between the systems. A complete echocardiographic study was performed using standard views and techniques according to established guidelines, and data were digitally recorded for off-line analysis.11

RVOT_SE was measured from magnified cine-loops of the RVOT area acquired from the parasternal short-axis view using M-Mode echocardiography. Imaging was performed at the level of the aortic valve at maximal RVOT diameter, with the ultrasound beam perpendicular to the RVOT walls, after optimization of focus, gain, and compression settings. RVOT_SE is defined as the systolic excursion of the endocardial surface of the anterior wall of the RVOT relative to the transducer (Figure 1). Each data point was averaged from three measurements. Reproducibility of RVOT_SE was assessed by repeating the measurements in 10 patients with normal RV function and 10 with reduced RV function by the same observer at >1 month interval and by a second observer. Both observers were blinded to all data except the M-mode images.

We also measured RVOT fractional shortening [100 × (RVOT diastolic diameter − RVOT systolic diameter)/RVOT diastolic diameter] using the same M-Mode images, as reported by Lindqvist et al.12

RV FAC was measured from the four-chamber view with a focus on the RV. The RV area (endocardial borders excluding trabeculi and papillary muscles) was measured at the end of diastole and the end of systole. FAC was calculated using the formula: FAC (%) = 100 × (diastolic RV area − systolic RV area)/diastolic RV area.13 TAPSE was measured using M-Mode echocardiography from the four-chamber view at the lateral tricuspid annulus.14 Tissue Doppler imaging was performed using the four-chamber view and pulsed-wave tissue Doppler of the lateral tricuspid valve annulus. The ultrasound beam was aligned with the longitudinal axis of the tricuspid valve annulus motion, and peak systolic velocity (S’) and IVA were measured.15 Systolic pulmonary artery pressure (sPAP) was estimated using TR peak velocity and right atrial pressure, which was estimated using inferior vena cava diameter and response to inspiration.11

Statistical analysis
Continuous variables are presented as means ± SD and categorical variables as absolute numbers and percentages. Baseline clinical and echocardiographic characteristics of the groups were compared using unpaired t-test for continuous variables and 2 × 2 tables with χ² statistics for categorical variables. RV and LV function parameters were compared using Spearman rank correlation. Inter-observer and intra-observer variability were calculated as a mean difference between the two measurements ± SD and data were presented using Bland–Altman plots.16 Multivariate linear regression was used to determine the effect of baseline characteristics on RVOT_SE. Survival curves according to RVOT_SE were constructed using the Kaplan–Meier method, and differences between curves examined using the log-rank test. Cox’s proportional hazard model was used...
to correct for age and sex. Differences were considered statistically significant at the two-sided $P < 0.05$ level.

### Results

**Patient characteristics**

Clinical characteristics of the patients with reduced ($n = 40$) and normal ($n = 50$) RV function are summarized in Table 1. Patients with reduced RV function were more likely to be males (88 vs. 56%, $P = 0.03$), and had a significantly higher incidence of coronary artery disease, heart failure, atrial fibrillation, and chronic lung disease. Patients with reduced RV function had also larger left atria and ventricles and a lower LVEF (Table 2). Significant valvular disease was infrequent in both groups, except for TR which was more common in patients with reduced RV function (35 vs. 2%, $P = 0.0001$). As expected, patients in group 1 had significantly larger RV and right atria, higher sPAP and right atrial pressure, and significantly lower values of all RV function parameters (Table 2).

**RVOT_SE as a parameter of RV systolic function**

RVOT_SE was significantly lower in patients with reduced RV function when compared with patients with normal RV function: 1.7 ±

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### Table 1  Patient characteristics

<table>
<thead>
<tr>
<th></th>
<th>Group 1: reduced RV ($n = 40$)</th>
<th>Group 2: normal RV ($n = 50$)</th>
<th>$P$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>68 ± 12</td>
<td>64 ± 18</td>
<td>0.2</td>
</tr>
<tr>
<td>Sex (males)</td>
<td>35 (88%)</td>
<td>28 (56%)</td>
<td>0.03</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>30 (75%)</td>
<td>13 (26%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>32 (80%)</td>
<td>7 (14%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Hypertension</td>
<td>20 (40%)</td>
<td>29 (58%)</td>
<td>0.6</td>
</tr>
<tr>
<td>Chronic lung disease</td>
<td>7 (18%)</td>
<td>0</td>
<td>0.007</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>1 (3%)</td>
<td>2 (4%)</td>
<td>0.8</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>9 (22%)</td>
<td>0</td>
<td>0.002</td>
</tr>
</tbody>
</table>

RV, right ventricle.

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### Table 2  Echocardiographic characteristics

<table>
<thead>
<tr>
<th></th>
<th>Group 1: reduced RV ($n = 40$)</th>
<th>Group 2: normal RV ($n = 50$)</th>
<th>$P$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEDD (cm)</td>
<td>5.3 ± 1.0</td>
<td>4.7 ± 0.6</td>
<td>0.0005</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>35 ± 17</td>
<td>63 ± 8</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Left atrium (cm)</td>
<td>4.5 ± 0.7</td>
<td>3.8 ± 0.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td>4 (10%)</td>
<td>3 (6%)</td>
<td>0.8</td>
</tr>
<tr>
<td>Aortic regurgitation</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Mitral stenosis</td>
<td>1 (3%)</td>
<td>0</td>
<td>0.9</td>
</tr>
<tr>
<td>Mitral regurgitation</td>
<td>3 (8%)</td>
<td>2 (4%)</td>
<td>0.8</td>
</tr>
<tr>
<td>Tricuspid regurgitation</td>
<td>14 (35%)</td>
<td>1 (2%)</td>
<td>0.0001</td>
</tr>
<tr>
<td>sPAP (mmHg)</td>
<td>55 ± 19</td>
<td>32 ± 10</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Right atrial pressure (mmHg)</td>
<td>13 ± 5</td>
<td>5 ± 0.4</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Right atrial area (cm$^2$)</td>
<td>22 ± 5</td>
<td>15 ± 3</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>RV diameter (mid, cm)</td>
<td>4.2 ± 0.6</td>
<td>3.2 ± 0.4</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Diastolic RV area (cm$^2$)</td>
<td>26 ± 5</td>
<td>16 ± 5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Systolic RV area (cm$^2$)</td>
<td>20 ± 5</td>
<td>8.5 ± 3</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>RV FAC (%)</td>
<td>22 ± 5</td>
<td>46 ± 7</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>TAPSE (cm)</td>
<td>1.0 ± 0.2</td>
<td>2.2 ± 0.4</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>RVOT FS (%)</td>
<td>17 ± 7</td>
<td>47 ± 7</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>S’ at TV annulus (cm/s)$^b$</td>
<td>7.8 ± 2.7</td>
<td>14.7 ± 3.4</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>IVA (m/s$^2$)</td>
<td>2.0 ± 0.7</td>
<td>3.0 ± 1.2</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

FAC, fractional area change; IVA, isovolumic acceleration; LVEDD, left ventricular end diastolic diameter; LVEF, left ventricular ejection fraction; RV, right ventricle; RVOT FS, right ventricular outflow tract fractional shortening; sPAP, systolic pulmonary artery pressure; TAPSE, tricuspid annulus systolic excursion; TV, tricuspid valve.

$^a$At least moderate.

$^b$Pulsed wave peak systolic velocity at the lateral tricuspid annulus (S’).
1.1 vs. 9.6 ± 1.5 mm, P < 0.0001. There was a complete separation of RVOT_SE values between the two groups, and RVOT_SE of <6 mm identified patients with reduced RV function with absolute accuracy, 100% sensitivity, and 100% specificity (Figure 2). By comparison, values of tissue Doppler parameters, peak systolic TV annular velocity S′, and IVA, overlapped between the two groups and were not as useful in segregating between patients with reduced and normal RV function (Figure 3). RVOT_SE did not change with age. There was no correlation between age and RVOT_SE (P = 0.3). There was also no association between RVOT_SE and gender (P = 0.9 in group 1 and P = 0.8 in group 2).

Multivariate linear regression using relevant baseline characteristics (age, gender, coronary disease, congestive heart failure, atrial fibrillation, chronic lung disease, LV and left atrial size, LVEF, TR, and sPAP) showed that the only independent correlates with RVOT_SE were group assignment (P < 0.0001) and pulmonary pressure (P = 0.02). Figure 1 shows RVOT_SE in a patient with normal (Figure 1A) and reduced (Figure 1B) RV function.

RVOT_SE measurements were highly reproducible. Intra-observer variability was 0.1 ± 0.4 mm and inter-observer variability was 0.3 ± 0.7 mm (Figure 4).

RVOT fractional shortening was 17 ± 7% in group 1 (range 2–33%) and 47 ± 7% in group 2 (range 33–62%), P < 0.0001. Although a cut-off value <30% was 95% sensitive and 100% specific for RV dysfunction, the separation of RVOT_SE values between the two groups was better (Figure 2). The reason was that RVOT fractional shortening is affected by LV function as well, and patients with preserved LVEF and reduced RV function had higher RVOT...
fractional shortening (Figure 1). In patients with reduced RV function (group 1), there was a moderate correlation between RVOT fractional shortening and LVEF ($r = 0.55$, $P = 0.0003$), while there was no correlation between RVOT_SE and the LVEF ($r = 0.26$, $P = 0.1$).

**RVOT_SE and RV loading**

In an attempt to determine the effect of RV pressure loading on RVOT_SE, we correlated RVOT_SE with sPAP. There was a moderate correlation between RVOT_SE and sPAP ($r = 0.44$, $P = 0.0005$), similar to TAPSE and sPAP ($r = 0.46$, $P = 0.0003$). After correction for patient group using multivariate regression, the correlation between RVOT_SE and sPAP was still significant, but weaker ($P = 0.02$). There was no correlation between RVOT_SE and sPAP in the subgroup of patients with good RV function ($P = 0.2$) or reduced RV function ($P = 0.8$).

**RVOT_SE and survival**

During a median follow-up time of 463 days, 22 (24%) patients died. Overall survival was worse for patients with RVOT_SE < 6 mm when compared with patients with RVOT_SE ≥ 6 mm ($P = 0.004$, Figure 5). One-year survival was 63% with RVOT_SE < 6 mm and 84% for RVOT_SE ≥ 6 mm. RVOT_SE < 6 mm predicted survival even after correcting for age and sex ($P = 0.04$). RVOT_SE was 4.8 ± 0.4 mm in patients who died during follow-up, compared with 6.5 ± 0.4 mm in patients who survived ($P = 0.08$). The cause of death in group 1 (reduced RV) was heart failure in seven patients, myocardial infarction in one, septic shock in three and undetermined in three. In group 2 the cause of death was septic shock in two patients, cancer in one and undetermined in five.

**Discussion**

In the present study, we evaluated a novel parameter of RV function using M-Mode echocardiography of the RVOT from the parasternal short-axis view—RVOT_SE. We found RVOT_SE to be a simple, quick, reproducible and highly distinctive parameter of RV function. The difference in RVOT_SE between patients with normal and reduced RV function was so obvious, that in many cases it can be evaluated visually without a need for a precise measurement (Figure 1). We were surprised to find how separated were RVOT_SE measurements between patients with normal as opposed to reduced RV function (Figure 2). RVOT_SE identified patients with reduced RV function more accurately than tissue Doppler parameters such as tricuspid annular peak systolic velocity ($S'$) or IVA. This method also appears to have important prognostic value, since survival of patients from the
reduced RVOT_SE group was significantly worse compared with the group with normal RVOT_SE.

To the best of our knowledge, the use of RVOT_SE as a parameter of RV function has never been reported before. Lindqvist et al. reported their findings with RVOT fractional shortening using M-Mode echocardiography. In their study, RVOT fractional shortening moderately correlated with TAPSE, and moderately and inversely correlated with RV-axial systolic pressure gradient. In Lindqvist’s study; however, RV function was defined only by TAPSE, pulmonary hypertension was used as a surrogate parameter of RV function and harmonic imaging was not used. In our study, RVOT fractional shortening separated well between patients with reduced and preserved RV function, but separation of RVOT_SE values was better, due to the fact that RVOT fractional shortening is affected by LV function as well, whereas LV function has no effect on RVOT_SE. In patients with good LV function the aorta is pushed anteriorly in systole, contributing to RVOT fractional shortening even in the presence of reduced RV function (Figure 1).

RVOT_SE was found to be a parameter of global RV function, although RVOT_SE directly measures contraction of the RVOT region, which results from superficial circular muscle fibre shortening. This area is less susceptible to ischaemia and regional RV abnormalities than other areas, such as the lateral free wall, which is often involved in RV infarction and can affect TAPSE even when global RV function is largely preserved. RVOT_SE also has the potential to be less sensitive to preload and afterload, because of the small diameter of the RVOT (relative to RV diameters at the base), resulting in a much lower wall stress according to Laplace’s law. We found a moderate effect of sPAP and no effect of TR on RVOT_SE. Further studies including animal studies may be needed to determine the effect of loading and especially afterload on RVOT_SE.

The present study showed that RVOT_SE <6 mm was associated with poor survival irrespective of age and sex, reflecting the poor prognosis of patients with reduced RV function. The same survival curves would have been produced using group assignment (group 1 vs. group 2), TAPSE or RV FAC. Our study could not establish RVOT_SE as an independent predictor of survival (see limitations).

Further study is needed to determine the value of RVOT_SE compared with established echocardiographic parameters in defining RV systolic function. Future studies should also determine the ability of RVOT_SE to predict prognosis and response to therapy, such as tricuspid valve repair or replacement in patients with severe TR and questionable RV function.

Study limitations

We defined RV function as normal or impaired according to both FAC and TAPSE, in accordance with current guidelines recommending use of at least two echocardiographic parameters of RV function. Use of non-echocardiographic and more accurate modalities to define RV function, such as magnetic resonance imaging or computed tomography, would have allowed us to better define RV function and to compare RVOT_SE to TAPSE and FAC.

The two study groups had different baseline characteristics (such as coronary and lung disease and LVEF) that could affect survival. The design of the study, aimed at establishing RVOT_SE as an echocardiographic parameter of RV function and not as a predictor of outcome, and the relatively small number of events in the study did not allow us to perform multivariate analysis to determine whether RVOT_SE is an independent predictor of survival.

Conclusions

RVOT_SE is a novel and promising echocardiographic parameter of systolic RV function. It is simple to get, requiring one easy and reproducible M-Mode measurement from the parasternal short-axis view. It can accurately separate patients with normal and reduced RV function. Larger studies are needed to further establish the value of RVOT_SE when compared with the currently used echocardiographic methods and to other imaging techniques in patients with various disease states affecting the right heart.

Conflict of interest: none declared.

References


Transthoracic echocardiography demonstrated the left ventricular (LV) ejection fraction of 35%, severe tricuspid regurgitation, calcified trileaflet aortic valve with severe stenosis (see Supplementary data online, Video S1), and a valve area of 0.6 cm². The peak and mean gradients were 60 and 34 mmHg, respectively.

Thoracic computed tomography (CT) showed severe and diffuse calcifications of the thoracic aorta (‘porcelain aorta’; Panels A and B) which precluded standard surgical aortic valve replacement (AVR) using aortic cross clamping. He was then considered for a transcatheter aortic-valve implantation (TAVI); however, his aortic annulus was too large for TAVI.

Instead, he underwent successful on-pump implantation of an LV apex to the descending thoracic aorta conduit with a porcine bioprosthetic valve (Freestyle®, Medtronic, Inc.). Postoperative contrast-enhanced cardiac CT with retrospective gating demonstrated the proximal end of the conduit inserting normally into the apical lateral wall (Panel C), and the distal end into an area of the descending thoracic aorta relatively spared from severe atherosclerosis (Panels D and E, see Supplementary data online, Video S2). Inside the conduit, the bioprosthetic valve leaflets (Image F) were seen opening and closing normally (see Supplementary data online, Video S3).

For patients with severe aortic stenosis who are not suitable candidates for either surgical AVR or TAVI, an apico-aortic conduit is a reasonable therapeutic option. Cardiac CT imaging is useful to identify potential immediate complications (i.e. apical pseudoaneurysms or kinking of the conduit) and for assessment of conduit valve function.

Supplementary data are available at European Heart Journal – Cardiovascular Imaging online.