The Impact of Arterial Hypertension on Right Ventricular Deformation

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06.04.2012
• Conflicts: None
Why we should measure the RV function?

• RV function may be impaired from:
  - PAH
  - Acute/chronic PE
  - CHD
  - Valvular heart diseases
  - LV dysfunction
  - Secondary from coronary heart diseases, cardiomyopathies
  - AH
Why we should measure the RV function?

- RV is not just a conduit of blood flow: has its unique function
- Prognostic significance in various clinical settings
- Risk stratification or guide to optimal therapy
RV function and prognosis

- RV ejection fraction: an indicator of increased mortality in patients with CHF associated with CAD
  (Polak et al. J Am Coll Cardiol 1983)

- RV function predicts exercise capacity and survival in advanced heart failure
  (Di Salvo et al. J Am Coll Cardiol 1983)

- RV function is a crucial determinant of short-term prognosis in severe chronic heart failure
  (Gavazzi et al. J Heart Lung Transplant 1997)
RV function and prognosis

- RV ejection fraction: independent predictor of survival in patients with moderate heart failure
  (De Groote et al. J Am Coll Cardiol 1998)

- RV function predicts prognosis in patients with chronic pulmonary disease

- RV contractile reserve is associated with one year mortality in patients with DCMP
The echocardiographic assessment of the right ventricle: what to do in 2010?

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Received 17 August 2009; accepted after revision 28 December 2009; online publish-ahead-of-print 2 February 2010

For many years, the echocardiographic quantitative assessment of right ventricular (RV) function has been difficult owing to the complex RV anatomy. Identifying an accurate and reliable echocardiographic parameter for the functional assessment of the RV still remains a challenge. The review presents a summary of the most studied and presently used parameters of RV function, with their reported normal values, as well as advantages and limitations of use. Combinations of these parameters are used in daily clinical practice, each one offering only partial information about the status of the RV. Myocardial velocity and strain rate imaging have promising results in the assessment of RV function. There is hope that novel myocardial deformation parameters and three-dimensional echocardiography-derived parameters may add value to the examination of the RV, but validation studies are still needed.

Keywords
Right ventricle • Echocardiography • Tissue Doppler • Strain rate imaging • 3D echocardiography
Doppler Echocardiography: Tissue Doppler Imaging

- Allows quantitative assessment of RV systolic and diastolic function by measurement of myocardial velocities

- **Peak systolic velocity (PSV)**
  - PSV < 11.5 cm/s identifies the presence of RV dysfunction
  - Sensitivity of 90%, specificity of 85%
  - Less affected by HR, loading condition, and degree of TR

- **Tricuspid lateral annular velocities**
  - Reduced in patients with inferior MI and RV involvement
  - Associated with the severity of RV dysfunction in patients with heart failure
Normal RV Regional Function

RV longitudinal motion

Pulsed Doppler

(4CH view)
RV free wall

Colour Doppler
Normal RV Regional Function

RV longitudinal motion

Systolic ring excursion vs ring velocity

Tricuspid ring

Mmode annular motion

DMI annular velocity
Doppler Echocardiography: Strain Rate Imaging

- RV longitudinal strain in apical view
  - Feasible in clinical setting
  - Baso-apical gradient with higher velocities at the base
  - RV velocities are consistently higher as compared to LV

- Strain and strain rate values
  - More inhomogeneously distributed in the RV
  - Reverse baso-apical gradient, reaching the highest values in the apical segments and outflow tract

- Acute increase in RV afterload
  - Increase in RV myocardial strain rate
  - Decrease in peak systolic strain, indicating a decrease in SV
Normal RV Longitudinal Deformation

Strain rate

Strain

Mean RR interval

Mean RR interval

RV lateral wall

apical

medial

basal
Speckle tracking
Comparison of Strain values in Different studies

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</thead>
<tbody>
<tr>
<td>RV basal Free wall</td>
<td>-27.817± 5.77 N 109</td>
<td>-28.69± 4.62</td>
<td>-33.802± 7.03</td>
<td>-44.8± 10.2</td>
<td>-22.8± 10.2</td>
<td>-19. ±6</td>
</tr>
<tr>
<td>RV Mid Free wall</td>
<td>-27.179± 5.6</td>
<td>-25.21± 4.97</td>
<td>-32.69± 5.9</td>
<td>-26.9± 5.3</td>
<td>-24.4± 7.7</td>
<td>-27. ±6</td>
</tr>
</tbody>
</table>

Basal to apical reduction in strain
Apiacal to basal reduction in strain
RV Global Function vs EF

\[ \text{Strain} = 0.08186 - 0.0047 \times \text{EF} \]

Correlation: \( r = -0.6902 \)

RV ejection fraction - % (MRI)

F. Rademakers  JACC, 2000
DTI vs. STE

A. TDI – derived strain
B. STE derived strain

Comparison between 2D strain and TDI

![Graph A: Scatter plot showing the relationship between peak systolic strain 2DSE (%) and peak systolic strain TDI (%). The correlation coefficient (r) is 0.73. The equation is 2DSE = 0.63(TDI) + 8.88.]

![Graph B: Scatter plot showing the difference between 2DSE and TDI strain (%). The data points are distributed around the baseline.]
RV function and AH

• RV performance in hypertensive's in not well evidenced

• Bernheim’s syndrome  (Selzer, 1955, Russek 1950)

↑ peripheral venous pressure
↑ RA pressure

RIGHT HEART FAILURE
RV function and AH

LV hypertrophy, LV pressure, LV diastolic dysfunction

RV hypertrophy, RV pressure, RV diastolic dysfunction

W. Motz. Internist, 2004
RV function and AH

- RV EF % in hypertensive's with LVH is higher than without LVH
  
  *(Olivari, Circulation, 1978)*

- Hypertensive pts have higher RV pressure than normal's and RV performance is lower RV EF %
  
  *(J. Ferlinz, Circulation, 1980)*

- Invasive studies showed the pathology of pulmonary circulation in pts with AH
  
  *(R. Fagard, JACC, 1998)*

- MRI study of RV remodelling in systemic hypertension
  
  *(Pedrinelli, Heart, July 2011)*
RV function and AH

- **Echocardiography studies** *(Myslinski, 1998, Akkos 1999)* about RV function and diastolic parameter
- AH is associated to RV longitudinal diastolic dysfunction – pulsed TDI imaging
  *(S. Cicala, EJEcho, 2002)*
- TDI provide quantitative analysis of RV alteration in hypertensive's
  *(Tumuklu, AJE, 2007)*
- Abnormal RV mechanic of IVS in hypertensive’s
  *(R. Pedrinelli, EJ Echo, 2010)*
The impact of arterial hypertension on right ventricular deformation

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²Catholic University of Leuven - Leuven - Belgium

• First STE study with hypertensive patients
Purpose:

- HT has long been known to be a major risk factor for heart failure.
- The Framingham study provides some of the best evidence with regard to the association between BP and HF. Around 90% of those developing HF had an prior history of HT. This is three times the risk seen in normotensives.
- HT is the most important modifiable factor in congestive cardiac failure. However, almost 40% of episodes are associated with diastolic dysfunction.
- Diastolic dysfunction ranges from a failure of end diastolic volume to rise appropriately with exercise to that of overt diastolic HF.
Aim:

The aim of our study was to determine the effect of arterial hypertension and hypertensive cardiac remodelling on right ventricular (RV) function, using vector velocity echocardiography to determine strain and strain rate.
Methods:

- **40 patients** with arterial hypertension and left ventricular hypertrophy (LVH group) with **20 healthy control subjects**. None of the hypertensive patients had symptomatic heart failure.

- Apical four-chamber images were acquired (frame rate 74 ± 6 frames/s) with Aloka alfa-10 and analyzed offline on Syngo US Workplace 2007, Siemens AG. in order to extract the strain (rate) curves.

- From these, the maximal systolic strain (PSS) and peak strain rate (PSR) on right and left ventricle were derived, using vector velocity imaging (VVI) software. Tricuspid annular plane systolic excursion (TAPSE) and as well as mid-apical and basal peak ejection strain (S) and strain rate (SR) of the RV free wall were measured.
2DSTE
2DSTE
2DSTE - VVI
2DSTE- VVI
Results:

Demographic and baseline data of study population

<table>
<thead>
<tr>
<th></th>
<th>AH - groups</th>
<th>Normals</th>
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</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td>68.9±13</td>
<td>31±5</td>
</tr>
<tr>
<td><strong>Height (cm)</strong></td>
<td>173.14±9.45</td>
<td>174±4</td>
</tr>
<tr>
<td><strong>Weight (kg)</strong></td>
<td>82.74±13.8*</td>
<td>67±11</td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td>30M/10F</td>
<td>12M/8F</td>
</tr>
<tr>
<td><strong>BSA( m2)</strong></td>
<td>1.99± 0.21</td>
<td>1.8±0.11</td>
</tr>
<tr>
<td><strong>HR (bpm)</strong></td>
<td>67.5±12.4</td>
<td>78.8±3.4</td>
</tr>
<tr>
<td><strong>SBP (mmHg)</strong></td>
<td>156.8±13.7*</td>
<td>115±12</td>
</tr>
<tr>
<td><strong>DBP (mmHg)</strong></td>
<td>87±12*</td>
<td>67±9</td>
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</table>
### Echocardiographic data of the study population

<table>
<thead>
<tr>
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<th>AH - groups</th>
<th>Normals</th>
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</thead>
<tbody>
<tr>
<td><strong>LVEF (%)</strong></td>
<td>62±6.3</td>
<td>66±10,1</td>
</tr>
<tr>
<td><strong>LVEDD (mm)</strong></td>
<td>48.7±8.7</td>
<td>45±4,4</td>
</tr>
<tr>
<td><strong>LVEDD/BSA (mm/m^2)</strong></td>
<td>24.47±4.7</td>
<td>25±3,7</td>
</tr>
<tr>
<td><strong>LVEDV (ml)</strong></td>
<td>109.6±31.10*</td>
<td>81.11±25*</td>
</tr>
<tr>
<td><strong>LVEDV/BSA (ml/m^2)</strong></td>
<td>54.77±14.8*</td>
<td>45.06±12.3*</td>
</tr>
<tr>
<td><strong>LVESV (ml)</strong></td>
<td>48.71±13.60*</td>
<td>38.37±13*</td>
</tr>
<tr>
<td><strong>LVESV/BSA (ml/m^2)</strong></td>
<td>24.48±6.7</td>
<td>21.31±6.8</td>
</tr>
<tr>
<td><strong>LVSV (ml)</strong></td>
<td>60.56±10</td>
<td>61.46±16</td>
</tr>
<tr>
<td><strong>LVSV/BSA (ml/m^2)</strong></td>
<td>30.43±5.5</td>
<td>34.14±8,2</td>
</tr>
<tr>
<td><strong>mass (g)</strong></td>
<td>264.30±.765*</td>
<td>173±11.3</td>
</tr>
<tr>
<td><strong>mass index(g/m^2)</strong></td>
<td>132.3±30.9*</td>
<td>92.02±5.6</td>
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<tr>
<td><strong>E/ A</strong></td>
<td>0.75 ± 0.41*</td>
<td>1.87 ± 0.48</td>
</tr>
<tr>
<td><strong>TAPSE (mm)</strong></td>
<td>21 ± 2,9 mm</td>
<td>23 ± 2,2 mm</td>
</tr>
</tbody>
</table>
Results:

<table>
<thead>
<tr>
<th>Global strain / rate</th>
<th>Normals (1)</th>
<th>AH - group (2)</th>
<th>p-value (2) vs (1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PeakSS(_{LV})</td>
<td>-19.69±1.82</td>
<td>16.4 ± 3.14</td>
<td>p=0.56</td>
</tr>
<tr>
<td>PeakSS(_{RV})</td>
<td>-15, 5 ± 2, 3</td>
<td>-10.03 ±4, 5 *</td>
<td>p&lt;0.01</td>
</tr>
<tr>
<td>PeakSR(_{RV})</td>
<td>1.75</td>
<td>0.45 *</td>
<td>p&lt;0.01</td>
</tr>
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</table>
### Results:

**Regional right ventricular myocardial function**

<table>
<thead>
<tr>
<th></th>
<th>Peak Systolic Strain – RV</th>
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<tbody>
<tr>
<td></td>
<td>Basal level</td>
</tr>
<tr>
<td>Normals</td>
<td>-14.03 ± 3.78</td>
</tr>
<tr>
<td>AH group</td>
<td>12, 31 ±3, 87</td>
</tr>
</tbody>
</table>
Conclusion:

• The present study demonstrates that measures of RV deformation are reduced in patients with LVH secondary to hypertension.

• Thus, this data suggests that LVH may cause early sub-clinical RV dysfunction even in the absence of overt diastolic heart failure.
Take home message:

- RV function is an important parameter in cardiac disease
- 2DE is a relatively feasible method to assess RV dysfunction in clinical practice
- Several new echocardiographic techniques such as TDI, SRI, RT3DE may give us further information in assessing RV function


“Don’t forget to check your Facebook account right before leaving, you might get a new friend request!”

Prognosis

Mark Elliot Zuckerberg
Thank you for your attention!
RV Systolic Function

LONGITUDINAL vs RADIAL FUNCTION
Longitudinal vs radial RV systolic velocities in young (16-40 y, n=19) and old normals (41-76 y, n=13)

*p=0.01  **p=0.005

LONGITUDINAL vs RADIAL FUNCTION

RV radial systolic motion

RV longitudinal systolic motion

systolic velocity (cm/s)

young normals

old normals
Normal RV Regional Function

RV DIASTOLIC FUNCTION

Early diastolic (e) velocities in young (16-40y) and old normals (41-76y)

* p<0.05