Assessment of left atrial function: does it offer added value?

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EAE Course, Sofia, April 2012
Case presentation

Grad = 113 mmHg

F, 54 y/o

Grad = 117 mmHg

M, 66 y/o
LAVi = 73 ml/m²

LAε = 10%

LAVi = 72 ml/m²

LAε = 7.8%
NYHA II

Sinus rhythm after 1 year follow-up

ASr = -0.83 s⁻¹

NYHA III

Atrial fibrillation after 1 year follow-up

ASr = -0.49 s⁻¹
Symptoms of heart failure were related to the severity of LA dysfunction.

Rosca M, Popescu BA et al. JASE 2010

Table 4 Correlates of symptomatic status in patients with hypertrophic cardiomyopathy

<table>
<thead>
<tr>
<th>Variables</th>
<th>Univariate analysis</th>
<th>Multivariable analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR</td>
<td>95% CI</td>
</tr>
<tr>
<td>Age</td>
<td>1.027</td>
<td>0.963-1.073</td>
</tr>
<tr>
<td>LVε</td>
<td>1.215</td>
<td>0.954-1.549</td>
</tr>
<tr>
<td>LAVi</td>
<td>1.091</td>
<td>1.018-1.170</td>
</tr>
<tr>
<td>ASr</td>
<td>3.377</td>
<td>1.349-8.616</td>
</tr>
<tr>
<td>MR degree</td>
<td>2.277</td>
<td>0.969-5.353</td>
</tr>
<tr>
<td>Presence of LV outflow tract obstruction (Y/N)</td>
<td>0.476</td>
<td>0.118-1.929</td>
</tr>
<tr>
<td>Peak LV outflow tract gradient</td>
<td>1.010</td>
<td>0.981-1.041</td>
</tr>
</tbody>
</table>
The mechanical function of the left atrium plays an important role in the overall cardiovascular performance.

The LA contributes up to 30% of total LV stroke volume in normal subjects.

The atrial contribution is particularly important in the setting of LV dysfunction, where this percentage may become higher.

The loss of atrial contribution to LV filling and stroke volume in atrial fibrillation often leads to symptomatic deterioration.

Importance of left atrial function
The left atrium and cardiovascular outcome

• The LA is an important determinant of cardiovascular morbidity and mortality

• This was shown both in the general population (LA size correlated with cardiovascular diseases or adverse clinical outcomes), and in patients with various pathological conditions

# Left atrial function

The key function of the left atrium is to modulate left ventricular filling and cardiovascular performance.

<table>
<thead>
<tr>
<th>LA function</th>
<th>Timing in cardiac cycle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reservoir</td>
<td>Ventricular systole</td>
</tr>
<tr>
<td>Conduit</td>
<td>Early diastole</td>
</tr>
<tr>
<td>Pump</td>
<td>End diastole</td>
</tr>
</tbody>
</table>

In addition:

- volume sensor of the heart, releasing natriuretic peptides
- contains receptors for various reflexes
The Frank-Starling mechanism is also operative in the LA (LA output increases as atrial diameter increases, which contributes to maintaining a normal stroke volume)

Payne RM et al. *J Appl Physiol* 1971

LA active emptying might decrease in the presence of severe LA dilation as the optimal Frank-Starling relationship is exceeded

Pagel PS et al. *Anesthesiology* 2003
Pathophysiology

Reservoir function
Modulated by:
- LV contraction
- RV systolic pressure
- LA relaxation
- LA chamber stiffness

Conduit function
Modulated by:
- LV relaxation
- LA afterload

Contractile function
Modulated by:
- LV compliance
- LA afterload (LV filling pressures)
- LA preload
- Intrinsic LA contractility
LA function assessment

Invasive assessment

• LA pressure-volume relationship
  – gold standard

Echocardiographic assessment

• Conventional parameters
• New techniques (TDI/STE/3D echocardiography)

Assessment of LA function by echocardiography

- PW Doppler of transmitral flow
- PW Doppler of pulmonary venous flow
- Left atrial phasic volumes
- Myocardial velocities (TDI)
- Atrial deformation imaging (TDI/STE)
Conventional LA echocardiographic parameters

Limitation: influenced by loading conditions

LA expansion index = \( \frac{\text{Vol}_{\text{max}} - \text{Vol}_{\text{min}}}{\text{Vol}_{\text{min}}} \times 100 \) - reservoir

LA passive emptying fraction = \( \frac{\text{Vol}_{\text{max}} - \text{Vol}_p}{\text{Vol}_{\text{max}}} \times 100 \) - conduit

LA active emptying fraction = \( \frac{\text{Vol}_p - \text{Vol}_{\text{min}}}{\text{Vol}_p} \times 100 \) - contractile
Myocardial velocities

• TDI allows the measurement of myocardial velocities, providing a less load dependent measure of both LV systolic and diastolic function.

Peak a’ wave velocity
- marker of atrial function

Limitations:
- angle dependent, influenced by translation and tethering

Contractile function
Regional LA function by TDI

Color TDI allows the simultaneous assessment of several atrial segments

Parameters:
- velocities of different atrial segments
- atrial electomechanical delay

Limitations:
- inability to distinguish atrial contraction from mitral annular and ventricular motion
The technique has a good site specificity - the longitudinal shortening and lengthening of the LA are opposite in time to those of the LV.

Limitations
- angle dependent
- time-consuming (the wall-by-wall sampling limits the use in clinical practice)
LA deformation imaging by STE

Angle-independent tool for a thorough assessment of LA performance

Limitations
- dependent on image quality
- STE has not been validated for LA function assessment
Atrial strain and strain rate imaging - TDI/STE

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Intra-observer variability</th>
<th>Inter-observer variability</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>95%CI</td>
</tr>
<tr>
<td>Peak $\varepsilon$ CT (%)</td>
<td>-24.5</td>
<td>± 2.6</td>
</tr>
<tr>
<td>$\varepsilon$--CT (%)</td>
<td>-17.9</td>
<td>± 1.6</td>
</tr>
<tr>
<td>Peak $\varepsilon$ R (%)</td>
<td>71.5</td>
<td>± 4.9</td>
</tr>
<tr>
<td>$\varepsilon$--R (%)</td>
<td>66</td>
<td>± 4.1</td>
</tr>
<tr>
<td>Peak $\varepsilon$ CD (%)</td>
<td>-54</td>
<td>± 6.3</td>
</tr>
<tr>
<td>$\varepsilon$--CD (%)</td>
<td>-47</td>
<td>± 3.1</td>
</tr>
<tr>
<td>Peak SR CT (s$^{-1}$)</td>
<td>-4.86</td>
<td>± 0.12</td>
</tr>
<tr>
<td>Peak SR ER (s$^{-1}$)</td>
<td>4.68</td>
<td>± 0.66</td>
</tr>
<tr>
<td>Peak SR LR (s$^{-1}$)</td>
<td>5.72</td>
<td>± 0.98</td>
</tr>
<tr>
<td>Peak SR CD (s$^{-1}$)</td>
<td>-7.58</td>
<td>± 1.82</td>
</tr>
</tbody>
</table>


<table>
<thead>
<tr>
<th>Parameter</th>
<th>Interobserver</th>
<th>Intraobserver</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Change in mean (%)</td>
<td>95% CI</td>
</tr>
<tr>
<td>Velocity (cms$^{-1}$)</td>
<td>-5.7</td>
<td>(-5.8 to -5.6)</td>
</tr>
<tr>
<td>Strain (%)</td>
<td>-6.5</td>
<td>(-6.6 to -6.5)</td>
</tr>
<tr>
<td>Strain rate (s$^{-1}$)</td>
<td>2.5</td>
<td>(2.4–2.6)</td>
</tr>
</tbody>
</table>

Clinical applications

- Left ventricular dysfunction
- Cardiomyopathies
- Valvular heart disease
- Atrial fibrillation
LA function in patients with LV dysfunction

Mitral annular velocities were assessed using colour TDI.

- systolic
- early diastolic
- late diastolic (a’)

Tan at al. *Heart* 2010;96:1017.
Hypertensive subjects w/o HF appear to be able to compensate for their increased E/E’ on exercise by increasing a’, which HFNEF patients failed to achieve.

Tan at al. *Heart* 2010;96:1017.
LA function in patients with LV dysfunction

75 patients with LV systolic dysfunction, CHF, NYHA class II to III.

LA function in patients with LV dysfunction

Stepwise Linear Multivariate Analysis

**Predictors of maximal workload** Echocardiographic parameters that were independently associated with maximal workload at a $P$ value of less than .05 were included in the model. The best prediction was obtained associating pulsed DTI Aa peak velocity with peak strain in the RV free wall and isovolumic relaxation time (milliseconds) ($R^2 = 34.3$).

**Predictors of peak $O_2$** Using the same stepwise protocol, the best predictive parameter of peak $O_2$ was the ratio $E/Ea$ ($R = 0.53$, $F = 7.7$, $P = .01$). After including LA volume and Aa ($R^2 = 54.6$) or peak strain in the LA septal wall ($R^2 = 50.2$) into the model, the correlation became stronger.

Adding a’ peak velocity in resting echocardiographic evaluation of patients with CHF could be useful.

MA-Aw was recorded in 96 patients with LV systolic dysfunction who were followed for 29 ± 10 months.

• MA-Aw < 5 cm/s was the most powerful predictor of cardiac death or hospitalization for worsening HF compared with clinical, hemodynamic, and the other echocardiographic variables.

LA function in cardiomyopathies

36 consecutive pts with DCM were studied.

After inotropic stimulation

LAEF $\uparrow$ $\rightarrow$ $VO_2 > 14 \text{ mL/kg/min}$

LAEF $\downarrow$ $\rightarrow$ $VO_2 < 14 \text{ mL/kg/min}$

LA contractile reserve impairment might be an earlier finding in the process of heart failure.

LA longitudinal function is reduced in HCM compared to non-HCM LVH and healthy controls.

2D atrial strain has an additive value in differentiating HCM from non-HCM LVH and it is more reproducible than TDI strain.

Paraskevaidis et al. *Heart* 2009;95;483.
In multivariate analysis (including age, LAV, MVA, and LA SR average), the best predictor of events in pts with MS was LA peak systolic SR.

In ROC analysis a cut-off value of 1.69 s\(^{-1}\) for LA peak systolic SR was associated with a sensitivity of 88%, a specificity of 80.6%, an area under the ROC curve of 0.85 in predicting events.

In patients with severe AS, the 3 components of LA function are reduced. Active LA dysfunction is related to the AS severity. The impact of reduction in LA active function on the clinical status need further studies.

O’Connor K. Am J Cardiol 2010;106:1157.
55 y/o man

Case presentation
Same patient after 4 weeks
PW Doppler mitral inflow

LA stunning

3 days after spontaneous cardioversion to NSR

3 days later

PW Doppler tricuspid inflow

Normal RA function

Dissociated LA stunning
Atrial myocardial properties assessed by myocardial velocities, strain and strain rate imaging are reduced in patients with AF.

- 65 pts, lone AF; FU: 9 mo after cardioversion

LA function in atrial fibrillation

<table>
<thead>
<tr>
<th>Parameter</th>
<th>MSR Patients (n=25)</th>
<th>AFR Patients (n=40)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>60±11</td>
<td>57±11</td>
<td>0.6</td>
</tr>
<tr>
<td>Male sex, %</td>
<td>72</td>
<td>77</td>
<td>0.8</td>
</tr>
<tr>
<td>BSA, m²</td>
<td>1.8±0.2</td>
<td>1.9±0.2</td>
<td>0.7</td>
</tr>
<tr>
<td>AF duration, wk</td>
<td>8.2±2.9</td>
<td>8.6±2.6</td>
<td>0.5</td>
</tr>
<tr>
<td>Previous history of AF, %</td>
<td>65</td>
<td>64</td>
<td>0.3</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>136±12</td>
<td>134±9</td>
<td>0.4</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>81±8</td>
<td>79±7</td>
<td>0.3</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>93±24</td>
<td>85±19</td>
<td>0.1</td>
</tr>
<tr>
<td>IVS end-diastole, cm</td>
<td>1.1±0.1</td>
<td>1.1±0.1</td>
<td>&gt;0.9</td>
</tr>
<tr>
<td>LV end-diastole, cm</td>
<td>5.0±0.4</td>
<td>5.0±0.5</td>
<td>&gt;0.9</td>
</tr>
<tr>
<td>PW end-diastole, cm</td>
<td>1.1±0.1</td>
<td>1.1±0.1</td>
<td>&gt;0.9</td>
</tr>
<tr>
<td>LV ejection fraction, %</td>
<td>54±3</td>
<td>54±5</td>
<td>&gt;0.9</td>
</tr>
<tr>
<td>LV mass index, g/m</td>
<td>135±22</td>
<td>131±16</td>
<td>0.4</td>
</tr>
<tr>
<td>LA, cm</td>
<td>4.5±0.3</td>
<td>4.4±0.5</td>
<td>0.4</td>
</tr>
<tr>
<td>LA maximal volume, mL</td>
<td>66.5±16</td>
<td>62.7±18</td>
<td>0.4</td>
</tr>
<tr>
<td>LA compliance index, %</td>
<td>32±7</td>
<td>29±10</td>
<td>0.2</td>
</tr>
<tr>
<td>Pulmonary artery wedge pressure, mm Hg</td>
<td>7±6</td>
<td>8±7</td>
<td>0.6</td>
</tr>
<tr>
<td>LA appendage peak velocity flow, cm/s</td>
<td>39±12</td>
<td>32±15</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Peak Systolic Value</th>
<th>MSR Patients (n=25)</th>
<th>AFR Patients (n=40)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrial septum</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Velocity, cm/s</td>
<td>3.9±1.9</td>
<td>3.4±1.4</td>
<td>0.2</td>
</tr>
<tr>
<td>S, %</td>
<td>37±18</td>
<td>19±14</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>SR, s⁻¹</td>
<td>2.7±0.7</td>
<td>1.4±0.8</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LA lateral wall</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Velocity, cm/s</td>
<td>3.2±1.9</td>
<td>2.7±1.4</td>
<td>0.2</td>
</tr>
<tr>
<td>S, %</td>
<td>39±26</td>
<td>18±13</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>SR, s⁻¹</td>
<td>3±1.7</td>
<td>1.3±0.9</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>RA free wall</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Velocity, cm/s</td>
<td>5±2.3</td>
<td>4.3±2</td>
<td>0.2</td>
</tr>
<tr>
<td>S, %</td>
<td>58±44</td>
<td>33±23</td>
<td>0.003</td>
</tr>
<tr>
<td>SR, s⁻¹</td>
<td>3.8±1.6</td>
<td>2.3±1.6</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LA inferior wall</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Velocity, cm/s</td>
<td>3.7±1.8</td>
<td>2.9±1.9</td>
<td>0.2</td>
</tr>
<tr>
<td>S, %</td>
<td>33±27</td>
<td>17±9</td>
<td>0.0007</td>
</tr>
<tr>
<td>SR, s⁻¹</td>
<td>2.7±1</td>
<td>1.6±0.6</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LA anterior wall</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Velocity, cm/s</td>
<td>3.9±2</td>
<td>3±1.6</td>
<td>0.1</td>
</tr>
<tr>
<td>S, %</td>
<td>24±13</td>
<td>13±10</td>
<td>0.0002</td>
</tr>
<tr>
<td>SR, s⁻¹</td>
<td>2.1±0.6</td>
<td>1.4±0.8</td>
<td>0.0003</td>
</tr>
</tbody>
</table>
LA function in atrial fibrillation

- LA wall fibrosis by delayed-enhancement MRI is inversely related to LA strain/SR, and these are related to the AF burden.

Noninvasive imaging of LA fibrosis may be helpful in:
- predicting the risk of AF
- guiding therapeutic strategies
- predicting the outcomes in patients with AF

Conclusions

- An accurate assessment of LA function remains cumbersome.
- LA EF requires a skillful acquisition technique and calculations that are not routinely performed.
- The newer parameters assessed by TDI/STE are reproducible and probably more sensitive than traditional ones.
- A thorough evaluation of atrial function
  - may assist in the early detection of “subclinical disease”
  - could refine risk stratification
  - could guide therapy
- The extent of reversibility of LA remodeling with medical therapy, and the impact of such changes on outcomes need further studies.
Thank you!