RV and pulmonary circulation during exercise

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Disclosures: none
RV function is most relevant when the RV has to work

1. Pathological load
   - Increased afterload due to LV dysfunction
   - Pulmonary vascular disorders
   - Chronically increased preload

2. Activity, exercise, exertion
   - Increased RV wall stress
   - Increased RV work
   - Increased O$_2$ demand
The healthy RV at rest
- it doesn’t need to do anything

Brown and Ditchey *Circulation* 1988

Pagnamenta, Naeije et al.
*J Appl Physiol* 2010
The LV does **not** do all of the work

- Potentially misleading conclusions:
  - LV contributes substantially to RV pressure generation (Seki 1975, Yamaguchi 1991, Feneley 1995)

BUT

- When RV afterload is increased then, in the absence of a functional RV, CO rapidly falls (J Hoffman Thorac Cardiovasc Surg 1994)
Independent and Additive Prognostic Value of Right Ventricular Systolic Function and Pulmonary Artery Pressure in Patients With Chronic Heart Failure

Stefano Ghio, MD, FESC,* Antonello Gavazzi, MD, FESC,* Carlo Campana, MD,* Corinna Inserra, MD,* Catherine Klersy, MD,† Roberta Sebastiani, MD,* Eloisa Arbustini, MD,‡ Franco Recusani, MD,* Luigi Tavazzi, MD, FESC, FACC*
Why do we have a right ventricle?
Exercise = RV work

La Gerche, Prior et al.  
*J App Physiol* 2010

Argiento, Naeije et al.  
*Eur Resp J* 2010

Lewis, Semigran et al.  
*Circ Heart Failure* 2011
Investigating ventricular hemodynamics during exercise

- **Echocardiography**
  - Volumes
  - Cardiac Output
  - Pulmonary artery pressures
- **Cardiac MRI**
- **Radial arterial catheter**
Measuring wall stress

Hybrid technique

- Volumes and mass from CMR
- Change in volumes from echo VTI
- Pressures
  - SBP: arterial line
  - PASP: echo
Change in wall stress with exercise


**Left vs. Right Ventricle**

- **RV - all subjects**
- **LV - all subjects**

* $p < 0.0001$

$\sigma$ (Kdynes/cm$^2$)

Baseline: $p < 0.0001$

Peak Exercise: $p = 0.083$
...and after 8 hours
Methods - echocardiography

- **3D Echo (GE Vivid 7)**
  LV and RV full volume acquisitions
  TomTec software
  Validated against CMR

- **Strain and SR imaging**
  2D speckle tracking
  RV and LV separately
  60 - 90 frames per second

- **Traditional**
  RV Fractional Area Change
  Tricuspid Annular Plane Systolic Excursion
Results: RV but not LV dysfunction

<table>
<thead>
<tr>
<th>Measure</th>
<th>Baseline</th>
<th>Post-race</th>
<th>Follow-up</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Right Ventricular Measures</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RVEF (%)</td>
<td>51.0 ± 3.6</td>
<td>46.4 ± 6.5</td>
<td>50.0 ± 3.8</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>RVFAC (%)</td>
<td>51.5 ± 6.0</td>
<td>44.3 ± 11.2</td>
<td>49.8 ± 6.6</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>TAPSE</td>
<td>24.9 ± 3.9</td>
<td>24.0 ± 4.5</td>
<td>26.5 ± 4.1</td>
<td>0.035</td>
</tr>
<tr>
<td>RV strain (%)</td>
<td>27.2 ± 3.4</td>
<td>23.7 ± 3.7</td>
<td>25.6 ± 3.0</td>
<td>0.001</td>
</tr>
<tr>
<td>RV SRs (s⁻¹)</td>
<td>1.42 ± 0.24</td>
<td>1.26 ± 0.23</td>
<td>1.29 ± 0.19</td>
<td>0.008</td>
</tr>
<tr>
<td><strong>Left Ventricular Measures</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>56.4 ± 5.2</td>
<td>57.5 ± 5.6</td>
<td>58.8 ± 5.1</td>
<td>0.147</td>
</tr>
<tr>
<td>LV strain (%)</td>
<td>18.4 ± 3.7</td>
<td>16.9 ± 2.8</td>
<td>17.7 ± 2.3</td>
<td>0.071</td>
</tr>
<tr>
<td>LV SRs (s⁻¹)</td>
<td>0.98 ± 0.26</td>
<td>0.95 ± 0.15</td>
<td>0.89 ± 0.13</td>
<td>0.13</td>
</tr>
</tbody>
</table>

*ALL RV measures decreased whilst NO LV measures changed*

La Gerche, Heidbuchel, Prior et al. Eur Heart J In Press
RV *dilates* whilst the LV *shrinks*.

**End-diastolic Volume**
- Baseline: 170 ± 30 ml
- Post-race: +9 ml, p = 0.015

**End-systolic Volume**
- Baseline: 83 ± 17 ml
- Post-race: +13 ml, p < 0.001

La Gerche, Heidbuchel, Prior et al. Eur Heart J In Press
Effect of prolonged intense exercise on the RV

Pre

Post
Effect of prolonged intense exercise on the RV

Pre

Post
Effect of prolonged intense exercise on the RV

Pre

Post
The RV

“the Achilles heel of the exercising heart”
Why is the RV the Achilles heel?

<table>
<thead>
<tr>
<th></th>
<th>Left Ventricle</th>
<th>Right Ventricle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac Output (L/min)</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Vascular resistance (dyne-sec.cm$^5$)</td>
<td>1100</td>
<td>70</td>
</tr>
<tr>
<td>Afterload Pressure (mmHg)</td>
<td>130/ 75 (85)</td>
<td>25/ 9 (15)</td>
</tr>
</tbody>
</table>

**Exercise**

<table>
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<tr>
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<th>Left Ventricle</th>
<th>Right Ventricle</th>
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</thead>
<tbody>
<tr>
<td>Cardiac Output (L/min)</td>
<td>25</td>
<td>25</td>
</tr>
<tr>
<td>Vascular resistance (dyne-sec.cm$^5$)</td>
<td>↓↓↓↓</td>
<td>↓</td>
</tr>
<tr>
<td>Afterload Pressure (mmHg)</td>
<td>↑</td>
<td>↑↑↑</td>
</tr>
</tbody>
</table>
It’s all about hoses
In a pulsatile circulation we need to consider compliance.

- To maximise *pulsatile* flow you want big vessels that can distend.
- Flow \( \propto P \times r^4 \div \eta \times L \)

Lankhaar et al. *EHJ* 2008

Tedford, Kass et al. *Circ* 2011
Gelofusine Bubbles

BUBBLES = 21.2 ± 6.2 µ

CAPILLARIES = 6 – 8 µ
and larger calibre vessels result in lower pressures and resistance and higher flows
Pulmonary transit of contrast (PTAC)

- 40 athletes and 15 non-athletes
- Graded PTAC
- Equal proportion of PTAC

La Gerche, Voigt, Heidbuchel, Prior et al. *J App Physiol* 2010
Lower pulmonary pressures and resistance

La Gerche, Voigt, Heidbuchel, Prior et al. *J App Physiol* 2010
PTAC predicts performance

- PTAC was associated with a 16% greater peak exercise CO

- PTAC was an independent predictor of VO\textsubscript{2} max and maximal exercise output
### Better RV function associated with PTAC

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>p-value</th>
<th>Peak-Exercise</th>
<th>p-value</th>
<th>Interaction with exercise*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>High-PTAC</td>
<td>Low-PTAC</td>
<td>Baseline</td>
<td>High-PTAC</td>
<td>Low-PTAC</td>
</tr>
<tr>
<td><strong>RV Function</strong></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>RV Sm (cm/s)</td>
<td>11.2±2.4</td>
<td>11.1±1.4</td>
<td>0.72</td>
<td>21.5±4.5†</td>
<td>18.9±2.9†</td>
</tr>
<tr>
<td>RV Em (cm/s)</td>
<td>11.3±3.4</td>
<td>10.9±2.1</td>
<td>0.98</td>
<td>33.6±6.7†</td>
<td>30.4±6.8†</td>
</tr>
<tr>
<td>RV IVA (cm/s²)</td>
<td>1.7±0.7</td>
<td>1.7±0.8</td>
<td>0.83</td>
<td>6.9±2.7†</td>
<td>5.1±1.9†</td>
</tr>
<tr>
<td><strong>LV function</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV Sm (cm/s)</td>
<td>6.4±1.6</td>
<td>6.3±1.2</td>
<td>0.64</td>
<td>13.5±3.2†</td>
<td>12.3±2.5†</td>
</tr>
<tr>
<td>LV Em (cm/s)</td>
<td>9.0±1.8</td>
<td>8.7±1.9</td>
<td>0.63</td>
<td>19.6±3.7†</td>
<td>17.6±4.0†</td>
</tr>
<tr>
<td>LV IVA (cm/s²)</td>
<td>1.4±1.4</td>
<td>1.3±1.0</td>
<td>0.75</td>
<td>3.4±1.1†</td>
<td>2.9±1.5†</td>
</tr>
</tbody>
</table>

La Gerche, Voigt, Heidbuchel, Prior et al. *J App Physiol* 2010
Attenuated BNP increase in high PTAC

La Gerche et al. J App Physiol 2010
Trans-pulmonary bubbles

- Are associated with enhanced pulmonary vessel hemodynamics, RV function and exercise capacity
- May be a surrogate measure of pulmonary vessel size and compliance
- ??? Early marker of pulmonary vessel pathology

Lankhaar et al. *EHJ* 2008
So, RV function during exercise may be important but how can I measure it?
RV Stress test

RV areas

PASP estimates

RV velocities, strain/ rate

La Gerche, D’Hooge, Heidbuchel, Prior et al. JASE 2012
Deformation during exercise
RV strain rate increases with exercise

La Gerche, D’Hooge, Heidbuchel, Prior et al. JASE 2012
Two non-invasive surrogates singing the same tune?

La Gerche, D’Hooge, Heidbuchel, Prior et al. JASE 2012
Translational exercise science - from normal to pathology
Failure of RV function limits exercise cardiac output in PHTn

Holverda, Vonk Noordegraaf et al. *Heart* 2009
Elite cyclist with ? subtle ARVC
28 yo lady with recently diagnosed ASD
ASD exercise
Increased pulmonary vascular resistance in unrepaired ASD’s
Conclusions

• Recognise the limitations of assessing the RV at rest
• Exercise places an important (and possibly disproportionate) load on the RV
• RV/ pulmonary vascular function can limit exercise performance – in disease and health
• ‘RV reserve’ may be useful in predicting functional decline and prognosis
Thank you
How can big bubbles get through a little tube?

- Bubbles break up
- AV shunts open
- Pulmonary vessels distend
AV Shunts ??

- SaO$_2$ and PaO$_2$ decreased in athletes but there was no relationship between exercise induced hypoxemia and PTAC.
- PTAC is unlikely to represent shunting (alone).
Distensibility ??

- Pulmonary capillary distensibility has been demonstrated in rapid freeze animal models (Sobin 1972, Glazier 1969)

- Reeves compared a theoretical schema with human data and determined that:
  
pulmonary vessels distend 2% for every 1mmHg increase in mPASP
Increased afterload

- Pulmonary hypertension, pulmonary arterial hypertension etc.
- Need to consider both deformation and load