The right heart: the Cinderella of heart failure

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Disclosure

none
Look into the Heart Failure ESC guidelines

ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012

The Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2012 of the European Society of Cardiology.

Developed in collaboration with the Heart Failure Association (HFA) of the ESC

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26 authors
61 pages
Right heart (RV) in the HF ESC Guidelines

1. RV & LV interaction – cause of limited cardiac reserve

2. RVF – other cause of elevated BNP / NT-proBNP (diagnosis)

3. Echocardiography – information about RV function / RA pressure

4. AVRC – imaging techniques

5. RV failure as a potential cause of renal dysfunction

6. RV infarction – cause of acute deterioration / AHF

7. Adult congenital heart disease

8. MCS – need for RV function assessment; deteriorating RV function as an indication for VAD implantation

9. Isolated RV failure
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12.7.2 Isolated right ventricular failure

New-onset isolated right ventricular failure may occur secondarily to an acute coronary syndrome (and is managed as described above) and following massive pulmonary embolism (see pulmonary embolism guidelines\textsuperscript{239}). In both situations, diuretics and vasodilators should be used cautiously or avoided so as not to reduce right ventricular filling.

Progressive isolated right ventricular failure may occur in patients with pulmonary hypertension. Type V phosphodiesterase inhibitors, endothelin antagonists, and prostacyclin analogues may help by decreasing pulmonary arterial resistance (see guidelines\textsuperscript{240}).

9. Isolated RV failure
The normal right ventricle

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>RV</th>
<th>LV</th>
</tr>
</thead>
<tbody>
<tr>
<td>End Diastolic Volume (ml)</td>
<td>75±13 (49-101)</td>
<td>66±12 (44-89)</td>
</tr>
<tr>
<td>Mass (g/m²)</td>
<td>26±5 (17-34)</td>
<td>87±12 (64-109)</td>
</tr>
<tr>
<td>Wall thickness (mm)</td>
<td>2 – 5</td>
<td>7 – 11</td>
</tr>
<tr>
<td>Ejection Fraction (%)</td>
<td>61±7 (47-76)</td>
<td>67±5 (57-78)</td>
</tr>
<tr>
<td>Vascular pressure (mmHg)</td>
<td>25/4 [(15-30)/(1-7)]</td>
<td>130/8 [(90-140)/(5-12)]</td>
</tr>
<tr>
<td>Vascular Resistance (dyne·s·cm⁻⁵)</td>
<td>70 (20-130)</td>
<td>1100 (700-1600)</td>
</tr>
</tbody>
</table>

The response of the RV and LV to experimental increase in afterload

Comparison of normal LV and RV structure and function

RV: “the take home message”

the main aim of the RV is to keep RVEDP or RAP as low as possible to optimize the venous return to provide sustained low-pressure perfusion through the lungs
Right ventricle

• generates **flow**

• pulmonary vessels:
  – low resistances, transpulmonary gradient = 5mmHg
  – great distensibility
  – no changes in compliance throughout the lungs
  – no reflection
Special Report

Right Ventricular Function and Failure
Report of a National Heart, Lung, and Blood Institute Working Group on Cellular and Molecular Mechanisms of Right Heart Failure

Norbert F. Voelkel, MD; Robert A. Quaife, MD; Leslie A. Leinwand, PhD; Robyn J. Barst, MD; Michael D. McGoon, MD; Daniel R. Meldrum, MD; Jocelyn Dupuis, MD, PhD; Carlin S. Long, MD; Lewis J. Rubin, MD; Frank W. Smart, MD; Yuichiro J. Suzuki, PhD; Mark Gladwin, MD; Elizabeth M. Denholm, PhD; Dorothy B. Gail, PhD

Contemporary Reviews in Cardiovascular Medicine

Right Ventricular Function in Cardiovascular Disease, Part II
Pathophysiology, Clinical Importance, and Management of Right Ventricular Failure

François Haddad, MD; Ramona Doyle, MD; Daniel J. Murphy, MD; Sharon A. Hunt, MD

Circulation 2006;114:1883-91

Right ventricular failure

RV failure is a complex clinical syndrome that can result from any structural or functional cardiovascular disorder that impairs the ability of the RV to fill or to eject blood.

The cardinal clinical manifestations of RV failure are:
(1) fluid retention, which may lead to peripheral edema, ascites, and anasarca;
(2) decreased systolic reserve or low cardiac output, which may lead to exercise intolerance and fatigue;
(3) atrial or ventricular arrhythmias

RV and LV are interlinked:

- by a shared wall (the septum)
- by mutually encircling epicardial fibers
- by attachment of the RV free wall to the anterior and posterior septum
- by sharing the pericardial space

Voelkel NF et al. *Circulation* 2006;114:1883-91

Right ventricular failure

Mechanisms & Specific Causes

1. **Pressure overload**
   - left-sided HF (most common cause)
   - other causes of pulmonary hypertension
   
2. **Volume overload**

3. **Ischaemia and infarction**

4. **Intrinsic myocardial process**
   - cardiomyopathy and heart failure

5. **Inflow limitation**

6. **Complex congenital defects**

7. **Pericardial disease**

STARLINGS LAW

• RV is subject to the same haemodynamic mechanisms as the left: as the right heart fails, so a higher filling pressure is required to maintain right heart output. In health, the CVP is around zero.

• RV -very sensitive to changes in afterload, more compliant than the LV.
  
  **accomodates volume overload well**, with only small increases in systemic venous pressure

  **tolerates pressure overload poorly** (hence R heart failure in L heart failure or pulmonary vascular pathology)
Auto-aggravation of CO decrease in ARVF

Right Ventricular Failure

Reduction in CO + RV dilatation + reduction in LV preload

tricuspid regurgitation

hypotension

decrease in RCPP

organ's hypoperfusion + congestion (acidosis, ...)

greater reduction in CO

Dilated Right Ventricle!
The Syndrome of “Right Heart Failure”

- Characterised by Peripheral Oedema
  - Ankle, sacral oedema
  - Ascites
- Anasarca-ανασαρα
- Genuine Fluid overload
  - At least 5 litres for ankle oedema
  - Often, 20 litres or more
- Gradual
  - Key stage, neurohormonal response to poor renal perfusion
  - Increase venous hydrostatic pressure
  - Starling forces
  - Fluid loss from vessels into tissues
The Syndrome of “Right Heart Failure”

Clinical signs

- Ankle oedema
  - Legs
  - Abdominal wall
- Pleural effusions
- Ascites
- Pericardial effusions
- Sinus tachycardia or AF,
- low SBP, ↑JVP, TR, cardiomegaly, S3
  - Clear lungs or some pulmonary oedema
Better understanding of Acute Heart Failure pathophysiology

**MORTALITY**

- Myocardial injury (*Tn release*)
- Renal dysfunction (CRS)
- Liver dysfunction

**PREVENTION OF END-ORGAN DAMAGE**

**Congestion**

- Viable but dysfunctional myocardium

**Mechanisms which can be targeted**

- Neurohormonal & inflammatory activation
- Metabolic factors
- Vascular resistance/stiffness

**Hemodynamic deterioration**

(↑LVFP, ↓CO, ↓perfusion)
Venous congestion as relevant hemodynamic factor underlying WRF in acute heart failure

ROC for CVP and CI on Admission for the Development of WRF

Relative Contributions of CVP and CI to GFR at Time of PAC Removal

Clinical characteristics of pts with liver dysfunction

### Clinical signs at baseline

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Abnormal</th>
<th>p-value</th>
<th>Normal</th>
<th>Abnormal</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP (mmHg)</td>
<td>117</td>
<td>114</td>
<td>0.013</td>
<td>117</td>
<td>114</td>
<td>0.012</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>71</td>
<td>69</td>
<td>0.073</td>
<td>70</td>
<td>71</td>
<td>NS</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>83</td>
<td>83</td>
<td>NS</td>
<td>81</td>
<td>87</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Peripheral edema</td>
<td>65.8</td>
<td>79.3</td>
<td>&lt;0.001</td>
<td>70.0</td>
<td>63.7</td>
<td>0.023</td>
</tr>
<tr>
<td>Ascites (%)</td>
<td>16.9</td>
<td>31.0</td>
<td>&lt;0.001</td>
<td>22.0</td>
<td>17.1</td>
<td>0.049</td>
</tr>
<tr>
<td>Cold extremities</td>
<td>20.8</td>
<td>26.1</td>
<td>0.076</td>
<td>19.6</td>
<td>25.5</td>
<td>0.022</td>
</tr>
</tbody>
</table>

### Biological parameters at baseline

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Abnormal</th>
</tr>
</thead>
<tbody>
<tr>
<td>BNP (pg/mL)</td>
<td>1465.1</td>
<td>2250.9</td>
</tr>
</tbody>
</table>

### Initial hospitalization characteristics (%)

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Abnormal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute MI</td>
<td>19.0</td>
<td>10.7</td>
</tr>
<tr>
<td>LVEF</td>
<td>24.0</td>
<td>23.3</td>
</tr>
<tr>
<td>Tricuspid regurgitation</td>
<td>45.8</td>
<td>52.9</td>
</tr>
</tbody>
</table>

### All-cause mortality (%)

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Abnormal</th>
</tr>
</thead>
<tbody>
<tr>
<td>at 31 d</td>
<td>11.1</td>
<td>14.6</td>
</tr>
<tr>
<td>at 180 d</td>
<td>23.5</td>
<td>34.9</td>
</tr>
</tbody>
</table>

Nikolaou et al, Eur Heart J (in press)
Cardio-hepatic syndromes

- **Abnormal transaminases:**
  - systemic and peripheral **hypoperfusion**
  - « Forward » HF

- **Abnormal Alkaline Phosphatase:**
  - clinical and biological signs of marked systemic - mostly right-sided- **congestion**
  - Varies with BNP changes
  - « backward » HF, marker of « congested liver ».

*Nikolaou et al, Eur Heart J (in press)*
Markers of RV Dysfunction Associated With Clinical Status and Prognosis

Right ventricular ejection fraction (echocardiography, radionuclide angiography or thermodilution) 28,29,31,33,35-38,77
Right ventricular ejection fraction response to pulmonary vasodilation 65
Right ventricular dilation 78
Degree of right ventricular dilation compared with left ventricular dilation 32
Tricuspid annular velocity (systolic and/or diastolic) or excursion, or echo right ventricular descent (shortening) 30,34,79-81
Right ventricular index of myocardial performance 80,82,83
Doppler-estimated dP/dt 84
Tricuspid regurgitation 85-87
Doppler echo-derived right ventricular tissue displacement and strain 88
Right atrial size 85,89
Radionuclide angiographic, invasive angiographic, or echo/catheterization pressure-volume or pressure-area loops 90-92
Brain natriuretic peptide level 15-19
Heart rate variability 93
How can I detect a RVF in acute settings: *Biology*?

- Plasma lactate seems much higher than expected by systemic hemodynamic
- Elevated liver enzymes
- Decrease in PT
- Oliguria

*Courtesy A. Mebazaa*
How can I detect a RVF in acute settings: *Biology*?

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How to diagnose RVF in the context of advanced LVF?

- Inability to reduce congestion
- Persistent organ dysfunction
  - Renal failure
  - Liver failure

*Courtesy A. Mebazaa*
# Factors Relating to Survival in Chronic Congestive Cardiomyopathy

<table>
<thead>
<tr>
<th>Factors</th>
<th>Survivors</th>
<th>Non-Survivors</th>
<th>$p$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEF</td>
<td>18.7±6.8 %</td>
<td>18.1±10.3 %</td>
<td>NS</td>
</tr>
<tr>
<td>RVEF</td>
<td>41.4±23.0 %</td>
<td>23.9±10.2 %</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>LV dyskinesia</td>
<td>20.9±11.8 %</td>
<td>29.7±15.0 %</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Ventricular arrhythmia</td>
<td>50%</td>
<td>44%</td>
<td>NS</td>
</tr>
<tr>
<td>Functional class (NYHA)</td>
<td>3.0 ± 0.7</td>
<td>3.6 ± 0.5</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Diabetes</td>
<td>19 %</td>
<td>22 %</td>
<td>NS</td>
</tr>
<tr>
<td>Hypertension</td>
<td>19 %</td>
<td>33 %</td>
<td>NS</td>
</tr>
<tr>
<td>Previous inferior infarction</td>
<td>17 %</td>
<td>42 %</td>
<td>NS</td>
</tr>
<tr>
<td>COPD</td>
<td>6 %</td>
<td>33 %</td>
<td>NS</td>
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Polak JF et al. JACC 1983; 2: 217
In-hospital mortality in EHFS II by history of HF and clinical class

Nieminen, M. S. et al. EHJ 2006 27:2725
Adjusting for 10 most important baseline covariates, including LVEF, each 5% decrease in RVFAC was associated with a 53% increased risk of adverse outcomes (HR 1.53, 95% CI 1.24-1.88).

Anavekar et al. for the VALIANT Investigators AJC 2007
RV dysfunction/ failure: treatment

**Chronic right heart failure**

**With left heart disease**
- Chronic heart failure
- ACC/AHA guidelines
- Consider sildenafil therapy

**Predominately right heart failure**

**PAH**
- Assess cardiopulmonary risk
- Oxygen C-PAP
- Prostanoid ERB PDE5 inhibitors anticoagulation

**CPD**
- Oxygen C-PAP

**Thromboembolic disease**
- Anticoagulation
- Consider - Endarterectomy (CTED)
- Thrombolysis (acute PE)

**Systemic RV**
- Consider - ACEI or ARB
- Beta-blockade
- Resynchronization

**TOF**
- PVR
- TV repair or complex palliation

In refractory cases
- Transplantation
- Experimental therapies

Haddad, 2010
Treatment of LVF versus RVF

LV failure

• Often decrease BP
• NIV often needed
• Volume loading might be needed

RV failure

• Often increase Blood Pressure (Right Coronary Artery!)
• Caution if volume loading: it might aggravate RVF

Mebazaa et al. Critical Care Medicine, 2008, Suppl 36:129-139
Treatment of RVF

• What I should do
  – Decrease RV afterload: to decrease $\text{MVO}_2$
  – Decrease volemia: to decrease RVV and TR
  – Improve contractility
  – Increase MAP: to improve RC and organ PP

• What I should avoid
  – Mechanical ventilation as much as possible
  – Volume loading

Mebazaa et al. Critical Care Medicine, 2008, Suppl 36:129-139
Right Heart: Cinderella of HF

RV dysfunction / failure

• Syndrome of congestion
• High Morbidity and Mortality
• Heterogenous aetiology
• Not EBM therapy
• Much less studied than the Left!