ECHOCARDIOGRAPHIC ASSESSMENT OF RIGHT HEART

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Introduction

- For many years, the echocardiographic quantitative assessment of RV function has been difficult owing to the complex RV anatomy and consequently ignored.

- The RV has a great impact on the prognosis of patients with pulmonary hypertension, myocardial infarction involving the RV, and left ventricular (LV) dysfunction.

- In the second half of the past century, after recognizing its key role in various physiological and pathological conditions, the RV regained attention.
Anatomy and physiology of the right ventricle

- **Echocardiographic assessment of the RV is complicated by the complex geometry of this chamber, the pronounced trabeculation that compromises accurate endocardial delineation, and the anterior position that often limits echo image quality.**

- This **complex geometry** cannot be fitted to simple geometric models, which presents important limitations for the estimation of RV volume and function based on two-dimensional (2D) tomographic views.

- Three anatomical parts of the RV can be distinguished: the **inlet part** which accommodates the tricuspid valve, the **trabeculated apical part**, and the **outlet part**.

- Myocytes are predominantly oriented in the **longitudinal direction** in the subendocardial layer. Circumferentially oriented myocytes are found in the thinner subepicardium. Consequently, the RV contraction pattern is predominantly longitudinal.
Pathophysiology of the right ventricle

1. RV Intrinsic contractile dysfunction
   - RV myocardial ischemia or infarction is the major primary cause of contractile dysfunction
   - Arrhythmogenic RV cardiomyopathy
Pathophysiology of the right ventricle

1. RV Intrinsic contractile dysfunction
   - RV myocardial ischemia or infarction is the major primary cause of contractile dysfunction
   - Arrhythmogenic RV cardiomyopathy

2. Change in loading conditions beyond physiological limits
   - An acute change in afterload of sufficient magnitude, as produced by pulmonary embolism (PE), can quickly result in RV failure as the RV has little ability to cope with this condition
   - A chronic exposure to an increased afterload results in RV hypertrophy and altered geometry, which temporarily reduces wall stress but ultimately results in RV failure
   - RV volume overload
Pathophysiology of the right ventricle

1. Right Ventricular Intrinsic Contractile Dysfunction
   - Right ventricular myocardial ischemia or infarction is the major primary cause of contractile dysfunction
   - Arrhythmogenic Right Ventricular Cardiomyopathy

2. Change in Loading Conditions Beyond Physiological Limits
   - An acute change in afterload of sufficient magnitude, as produced by pulmonary embolism (PE), can quickly result in Right Ventricular failure as the RV has little ability to cope with this condition
   - A chronic exposure to an increased afterload results in RV hypertrophy and altered geometry, which temporarily reduces wall stress but ultimately results in RV failure
   - Right Ventricular Volume Overload

3. Altered Performance of the LV can all have detrimental effects on Right Ventricular function
The assessment of Right ventricular morphology
Measurement of right ventricular outflow tract diameter at subpulmonary region ($RVOT_1$) and pulmonic valve annulus ($RVOT_2$) and main pulmonary artery from parasternal short-axis view.
Reference limits and partition values of right ventricular and pulmonary artery size

<table>
<thead>
<tr>
<th>Severe</th>
<th>Reference range</th>
<th>Mild</th>
<th>Moderate</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Right ventricular /RV/mm:</strong></td>
<td></td>
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<tr>
<td>Basal RV diameter (RV D 1)</td>
<td>20–28</td>
<td>29–33</td>
<td>34–38</td>
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<tr>
<td>Mid-RV diameter (RV D 2)</td>
<td>27–33</td>
<td>34–37</td>
<td>38–41</td>
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<td>80–85</td>
<td>86–91</td>
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<tr>
<td><strong>Right ventricular outflow tract /RVOT/mm:</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Above aortic valve (RVOT 1)</td>
<td>25–29</td>
<td>30–32</td>
<td>33–35</td>
</tr>
<tr>
<td>Above pulmonic valve (RVOT 2)</td>
<td>17–23</td>
<td>24–27</td>
<td>28–31</td>
</tr>
<tr>
<td><strong>Pulmonary artery /PA/mm:</strong></td>
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The assessment of Right ventricular morphology

- In an **apical four-chamber view**, both the long- and short-axis **diameters** can be measured and the end-systolic and end-diastolic **areas** can be determined.

- Identify features /**kinetics**/ which suggest a particular etiology, such as RV infarct or ARVC.

- Normal **thickness of the RV free wall** is 5 mm, above which the ventricle is considered to be hypertrophied.

- For **RV mass quantification**, real-time 3D echocardiography (RT3DE) is superior to 2D Echo.
The assessment of Right ventricular morphology

- When the RV is overloaded, the **crescent shape** is lost and the septum becomes **flat**, the LV taking the shape of the **letter ‘D’**, resulting in an impaired LV filling and a decrease in cardiac output.

- In conditions characterized by RV **volume overload**, the flattening of the septum is seen only in diastole, the septum regaining its normal shape in systole.

- When the RV is subjected to a **pressure overload**, the septum will move towards the RV in systole in a first stage, maintaining the altered shape during the entire cardiac cycle.
(A and B) - Patient with RV volume overload - ASD type ostium secundum.

(C and D) - Patient with severe pulmonary arterial hypertension and consequent severe right ventricular pressure overload—flattening of the interventricular septum both at end diastole (C) and end systole (arrow) (D)
Methods of measuring right ventricular wall thickness (arrows) from M-mode (left) and subcostal transthoracic (right) echocardiograms. 5mm is borderline for RV hypertrophy.
Assessment of right ventricular function

Classical methods:

- Tricuspid annular plane systolic excursion (TAPSE)
- Myocardial performance index (MPI, Tei index)
- RV outflow tract shortening fraction (RVOT-SF)
- RV fractional area change
Assessment of right ventricular function

Tricuspid annular plane systolic excursion - TAPSE

Measurements of TAPSE in a normal individual ~24 mm (A) and in a patient with pulmonary hypertension ~9 mm (B).
Assessment of right ventricular function

Tricuspid annular plane systolic excursion (TAPSE)

- Tricuspid annular plane systolic excursion (TAPSE) has proved a useful index for evaluating RV longitudinal function.
- It has been shown to have a good correlation with isotopic derived RVEF, although A navekar et al. failed to find any correlation between TAPSE and MRI-derived ejection fraction.
- Normal values for TAPSE are 20 mm ±3. The prognostic value of TAPSE was emphasized in cardiac failure and myocardial infarction. TAPSE ≤15 mm was associated with increased mortality (45% at 2 years) compared with patients having TAPSE >20 mm (4%).
- TAPSE has some inherent limitations mostly because assessment is restricted to the longitudinal function of the RV free wall, disregarding the contribution of the interventricular septum and the RVOT.
Assessment of right ventricular function

- **Methods of determining indices of right ventricular systolic function.**

- **(A) RV outflow tract shortening fraction**
  \[ \text{RV OTSF} \% = \frac{\text{EDRVOTD} - \text{ESRVOTD}}{\text{EDRVOTD}} \]
  \[ \text{N} \sim 61\% \pm 13\% \]

- **(B) RV Fractional area change**
  \[ \text{RVFAC} \% = 100 \times \frac{\text{RVEDA} - \text{RVESA}}{\text{RVEDA}} \]
  \[ \text{N} \sim 56\% \pm 13\% \]
Myocardial performance index (MPI, Tei index)

Normal values for RV MPI are 0.28 ± 0.04, it increases with RV dysfunction

Pseudonormalization of the right ventricular Tei index
Novel methods

Tissue Doppler imaging (TDI)

- Tricuspid ring systolic velocities - cut-off value of <11.5 cm/s for RV dysfunction (defined as RV Ejection Fraction <45%)
- IVRT
- Tei index
- Isovolumic myocardial acceleration
- Strain (S)/strain rate (SR)

Speckle-tracking techniques

- Strain (S)/strain rate (SR)
Pulsed-wave TDI at the tricuspid level of the right ventricular free wall in a normal individual (A) and in a patient with pulmonary hypertension (B).
Measuring isovolumic acceleration (IVA) during isovolumic contraction at the basal segment of the right ventricular free wall.

IVA measured in the basal segment of the RV free wall of >1.1 m/s² correlates well with MRI RVEF >45% (90% sensitivity and specificity)
Strain measurements in the RV free wall of a normal individual using Tissue Doppler techniques (A) and Speckle-tracking techniques (B).
Strain rate of the RV free wall in a pulmonary hypertension patient using Tissue Doppler techniques (A) and Speckle-tracking techniques (B).
Three-dimensional echocardiography has emerged as the non-invasive technique that would overcome the geometric limitations of standard 2D Echo.

Volumes and, consequently, ejection fraction should be determined with high accuracy and without any geometrical assumptions.

In a recent study, Niemann et al. found an excellent correlation between 3D Echo- and MRI-derived RVEF.

Further studies are needed to assess the technical aspects of acquiring data sets and to determine the range of RV volumes and RVEF in health and pathology.
Three-dimensional imaging of the right ventricle with measurements of the right ventricular volumes and ejection fraction in a healthy individual.
Right ventricular diastolic function

- **Conventional Doppler** – *Tricuspid inflow* pattern in a four-chamber view. There are marked inspiratory variations/

- The ratio between the tricuspid flow early diastolic velocity ($E$) *conventional Doppler* and peak early diastolic velocity of the lateral tricuspid annulus ($E'$) *TDI* /

- $E/E'$ ratio was found to have a good correlation with mean invasively measured RA pressure ($r = 0.75; P < 0.001$)

- An $E/E' > 6$ had a sensitivity of 79% and a specificity of 73% to detect an RA pressure $>10$ mm Hg

- A trial/total TAPDE diastolic RV function was defined as the ratio of the amplitude of tricuspid annular plane
Normal respiratory variations of the tricuspid inflow velocities.
Pulmonary hypertension

- PH is defined as a mean pulmonary arterial pressure that is greater than 25 mm Hg at rest or greater than 30 mm Hg during exercise along with a pulmonary capillary wedge pressure or left atrial pressure less than 15 mm Hg, measured at cardiac catheterization.

- Noninvasively, based on echocardiography, PH is defined as a measured Doppler tricuspid regurgitation velocity of greater than 2.8 m/sec.
Schematic representation of M-mode of normal and abnormal pulmonary valves and M-mode echocardiograms recorded in patient with pulmonary hypertension.
The PASP is measured using the simplified Bernoulli equation applied to peak TR velocity by CW Doppler. To this value, RAP should be added to complete the calculation: \( \text{PASP} = 4(\text{TRV})^2 + \text{RAP} \)
### Estimation of Right Atrial Pressures by Echocardiography

**Based on inferior vena cava measurements using subcostal window**

<table>
<thead>
<tr>
<th>Normal</th>
<th>IVC - mm</th>
<th>IVC - inspiratory collapse</th>
<th>Corresponding right atrial pressures (mmHg)</th>
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<tbody>
<tr>
<td>&lt;17</td>
<td>&gt;50% collapse</td>
<td>&lt;5</td>
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<td>15 – 20 Not dilated</td>
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<td>17 – 22</td>
<td>&lt;50% collapse</td>
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<th>Severe</th>
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<tr>
<td>&gt;20 dilated &gt;25</td>
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<td>15 – 20 &gt;20</td>
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<td>&lt;12</td>
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<th>Athletes</th>
<th>IVC - mm</th>
<th>IVC - inspiratory collapse</th>
<th>Corresponding right atrial pressures (mmHg)</th>
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<tbody>
<tr>
<td>22 – 24</td>
<td>Normal collapse</td>
<td>N</td>
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Estimation of Right Atrial Pressures by Echocardiography
Based on inferior vena cava measurements using sub costal window

A: Recorded in a normal patient. Note the respiration-dependent phasic variation in IVC size

B: Normal IVC size but a loss of respiratory variation is shown

C: Dilated IVC also without respiratory variation
Estimate MPAP from the RVOT acceleration time (AcT):
Mahan's equation - MPAP = 79 - 0.45 (AcT) The acceleration phase becomes shorter with increased pulmonary artery pressure.
Spectral flow profiles recorded in a normal individual (A) with an acceleration time (AT) of 190 ms and a patient with significant pulmonary hypertension in whom the acceleration time is 80 ms (B).
Pulmonary hypertension and Cor pulmonale

- The right ventricle is a low-pressure pump that accommodates well to changes in volume but not pressure.

- The increased afterload to the right ventricle in PH initially results in the normal adaptive response of dilatation and hypertrophy.

- Eventually, the right ventricular contractile function declines when it is unable to further respond and tolerate this hemodynamic burden.

- Cor pulmonale is defined as right ventricular dysfunction and failure that directly results from PH associated with chronic lung disease.
# Diagnostic Classification of Pulmonary Hypertension

## 1. Pulmonary arterial hypertension

1.1 Idiopathic Familial

1.2 Related to

   (a) Connective tissue disease

   (b) Congenital systemic to pulmonary shunts

   (c) Portal hypertension

   (d) HIV infection

   (e) Drugs/toxins

      (1) Anorexigens

      (2) Cocaine

## 2. Pulmonary venous hypertension

2.1 Left-side atrial or ventricular heart disease

2.2 Left-side valvular heart disease

2.3 Extrinsic compression of central pulmonary veins

   (a) Fibrosing mediastinitis

   (b) Adenopathy/tumors

2.4 Pulmonary venoocclusive disease/pulmonary capillary hemangiomatosis

## 3. Pulmonary hypertension associated with disorders of the respiratory system and/or hypoxemia

3.1 Chronic obstructive pulmonary disease

3.2 Interstitial lung disease

3.3 Sleep-disordered breathing

3.4 Alveolar hypoventilatory disorders

3.5 Chronic exposure to high altitude

3.6 Neonatal lung disease

3.7 Alveolar-capillary dysplasia

## 4. Pulmonary hypertension caused by chronic thrombotic and/or embolic disease

4.1 Thromboembolic obstruction of proximal pulmonary arteries

4.2 Obstruction of distal pulmonary arteries

   (a) Pulmonary embolism (thrombus, tumor, ova and/or parasites, foreign material)

   (b) In situ thrombosis

## 5. Pulmonary hypertension as a consequence of disorders directly affecting the pulmonary vasculature

5.1 Inflammatory

   (a) Schistosomiasis

   (b) Sarcoidosis
Pulmonary embolism

Investigations:

- Arterial blood gas
- D-dimer
- Electrocardiography
- Chest X-ray
- V/Q scan
- Spiral CT-scan
- Pulmonary Angiography
- Echocardiography
- Doppler study for deep vein thrombosis
Echocardiographic Signs of Pulmonary Embolism

- **RV dilatation with RV hypertrophy** - RV end diastolic pressure >34 mm, ratio of RV/LV >1 in apical 4 chamber view was the most accurate predictor for PE, with a sensitivity and specificity of 66% and 77%.

- **RV dysfunction** - TAPSE was decreased in PE compared to healthy subjects (19 ± 5 vs. 26 ± 4 mm, P < 0.001), with greater reduction in patients with increased, compared to normal, RV pressure (16.6 ± 5 vs. 20.5 ± 5 mm, P < 0.05).
Echocardiographic Signs of Pulmonary Embolism

- **RV dilatation** without RV hypertrophy - RV end diastolic -RV D2 > 34 mm, ratio of RV/LV > 1 in apical 4 chamber view was the most accurate predictor for PE, with a sensitivity and specificity of 66% and 77%

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- The "McConnell sign" - defined as RV free wall hypokinesis or akinesis plus normokinesia or hyperkinesia of the RV apex - specificity for PE at 96%, but poor sensitivity, at just 16%

- Interventricular septal flattening and paradoxical motion toward the LV, resulting in a “D-shaped” LV in cross section
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- **Tricuspid regurgitation** - Pulmonary hypertension with a tricuspid regurgitant jet velocity $> 2.8\,m/sec$.

- **The 60/60 sign** - acceleration time of RV ejection $< 60\,ms$ in the presence of tricuspid insufficiency pressure gradient $< 60\,mm\,Hg$. 
Echocardiographic Signs of Pulmonary Embolism

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- **Tricuspid regurgitation** - Pulmonary hypertension with a tricuspid regurgitant jet velocity >2.8 m/sec

- **The 60/60 sign** - acceleration time of RV ejection <60 ms in the presence of tricuspid insufficiency pressure gradient <60 mmHg

- **Loss of inspiratory collapse of the inferior vena cava**

- **PA dilatation**

- **Direct visualization of thrombus** (more likely with TEE)
Interventricular septal flattening and paradoxical motion

Parasternal short-axis views illustrating measurements of the eccentricity index (EccIx)

(A) Normal heart with diastolic EccIx = 1

(B) Patient with pulmonary hypertension with diastolic EccIx = 1.8
TEE detected central pulmonary thromboemboli, mostly in the right pulmonary artery

Transthoracic echocardiogram of pulmonary artery thrombus
Parasternal short axis (PSAX) view at the aortic valve level shows echo-dense structure at the bifurcation of the main pulmonary artery, consistent with a pulmonary saddle embolus.
Diagnostic value of three sets of echocardiographic signs suggesting the presence of acute PE in subgroups with and without known previous cardiorespiratory diseases

<table>
<thead>
<tr>
<th>Patients without known previous cardiorespiratory diseases (n 46)</th>
<th>Patients with known previous cardiorespiratory diseases (n 54)</th>
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<tbody>
<tr>
<td>R V overload criteria</td>
<td>60/60 sign</td>
</tr>
<tr>
<td>Specificity (%)</td>
<td>78</td>
</tr>
<tr>
<td>Sensitivity (%)</td>
<td>81</td>
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<tr>
<td>PPV (%)</td>
<td>90</td>
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<tr>
<td>NPV (%)</td>
<td>64</td>
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RV overload criteria

The presence of 1 of four signs:

- right-sided cardiac thrombus
- RV diastolic dimension (parasternal view) >30 mm or a RV/LV ratio >1
- systolic flattening of the interventricular septum;
- acceleration time <90 ms
- tricuspid insufficiency pressure gradient >30 mm Hg in absence of RV hypertrophy
Suspected non-high-risk PE
i.e. without shock or hypotension

Assess clinical probability of PE
Implicit or prediction rule

Low/intermediate clinical probability
or "PE unlikely"

D-dimer

Negative
- No treatment*
- No PE‡

Positive
- Multidetector CT
- PE†

High clinical probability
or "PE likely"

Multidetector CT

PE†
- Treatment*

No PE
- No treatment* or investigate further*

No treatment*
Echocardiographic examination is not 
recommended as an element of 
elective diagnostic strategy in 
haemodynamically stable, 
normotensive patients with suspected 
PE
In patients with suspected high-risk PE presenting with shock or hypotension, the absence of echocardiographic signs of RV overload or dysfunction practically excludes PE as a cause of haemodynamic instability.
Pulmonary embolism

Echocardiography

- For the diagnosis of PE, echocardiography should not be used.
- Obstruction of >50% of the pulmonary vascular bed in PE is required to see significant elevation of the PA pressures.
- The normal RV is unable to generate PASP > 50 mm Hg (mean 40) acutely. Acute PE cannot explain a PASP > 50 mm Hg.
- Moreover, **80% of patients with shock and PE show evidence of right-heart dysfunction on echocardiography**.
- Echocardiography has been recommended as a tool for risk stratification in PE.
- Exclude other causes of shock:
  - acute left ventricular dysfunction
  - tamponade
  - acute valvular disease
  - aortic dissection
Prognostic markers in PE

- Hypotension
- RV dysfunction
- Elevated BNP
- Elevated troponins
- Presence of RV clot
DDx of A PE

Must Rule Out Other Potentially Life-Threatening Disorders

- A MI
- Pericardial Tamponade
- Aortic Dissection
- Fulminant Pneumonia

- H & P
- CXR
- ECG
- Echocardiogram
Problems with Echocardiography

- Operator dependent
- Only able to visualize thrombus in PA 0 –19% (more likely with TEE)
- Left PA distal to left main bronchus not examined
- Specificity of isolated RV dilatation is low (COPD, RV infarct, Cardiomyopathy, Valvular heart disease, Cardiac sarcoidosis, technical error)
- Low utility for TEE in critically-ill patients
Conclusions

- **Echocardiography**, being **non-invasive**, **widely available**, **relatively inexpensive**, and having **no side effects**, is the modality of choice for the assessment of morphology and function of the RV in clinical practice.

- Identifying an accurate and reliable echocardiographic parameter for the functional **assessment of the RV** still remains a challenge.

- **Combinations of parameters** are used in daily clinical practice, each one offering only partial information about the status of the RV.

- There is hope that **novel myocardial deformation parameters** and **3D Echo-derived parameters** may add value to the examination of the RV, but validation **studies** are still needed.