Right Heart Catheterization

8ο Συνέδριο Επεμβατικής Καρδιολογίας και Ηλεκτροφυσιολογίας
Καρδιολογική Εταιρεία Βορείου Ελλάδος
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ουδεμια
1. Definition - Indications - Contraindications - Technical - Possible sources of error

2. Complications

3. Principles of hemodynamics - Pressure Waveforms

4. Application in Pulmonary Arterial Hypertension

5. Interpretation measurement - results

6. Conclusions
Definition

- Promoting catheters in the right heart chambers and branches of the pulmonary artery in order to:

1. Record pressures and their waveforms
2. Measure cardiac output and the vascular reseasants
3. Evaluate O2 saturation and content — oxymetry run.
4. Angiographies
5. Interventional procedures
Indications

• **Diagnostic**
  • Differentiation of various etiologies of shock and pulmonary edema
  • Evaluation of pulmonary hypertension
  • Differentiation of pericardial tamponade from constrictive pericarditis and restrictive cardiomyopathy
  • Diagnosis of intracardiac shunts

• **Therapeutic**
  • Guide to fluid management and hemodynamic monitoring of patients after surgery, complicated myocardial infarction, patients in shock, heart failure
Contraindications

- **Absolute**
  - Endocarditis (right cavities)
  - Clot / mass in the right cavities
  - Mechanical valves in the tricuspid / pulmonary

- **Relative**
  - Haemorrhagic diathesis (INR > 2, platelet count < 50,000)
  - Poor life expectancy
  - Poor cooperation / patient reluctance
  - Bioprosthetic valves
  - Recent installation PM / ICD
  - LBBB (be possible pacing)
  - Contralateral pneumothorax
Catheter Swan-Ganz

These are 7F to 7.5F system catheters and are available as femoral vein insertion to continuous cardiac output catheters.
Technique (1)

- The introduction of catheters made with the *Seldinger technique*, comprising a vessel puncture and introduction guidewire
Technique (2)

- Venous access sites

<table>
<thead>
<tr>
<th>Site of Insertion</th>
<th>Distal to the Vena Cava/RA junction (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Internal jugular vein</td>
<td>15 to 20</td>
</tr>
<tr>
<td>Subclavian vein</td>
<td>10 to 15</td>
</tr>
<tr>
<td>Antecubital vein (Right)</td>
<td>35 to 40</td>
</tr>
<tr>
<td>Antecubital vein (Left)</td>
<td>45 to 50</td>
</tr>
<tr>
<td>Femoral vein</td>
<td>25 to 30</td>
</tr>
</tbody>
</table>

J Am Coll Cardiol 2006;48:2546–52
Technique (3)
Technique (4) — preparing the catheter
Technique (5) – inserting the catheter
Complications

- **Since probe**
  - Thrombophlebitis
  - Thrombosis catheter
  - Bacteremia-Endocarditis
  - Ruptured balloon embolization

- **Since launching**
  - Arrhythmias (PVC, NSVT, VF)
  - RBBB or PKKA
  - Valve Injury
  - Piercing PA or right ventricle of the catheter

- **Since the puncture**
  - Puncture artery
  - Pneumothorax
  - Nerve Injury / Horner’s
  - Embolization of air from the promotion
Cardiac Cycle

Right Sided Pressures

- EKG
- QRS Complex
- P wave
- T wave
- PA Pressure
- Dicrotic Notch
- Right Ventricular Pressure
- Right Atrial Pressure

Pressure (mm Hg)

- Atrial Systole
- Ventricular Systole
- Ventricular Diastole
Pressure Recordings (1)

- Always record pressure at end expiration (except in patients on PEEP)
- Under normal conditions, pressures will be lower in inspiration due to decrease in intrathoracic pressure
- Before any pressure measurements are taken, it is imperative to perform zeroing and referencing of the system
  - For every inch the heart is offset from the reference point of the transducer, a 2mm Hg of error will be introduced. If the heart is lower than the transducer, the pressure will be erroneously low and if the heart is higher, the pressure will be erroneously high
Pressure Recordings (2)

Figure 3: The phlebostatic axis, marked on the patient’s chest, is the precise anatomical point of origin of the hemodynamic pressures being measured.
Pressure Recordings (3)

- **Fast flush test/ Square wave testing**
  - The dynamic response of the pressure monitoring system is determined by measuring the resonant frequency and the damping coefficient of the system using the fast flush test.

- Performed by briefly opening and closing the valve in the continuous flush device.

- This produces a square wave pattern on the oscilloscope, an initial steep rise followed by a plateau, followed by steep fall below baseline which is then followed by oscillations. The pattern determines optimal versus suboptimal damping.
Intracardiac pressure waveforms during passage through the heart

Right atrial pressure

Right ventricular pressure

Pulmonary artery pressure

Pulmonary capillary wedge pressure

RAP = 0 - 6 mm Hg

RVP = 15 - 25
    0 - 8 mm Hg

PAP = 15 - 25
     8 - 10 mm Hg
Mean
PAP = 10 - 20 mm Hg

PCWP = 8 - 12 mm Hg

Right Heart Catheterization
RA pressure waveforms (1)

Right atrial pressure

Diastole

Ventricular systole

Diastole

a wave
Atrium contracting tricuspid valve open

x descent
Atrium relaxing then filling, tricuspid closed

v wave
Atrium tense, full; tricuspid closed

y descent
Atrium emptying, tricuspid open

RAP = 0 - 6 mm Hg
RA pressure waveforms (2)

- Effects of breathing in RAP
  - Normally:
    - **Inspiration**: ↓ intrathoracic pressure → ↓ RAP
    - **Expiration**: ↑ intrathoracic pressure → ↑ RAP
RA pressure waveforms (3)

- Low RAP
  - Hypovolemia
  - Improper zeroing of the transducer
- Elevated RAP
  - Overload volume
  - Right heart failure
  - Valvular heart disease (TS, TR, PS, PR)
  - Attack infarction (ischemia RV, cardiomyopathy)
  - No heart disease (MS, MR, AS, AR, cardiomyopathy)
  - Increased pulmonary resistance (PE, COPD, primary pulmonary hypertension)
  - Tamponade
  - Myxoma
RV pressure waveforms (1)

- RV ESP (Right Ventricular End Systolic Pressure)
- RVEDP (Right Ventricular End Diastolic Pressure)
- Atrial contraction (RV)
- Diastasis (RV)
- Rapid RV filling

Right ventricular pressure:

\[ RVP = \frac{15 - 25}{0 - 8} \text{ mm Hg} \]
RV pressure waveforms (2)

1. **Systolic pressure overload**
   1. Pulmonary Hypertension
   2. Pulmonary valve stenosis
   3. Right ventricular outflow obstruction
   4. Increased pulmonary resistance

2. **Low systolic pressure**
   1. Hypovolaemia
   2. Cardiogenic shock
   3. Tamponade

3. **End-diastolic pressure overload**
   1. Hypervolaemia
   2. Chronic heart failure
   3. Reduced even dototita - Hypertrophy
   4. Tamponade
   5. Deficiency tricuspid valve
   6. Constrictive pericarditis Decreased end-diastolic pressure

4. **Decreased end-diastolic pressure**
   1. Hypovolaemia
   2. Narrowing tricuspid valve
PA pressure waveforms (1)

Systolic

Dichrotic notch

Diastolic

Pulmonary artery pressure

PAP = 15 - 25
8 - 10 mm Hg

Mean
PAP = 10 - 20 mm Hg
PA pressure waveforms (2)

- **Increased systolic pressure**
  - Primary pulmonary hypertension
  - Pulmonary disease
  - Failure of the mitral valve (MR / MS)
  - Chronic heart failure
  - Restrictive cardiomyopathy

- **Decreased systolic pressure**
  - Hypotension
  - Pulmonary valve stenosis
  - Pulmonary artery stenosis
  - Tricuspid valve stenosis
  - Tricuspid valve atresia
  - Ebstein’s anomaly
PCWP waveforms (1)

Pulmonary capillary wedge pressure

PCWP = 8 - 12 mm Hg
PCWP waveforms (3)

- PCW tracing “approximates” actual LA tracing but is slightly delayed since pressure wave is transmitted retrograde through pulmonary veins
- Normal LA pressure slightly higher

Abnormalities in PCWP Tracing (1)

**Low mean pressure**
- Hypovolemia
- Improper zeroing of the transducer

**Elevated mean pressure**
- Intravascular volume overload
- Left ventricular failure
  - Valvular disease (MS, MR, AS, AR)
  - Myocardial disease (LV ischemia, cardiomyopathy)
  - Left heart failure secondary to HTN
- Pericardial effusion with tamponade
- Atrial myxoma
Abnormalities in PCWP Tracing (2)

• Elevated a wave
  • Mitral stenosis
  • Decreased LV compliance due to LV failure / valve disease

• Cannon a wave
  • A-V asynchrony (3rd degree AVB, VT, V-pacer)

• Elevated v wave
  • MR
  • LRV failure
  • Ventricular septal defect

• Equal a and v waves
  • Tamponade
  • Constrictive physiology
Abnormalities in PCWP Tracing (4)

*PCWP* not equal to *LV* end diastolic pressure

- Mitral stenosis
- Atrial myxoma
- Cor triatriatum
- Pulmonary venous obstruction
- Decreased ventricular compliance
- Increased pleural pressure
1. Important pathophysiological and clinical definitions
2. Haemodynamic definitions of Pulmonary Hypertension
3. Recommendations for RHC in PH
4. Recommendations for vasoreactivity testing
5. Risk assessment in PAH
6. Suggested assessment and timing for the follow-up of patients with PAH
**Important pathophysiological and clinical definitions**

1. Pulmonary hypertension (PH) is a haemodynamic and pathophysiological condition defined as an increase in mean pulmonary arterial pressure ≥25 mmHg at rest as assessed by right heart catheterization (Table 3). PH can be found in multiple clinical conditions (Table 4).

2. Pulmonary arterial hypertension (PAH, group 1) is a clinical condition characterized by the presence of pre-capillary PH (Table 3) and pulmonary vascular resistance >3 Wood units, in the absence of other causes of pre-capillary PH such as PH due to lung diseases, chronic thromboembolic PH, or other rare diseases (Table 4). PAH includes different forms that share a similar clinical picture and virtually identical pathological changes of the lung microcirculation (Table 4).

3. There is no sufficient data to support the definition of ‘PH on exercise’.
**Haemodynamic definitions of Pulmonary Hypertension**

<table>
<thead>
<tr>
<th>Definition</th>
<th>Characteristics</th>
<th>Clinical group(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PH</td>
<td>PAPm ≥25 mmHg</td>
<td>All</td>
</tr>
<tr>
<td>Pre-capillary PH</td>
<td>PAPm ≥25 mmHg, PAWP ≤15 mmHg</td>
<td>1. Pulmonary arterial hypertension 2. PH due to left heart disease 3. PH due to lung diseases 4. Chronic thromboembolic PH 5. PH with unclear and/or multifactorial mechanisms</td>
</tr>
<tr>
<td>Post-capillary PH</td>
<td>PAPm ≥25 mmHg, PAWP &gt;15 mmHg</td>
<td>2. PH due to left heart disease 5. PH with unclear and/or multifactorial mechanisms</td>
</tr>
<tr>
<td>Isolated post-capillary PH (Ipc-PH)</td>
<td>DPG &lt;7 mmHg and/or PVR ≤3 WU</td>
<td></td>
</tr>
<tr>
<td>Combined post-capillary and pre-capillary PH (Cpc-PH)</td>
<td>DPG ≥7 mmHg and/or PVR &gt;3 WU</td>
<td></td>
</tr>
</tbody>
</table>

CO = cardiac output; DPG = diastolic pressure gradient (diastolic PAP – mean PAWP); mPAP = mean pulmonary arterial pressure; PAWP = pulmonary arterial wedge pressure; PH = pulmonary hypertension; PVR = pulmonary vascular resistance; WU = Wood units.
Recommendations for RHC in PH

RHC is recommended to confirm the diagnosis of pulmonary arterial hypertension (group 1) and to support treatment decisions.

In patients with PH, it is recommended to perform RHC in expert centres (see section 12) as it is technically demanding and may be associated with serious complications.

RHC is indicated in patients with CTEPH (group 4) to confirm the diagnosis and support treatment decisions.

RHC is recommended in patients with congenital cardiac shunts to support decisions on correction (Table 24).

RHC is recommended in patients with PH due to left heart disease (group 2) or lung disease (group 3) if organ transplantation is considered.

RHC should be considered in pulmonary arterial hypertension (group 1) to assess the treatment effect of drugs (Table 16).

When measurement of PAWP is unreliable, left heart catheterization should be considered to measure LVEDP.

RHC may be considered in patients with suspected PH and left heart disease or lung disease to assist in the differential diagnosis and support treatment decisions.
**Recommendations for vasoreactivity testing**

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Grade</th>
<th>Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vasoreactivity testing is indicated only in expert centres</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Vasoreactivity testing is recommended in patients with IPAH, HPAH and PAH associated with drugs use to detect patients who can be treated with high doses of a CCB</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>A positive response to vasoreactivity testing is defined as a reduction of mean PAP ≥ 10 mmHg to reach an absolute value of mean PAP ≤ 40 mmHg with an increased or unchanged cardiac output</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Nitric oxide is recommended for performing vasoreactivity testing</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Intravenous epoprostenol is recommended for performing vasoreactivity testing as an alternative</td>
<td>I</td>
<td>C</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Additional Recommendations</th>
<th>Grade</th>
<th>Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenosine should be considered for performing vasoreactivity testing as an alternative</td>
<td>IIa</td>
<td>C</td>
</tr>
<tr>
<td>Inhaled iloprost may be considered for performing vasoreactivity testing as an alternative</td>
<td>IIb</td>
<td>C</td>
</tr>
<tr>
<td>The use of oral or intravenous CCBs in acute vasoreactivity testing is not recommended</td>
<td>III</td>
<td>C</td>
</tr>
<tr>
<td>Vasoreactivity testing to detect patients who can be safely treated with high doses of a CCB is not recommended in patients with PAH other than IPAH, HPAH and PAH associated with drugs use and is not recommended in PH groups 2, 3, 4 and 5</td>
<td>III</td>
<td>C</td>
</tr>
</tbody>
</table>
## Risk assessment in PAH

<table>
<thead>
<tr>
<th>Determinants of prognosis* (estimated 1-year mortality)</th>
<th>Low risk &lt;5%</th>
<th>Intermediate risk 5–10%</th>
<th>High risk &gt;10%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical signs of right heart failure</td>
<td>Absent</td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td>Progression of symptoms</td>
<td>No</td>
<td>Slow</td>
<td>Rapid</td>
</tr>
<tr>
<td>Syncope</td>
<td>No</td>
<td>Occasional syncope b</td>
<td>Repeated syncope c</td>
</tr>
<tr>
<td>WHO functional class</td>
<td>I, II</td>
<td>III</td>
<td>IV</td>
</tr>
<tr>
<td>6MWD</td>
<td>&gt;440 m</td>
<td>165–440 m</td>
<td>&lt;165 m</td>
</tr>
<tr>
<td>Cardiopulmonary exercise testing</td>
<td>Peak VO₂ &gt;15 ml/min/kg (&gt;65% pred.) VE/VCO₂ slope &lt;36</td>
<td>Peak VO₂ 11–15 ml/min/kg (35–65% pred.) VE/VCO₂ slope 36–44.9</td>
<td>Peak VO₂ &lt;11 ml/min/kg (&lt;35% pred.) VE/VCO₂ ≥45</td>
</tr>
<tr>
<td>NT-proBNP plasma levels</td>
<td>BNP &lt;50 ng/l NT-proBNP &lt;300 ng/ml</td>
<td>BNP 50–300 ng/l NT-proBNP 300–1400 ng/l</td>
<td>BNP &gt;300 ng/l NT-proBNP &gt;1400 ng/l</td>
</tr>
<tr>
<td>Imaging (echocardiography, CMR imaging)</td>
<td>RA area &lt;18 cm² No pericardial effusion</td>
<td>RA area 18–26 cm² No or minimal, pericardial effusion</td>
<td>RA area &gt;26 cm² Pericardial effusion</td>
</tr>
<tr>
<td>Haemodynamics</td>
<td>RAP &lt;8 mmHg CI ≥2.5 l/min/m² SvO₂ &gt;65%</td>
<td>RAP 8–14 mmHg CI 2.0–2.4 l/min/m² SvO₂ 60–65%</td>
<td>RAP &gt;14 mmHg CI &lt;2.0 l/min/m² SvO₂ &lt;60%</td>
</tr>
</tbody>
</table>

6MWD = 6-minute walking distance; BNP = brain natriuretic peptide; CI = cardiac index; CMR = cardiac magnetic resonance; NT-proBNP = N-terminal pro-brain natriuretic peptide; pred. = predicted; RA = right atrium; RAP = right atrial pressure; SvO₂ = mixed venous oxygen saturation; VE/VCO₂ = ventilatory equivalents for carbon dioxide; VO₂ = oxygen consumption; WHO = World Health Organization.

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2015 ESC/ERS Guidelines for the diagnosis and treatment of PH
### Suggested assessment and timing for the follow-up of patients with PAH

<table>
<thead>
<tr>
<th></th>
<th>At baseline</th>
<th>Every 3–6 months&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Every 6–12 months&lt;sup&gt;a&lt;/sup&gt;</th>
<th>3–6 months after changes in therapy&lt;sup&gt;a&lt;/sup&gt;</th>
<th>In case of clinical worsening</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical assessment and determination of functional class</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>ECG</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>6MWT/Borg dyspnoea score</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>CPET</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
<td>+&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Echo</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Basic lab&lt;sup&gt;b&lt;/sup&gt;</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Extended lab&lt;sup&gt;c&lt;/sup&gt;</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Blood gas analysis&lt;sup&gt;d&lt;/sup&gt;</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Right heart catheterization</td>
<td>+</td>
<td>+&lt;sup&gt;f&lt;/sup&gt;</td>
<td>+</td>
<td>+&lt;sup&gt;e&lt;/sup&gt;</td>
<td>+&lt;sup&gt;e&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<sup>a</sup>Intervals to be adjusted according to patient needs.

<sup>b</sup>Basic lab includes blood count, INR (in patients receiving vitamin K antagonists), serum creatinine, sodium, potassium, ASAT/ALAT (in patients receiving ERAs), bilirubin and BNP/NT-proBNP.

<sup>c</sup>Extended lab includes TSH, troponin, uric acid, iron status (iron, ferritin, soluble transferrin receptor) and other variables according to individual patient needs.

<sup>d</sup>From arterial or arterialized capillary blood; may be replaced by peripheral oxygen saturation in stable patients or if BGA is not available.

<sup>e</sup>Should be considered.

<sup>f</sup>Some centres perform RHCs at regular intervals during follow-up.
Cardiac output measurement

• Definition
  • Quantity of blood delivered to the systemic circulation per unit time

• Techniques
  • Fick-Oxygen Method
  • Thermodilution
Cardiac output measurement

**Fick Oxygen Method**

- **Fick Principle:** The total uptake or release of any substance by an organ is the product of blood flow to the organ and the arteriovenous concentration difference of the substance.

- In the absence of a shunt, systemic blood flow (Qs) is estimated by pulmonary blood flow (Qp)

\[
Q = \frac{\dot{V}O_2}{C_A O_2 - C_V O_2}
\]

Where:
- \(Q\) is the systemic blood flow
- \(\dot{V}O_2\) is the rate of oxygen consumption
- \(C_A O_2\) is the arterial oxygen content
- \(C_V O_2\) is the venous oxygen content
Cardiac output measurement

Fick Oxygen Method

- **Most accurate in low output states**
- The arteriovenous oxygen content difference (Ao – PA O$_2$ content) can be calculated (in ml oxygen) by the difference between the left ventricular oxygen content and the Mixed venous (pulmonary artery) oxygen content

\[ 1.36 \times \text{Hemoglobin concentration} \times \text{LV oxygen saturation} \]

\[ 1.36 \times \text{hemoglobin concentration} \times \text{PA oxygen saturation} \]

- The value 1.36 is derived from the fact that 1 gram of hemoglobin, when 100% saturated, combines with 1.36 ml of oxygen

\[
CO (l/min) = \frac{O_2 \text{ consumption (ml/min)}}{Ao-PA \ O_2 \text{ content (ml/l)}}
\]

\[
CI = \frac{CO (L/min)}{BSA (m^2)}
\]
Cardiac output measurement

Thermodilution Method

- **Principle:**
  - Injection of a given quantity of cold N/S to the central circulation changes the temperature of blood distally.

- **Technique:**
  - Injecting 10ml N/S at room temperature within 2 seconds from the proximal channel of the Swan-Ganz record temperature-time curve catheter.
Cardiac output measurement

Thermodilution Method

\[ CO \text{ (ml/sec)} = \frac{\text{Volume injected (ml)} \times \text{Temperature difference (°C)}}{\text{Area under the curve (°C.sec)}} \]
Cardiac output measurement

Thermodilution Method

- Imprecise measurement in tricuspid regurgitation
- Overestimates cardiac output in low volume situations
Intracardiac Shunts (1)

• Normally, pulmonary blood flow and systemic blood flow are equal

• When there is an abnormal communication between intracardiac chambers or great vessels, blood flow is shunted either from the systemic circulation to the pulmonary circulation (*left-to-right shunt*), from the pulmonary circulation to the systemic circulation (*right-to-left shunt*), or in both directions (*bidirectional shunt*)
**Intracardiac Shunts (2)**

**Oxymetric method**

- Blood sampling from various cardiac chambers for oxygen saturation determination
- A left-to-right shunt is detected when there is a significant increase in blood oxygen saturation between two right-sided vessels or chambers (step up)
- Despite its lack of sensitivity, clinically significant shunts are generally detected by this technique
“Oximetry run” in patient with...

..atrial septal defect

..ventricular septal defect

Right Heart Catheterization
Shunt Detection & Measurement (2)

- The flow ratio PBF/SBF (or QP/QS) is used clinically to determine the significance of the shunt
  - A ratio of less than 1.5 indicates a small left-to-right shunt
  - A ratio of 2.0 or more indicates a large left-to-right shunt and generally requires repair in order to prevent future pulmonary and/or right ventricular complications
  - A flow ratio of less than 1.0 indicates a net right-to-left shunt

\[
\frac{Q_p}{Q_s} = \frac{SAO_2 - MVO_2}{PVO_2 - PAO_2}
\]
Key points

- Right heart catheterization continues to be the gold standard in diagnosing patients with elevated right heart pressures, although this technique has complications.

- The development of noninvasive techniques has progressed, however prospective clinical trials are lacking.

- It is important to note that the use of the pulmonary artery catheter is a monitoring procedure and not a treatment.
ΣΑΣ ΕΥΧΑΡΙΣΤΩ
ΓΙΑ ΤΗΝ ΥΠΟΜΟΝΗ
ΚΑΙ ΤΗΝ ΠΡΟΣΟΧΗ ΣΑΣ