EVALUATION OF HEART FAILURE WITH PRESERVED EJECTION FRACTION / ΕΚΤΙΜΗΣΗ ΤΗΣ ΚΑΡΔΙΑΚΗΣ ΑΝΕΠΑΡΚΕΙΑΣ ΜΕ ΔΙΑΤΗΡΗΜΕΝΟ ΚΛΑΣΜΑ ΕΞΏΘΗΣΗΣ

ΠΑΤΡΙΑΝΑΚΟΣ ΑΛΕΞΑΝΔΡΟΣ
Δ/ΝΤΗΣ ΚΑΡΔΙΟΛΟΓΙΑΣ ΕΣΥ
ΠΑ.Γ.Ν. ΗΡΑΚΛΕΙΟΥ
ΚΡΗΤΗ, ΕΛΛΑΔΑ
Ασθενής 56 ετών, λεπτοσωματική με ανεξήγητη δύσπνοια από μηνός.
Cardiac amyloidosis
Differential diagnosis of heart failure in the setting of preserved left ventricular ejection fraction.

- Amyloidosis
- Haemochromatosis
- Endomyocardial fibrosis
- Radiation-induced
- Chemotherapy-induced
- Idiopathic

Pericardial disease
- Constrictive pericarditis
- Constrictive effusive disease
- Post-pericardiectomy syndrome

Restrictive CMP

Hypertrophic CMP

Right ventricular failure
- Pulmonary arterial hypertension
- ARVC
- Sarcoidosis
- Tricuspid regurgitation

HF signs and symptoms normal LVEF

Storage disease
- Fabry
- LAMP2
- PRKAG2

HF-PEF

Desai et al European Heart Journal (2016)
<table>
<thead>
<tr>
<th>Type of HF</th>
<th>HFrEF</th>
<th>HFmrEF</th>
<th>HFrEF</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CRITERIA</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Symptoms ± Signs(^a)</td>
<td>Symptoms ± Signs(^a)</td>
<td>Symptoms ± Signs(^a)</td>
</tr>
<tr>
<td>2</td>
<td>LVEF &lt;40%</td>
<td>LVEF 40–49%</td>
<td>LVEF ≥50%</td>
</tr>
<tr>
<td>3</td>
<td>-</td>
<td>Elevated levels of natriuretic peptides(^b); 1. Elevated levels of natriuretic peptides(^b); 2. At least one additional criterion: a. relevant structural heart disease (LVH and/or LAE), a. relevant structural heart disease (LVH and/or LAE), b. diastolic dysfunction (for details see Section 4.3.2). b. diastolic dysfunction (for details see Section 4.3.2).</td>
<td></td>
</tr>
</tbody>
</table>
2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

**DD**
Pathophysiologic condition: impaired relaxation, ↑LV filling pressures, ↓compliance

**DHF**
Normal LVEF plus sign/symptoms of HF due to DD

**HFrEF**
Normal LVEF plus signs/symptoms of HF (excluding severe valve disease, prior ↓LVEF, constriction)
In patients with normal LV EF

- Average E/e' > 14
- Septal e' velocity < 7 cm/s or Lateral e' velocity < 10 cm/s
- TR velocity > 2.8 m/s
- LA volume index > 34 ml/m²

- <50% positive
  - Normal Diastolic function

- 50% positive
  - Indeterminate

- >50% positive
  - Diastolic Dysfunction
Reduced LVEF

Mitral Inflow

- E/A ≤ 0.8 + E ≤ 50 cm/s
- E/A > 0.8 - <2
- E/A ≥ 2

3 criteria to be evaluated:

2 of 3 or 3 of 3
- Negative
- Average E/e’ > 14
- 2-TR velocity > 2.8 m/s
- 3-LV Vol. index > 34 ml/m²

2 of 3 or 3 of 3
- Positive

When only 2 criteria are available:

- 2 negative
- 1 positive and 1 negative
- 2 positive

Normal LAP
- Grade I Diastolic Dysfunction

If Symptomatic:
- Consider CAD, or proceed to diastolic stress test

Cannot determine LAP and Diastolic Dysfunction
- Grade II Diastolic Dysfunction

↑ LAP
- Grade III Diastolic Dysfunction
A. Echocardiography Parameters for Estimation of LV Filling Pressure

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Cutoff Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak Tricuspid Regurgitation Velocity</td>
<td>&gt;2.8 m/sec</td>
</tr>
<tr>
<td>E/e'</td>
<td>&gt;14</td>
</tr>
<tr>
<td>Left Atrial Maximal Volume Index</td>
<td>&gt;34 ml/m²</td>
</tr>
</tbody>
</table>
**Figure 3** Regression Plot: LV Filling Pressure

**Table 4** Accuracy of Diagnosis of Elevated LV Filling Pressure: Total Population

<table>
<thead>
<tr>
<th></th>
<th>Clinical (95% CI)</th>
<th>Echocardiographic (95% CI)</th>
<th>p Value* Clinical vs. Echo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensitivity</td>
<td>74 (68–79)</td>
<td>87 (81–91)</td>
<td>0.001</td>
</tr>
<tr>
<td>Specificity</td>
<td>69 (62–75)</td>
<td>88 (82–93)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PPV</td>
<td>77 (71–82)</td>
<td>91 (86–94)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>NPV</td>
<td>65 (58–72)</td>
<td>83 (76–88)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Overall accuracy</td>
<td>72 (67–76)</td>
<td>87 (84–91)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>
PATIENT WITH SUSPECTED HF* (non-acute onset)

ASSESSMENT OF HF PROBABILITY

1. Clinical history:
   - History of CAD (MI, revascularization)
   - History of arterial hypertension
   - Exposition to cardiotoxic drug/radiation
   - Use of diuretics
   - Orthopnoea / paroxysmal nocturnal dyspnoea

2. Physical examination:
   - Rales
   - Bilateral ankle oedema
   - Heart murmur
   - Jugular venous dilatation
   - Laterally displaced/broadened apical beat

3. ECG:
   - Any abnormality

≥1 present →

NATRIURETIC PEPTIDES
- NT-proBNP ≥125 pg/mL
- BNP ≥35 pg/mL

Yes →

ECHOCARDIOGRAPHY

If HF confirmed (based on all available data): determine aetiology and start appropriate treatment

All absent

HF unlikely: consider other diagnosis

Assessment of natriuretic peptides not routinely done in clinical practice

Normalβα
## 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class</th>
<th>Level</th>
<th>Ref</th>
</tr>
</thead>
<tbody>
<tr>
<td>TTE is recommended for the assessment of myocardial structure and function in subjects with suspected HF in order to establish a diagnosis of either HFrEF, HfmrEF or HFrEF.</td>
<td>I</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>TTE is recommended to assess LVEF in order to identify patients with HF who would be suitable for evidence-based pharmacological and device (ICD, CRT) treatment recommended for HFrEF.</td>
<td>I</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>TTE is recommended for the assessment of valve disease, right ventricular function and pulmonary arterial pressure in patients with an already established diagnosis of either HFrEF, HfmrEF or HFrEF in order to identify those suitable for correction of valve disease.</td>
<td>I</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>TTE is recommended for the assessment of myocardial structure and function in subjects to be exposed to treatment which potentially can damage myocardium (e.g. chemotherapy).</td>
<td>I</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>Other techniques (including systolic tissue Doppler velocities and deformation indices, i.e. strain and strain rate), should be considered in a TTE protocol in subjects at risk of developing HF in order to identify myocardial dysfunction at the preclinical stage.</td>
<td>IIa</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>CMR is recommended for the assessment of myocardial structure and function (including right heart) in subjects with poor acoustic window and patients with complex congenital heart diseases (taking account of cautions/contra-indications to CMR).</td>
<td>I</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>CMR with LGE should be considered in patients with dilated cardiomyopathy in order to distinguish between ischaemic and non-ischaemic myocardial damage in case of equivocal clinical and other imaging data (taking account of cautions/contra-indications to CMR).</td>
<td>IIa</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>CMR is recommended for the characterization of myocardial tissue in case of suspected myocarditis, amyloidosis, sarcoidosis, Chagas disease, Fabry disease non-compaction cardiomyopathy, and haemochromatosis (taking account of cautions/contra-indications to CMR).</td>
<td>I</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>Non-invasive stress imaging (CMR, stress echocardiography, SPECT, PET) may be considered for the assessment of myocardial ischaemia and viability in patients with HF and CAD (considered suitable for coronary revascularization) before the decision on revascularization.</td>
<td>IIb</td>
<td>B</td>
<td>116–118</td>
</tr>
<tr>
<td>Invasive coronary angiography is recommended in patients with HF and angina pectoris recalcitrant to pharmacological therapy or symptomatic ventricular arrhythmias or aborted cardiac arrest (who are considered suitable for potential coronary revascularization) in order to establish the diagnosis of CAD and its severity.</td>
<td>I</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>Invasive coronary angiography should be considered in patients with HF and intermediate to high pre-test probability of CAD and the presence of ischaemia in non-invasive stress tests (who are considered suitable for potential coronary revascularization) in order to establish the diagnosis of CAD and its severity.</td>
<td>IIa</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>Cardiac CT may be considered in patients with HF and low to intermediate pre-test probability of CAD or those with equivocal non-invasive stress tests in order to rule out coronary artery stenosis.</td>
<td>IIb</td>
<td>C</td>
<td></td>
</tr>
<tr>
<td>Reassessment of myocardial structure and function is recommended using non-invasive imaging: - in patients presenting with worsening HF symptoms (including episodes of A-HF) or experiencing any other important cardiovascular event; - in patients with HF who have received evidence-based pharmacotherapy in maximal tolerated doses, before the decision on device implantation (ICD, CRT); - in patients exposed to therapies which may damage the myocardium (e.g. chemotherapy) (serial assessments).</td>
<td>I</td>
<td>C</td>
<td></td>
</tr>
</tbody>
</table>
Heart Failure with Preserved Ejection Fraction

**A Traditional Model**

- **Left Ventricle**
  - Systemic hypertension
  - Vascular dysfunction
  - Concentric hypertrophy
  - Fibrosis
  - Diastolic dysfunction
  - Left atrial hypertension

- **Left Atrium**
  - Remodeling
  - Diastolic dysfunction
  - Systolic dysfunction
  - Pulmonary hypertension
  - Atrial fibrillation

- **Right Ventricle**
  - Remodeling
  - Diastolic dysfunction
  - Systolic dysfunction
  - Right atrial hypertension

- **Right Atrium**
  - Remodeling
  - Diastolic dysfunction
  - Systolic dysfunction

**B Emerging Model**

- **Proinflammatory coexisting conditions**
  - Systemic microvascular endothelial inflammation

  - **Increases in oxidative stress**
  - Decreases in NO–cyclic GMP signaling

  - **Muscle inflammation**

  - **Microvascular dysfunction and rarefaction**

  - **Myofiber stiffness**
  - **Cardiomyocyte hypertrophy**

  - **Fibrosis**

  - **Global cardiac remodeling and dysfunction**
  - Impaired coronary flow reserve
  - Impaired oxygen delivery, uptake, and utilization in skeletal muscle

Solomon NEJM 11/16
Impaired LV filling

- Increased ECM stiffness
  - Increased Type I collagen synthesis and deposition
  - Decreased ECM degradation
- Increased cardiomyocyte stiffness
  - Myocyte hypertrophy
  - Cytoskeletal protein dysfunction
  - Titin hypo-phosphorylation
  - Cross-bridge detachment

Diastolic dysfunction

HFpEF

Other contributory mechanisms

Ventricular-vascular uncoupling

- Increased vascular stiffness
- Decreased vascular distensibility
- Abnormal vaso-relaxation

Increased ventricular load

- Chronotropic incompetence
- Poor CV reserve
  - Abnormalities in beta receptor signaling
  - Myocardial ischemia
  - Abnormal myocardial energetics
MRI and Diastolic function

**Figure 9:** Volume Curves From Cardiac Magnetic Resonance of the LV

- **ED** and **ES** denote end-diastole and end-systole, respectively, with **Blood volume** highlighted.

**Figure 10:** LV Circumferential and Torsional Strain Derived From CMR Tagging

- **A**: Representative echocardiographic images showing end-diastole, end-systole, and end-diastole.
- **B**: Circumferential strain rate and peak circumferential strain during end-diastole and end-systole.
- **C**: Apical rotation, basal rotation, twisting rate, and peak unwinding rate over time.

LV volume on the y-axis and time after R wave on the x-axis. (Top) Normal filling in a young woman without cardiovascular disease. (Bottom) Slowed diastolic filling in a patient with long-standing severe hypertension and LV hypertrophy (mass, 159 g/m²; note the increased LV volume). The red squares identify inflection points of the curve, which can be used for quantification. Such curves can also be generated from nuclear or computed tomography data. ED — end-diastole, ES — end-systole, LV — left ventricular.
Εκτίμηση διαστολικής λειτουργικότητας της ΑΚ
# Echocardiographic Classification of Diastolic Dysfunction

<table>
<thead>
<tr>
<th>Stage</th>
<th>Normal Diastolic Function</th>
<th>Stage I: Impaired Relaxation</th>
<th>Stage II: Pseudonormal</th>
<th>Stage III: Reversible Restrictive</th>
<th>Stage IV: Fixed Restrictive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral Inflow</td>
<td>$E/A&lt;0.75$; $DT=140 ms$</td>
<td>$E/A&lt;0.75$</td>
<td>$E/A&gt;1.5$</td>
<td>$E/A&gt;1.5$</td>
<td>$E/A&gt;1.5$</td>
</tr>
<tr>
<td>Mitral Inflow at Peak Yavalva Maneuver</td>
<td>$\Delta E/A&lt;0.5$</td>
<td>$\Delta E/A&lt;0.5$</td>
<td>$\Delta E/A&lt;0.5$</td>
<td>$\Delta E/A&lt;0.5$</td>
<td>$\Delta E/A&lt;0.5$</td>
</tr>
<tr>
<td>Pulmonary Venous Flow</td>
<td>$S&gt;0$; $ARdur=Adur$</td>
<td>$S&gt;0$; $ARdur=Adur$</td>
<td>$S&lt;0$ or $ARdur=Adur+30 ms$</td>
<td>$S&lt;0$ or $ARdur=Adur+30 ms$</td>
<td>$S&lt;0$ or $ARdur=Adur+30 ms$</td>
</tr>
<tr>
<td>Color M-Mode Propagation Velocity</td>
<td>$Vp &lt; 45$</td>
<td>$Vp &lt; 45$</td>
<td>$Vp &lt; 45$</td>
<td>$Vp &lt; 45$</td>
<td>$Vp &lt; 45$</td>
</tr>
<tr>
<td>Doppler Tissue Imaging of Mitral Annular Motion</td>
<td>$E/E' &lt; 10$</td>
<td>$E/E' &lt; 10$</td>
<td>$E/E' &lt; 10$</td>
<td>$E/E' &lt; 10$</td>
<td>$E/E' &lt; 10$</td>
</tr>
</tbody>
</table>

LV Relaxation: Normal, Impaired
LV Compliance: Normal, Normal to ↓, ↓↓
Atrial Pressure: Normal, ↑↑, ↑↑↑
Ασθενής 25 ετών με ΣΔ, ΑΥΠ, ΥΠΕΡΧΟΛ/Α, BYPASS προσερχεται λόγω δυσπνοίας ηρεμίας και ευκολής καπωσής από μηνός
1) An L-wave is very often an unnoticed finding.

2) The L-wave may be seen in relatively bradycardic patients with normal hearts. (< 20 cm/s)

3) A pathologic L-wave typically is found in patients with delayed active relaxation with increased LV stiffness. In the echo laboratory patients will often have clinical heart failure, LVH with normal systolic function, or LV systolic dysfunction.

4) A pathologic L-wave is suggestive of elevated LV preload (pseudonormalization).

5) A pathologic L-wave has prognostic value, in that it is predictive of future hospitalizations with heart failure.
LV diastolic limitations
LV systolic limitations

- However, with exercise stress, the increase in ejection fraction is blunted in the patient with HFP EF owing to the inability to contract to as low an end-systolic volume, despite similar increases in cavity size at end diastole. This systolic impairment limits stroke volume reserve, which in tandem with chronotropic incompetence blunts the cardiac output response to exercise.

- Diminished reductions in ESV impair early diastolic suction, promoting left atrial hypertension, while also blunting the normal increase in forward stroke volume that is required with exercise.
The pathophysiology of heart failure with preserved ejection fraction

Barry A. Borlaug
Figure 30 A forward and a backward-traveling (reflected) wave contribute to pressure changes in the central aorta. In the young and healthy subject on the left, the backward-traveling wave arrives at end-systole, contributes to closing the aortic valve and to increasing diastolic perfusion pressure. In the hypertensive subject on the right, the backward-traveling wave reaches the proximal aorta in early systole and contributes to the late systolic peak in pressure. The magnitude of the reflected wave (and the late systolic pressure) has a well-validated and independent prognostic significance as summarized in the guidelines.
Diastolic left ventricular (LV) filling is shown with simultaneous recordings of left atrial (LA) and LV pressures, the diastolic transmitral pressure gradient, pulmonary venous, transmural, and transmitral flow velocity, and septal tissue (or mitral annulus) velocity. Flow velocity and tissue velocity data, together with LA size and septal pulmonary pressure, are used in echocardiography to infer diastolic LV pressures and hence diastolic LV function.
Em depends on
- left atrial driving pressure,
- LV relaxation kinetics, and
- age but

e' depends mostly on LV relaxation kinetics and age.

Hence, in the ratio E/e', effects of LV relaxation kinetics and age are eliminated and the ratio becomes a measure of left atrial driving pressure or LV filling pressure.

E' can also be conceptualized as the amount of blood entering the LV during early filling, whereas E represents the gradient necessary to make this blood enter the LV. ($dV/dp$)
Caution should be used when using E/e’ in LV disorders such as hypertrophic cardiomyopathy and myocardial infarction, as the downward movement during diastole can be influenced by upward movement during systole. Conversely, the transmitral E measurement may be the source of misleading information in the setting of moderate to severe mitral regurgitation (MR) and severe LV dysfunction. Patients with constrictive pericarditis,

Table 1. Situations where the use of E/e’ may be unreliable

- Tachycardia with fusion of E and A velocities
- Unreliable measurement of E velocity
  - Significant mitral regurgitation (>2+)
- Unreliable measurement of e’ velocity
  - Mitral valve repair or replacement
  - Severe mitral annular calcification
  - Significant mitral stenosis
  - Presence of left bundle branch block
- Significant aortic regurgitation (>2+)
Relaxation of the myocardial contraction and twist unloads this energy, and this process begins before the end of LV ejection.

LV pressure falls rapidly during the isovolumic relaxation period and produces an early diastolic pressure gradient between the left atrial (LA) and LV that sucks blood out of the LA and fills the LV rapidly.

Thus, in the normal heart, myocardial relaxation (e') and suction precede the onset of LV passive filling (E).

In contrast, the failing ventricle shows reduction of passive ventricular filling and elevation of LA pressure, so blood is pushed rather than sucked into the LV.

In this setting, myocardial diastolic motion (e') reflecting cardiac movement during diastole may be secondary to filling (E).
The 17% decrease in E-wave velocity reflects a reduced transmitral pressure gradient, which may result from either a lower left atrial pressure or a higher proto-diastolic LV pressure.

The e‘ average demonstrated a 28% decrease with position change from supine to upright, with comparable reductions in both e‘ septal and e‘ lateral.

Together with previous studies that used different interventions to induce changes in LV preload, these findings clearly demonstrate that e‘ is not load-independent.

This preload dependence may be more pronounced in patients with acompliant myocardium, which may be more susceptible to changes in external load than a stiff myocardium.

**Conclusions**—In patients with unexplained dyspnea, E/e’ ratio neither accurately estimates PAWP nor identifies patients with elevated PAWP consistent with heart failure with preserved ejection fraction. Positional changes in E/e’ ratio do not reflect changes in PAWP. *(Circ Heart Fail. 2015;8:749-756. DOI: 10.1161/CIRCHEARTFAILURE.115.002161.)*
40 ετών με ιδιοπαθή θρομβοκυτταρωσή, παλαιό ΕΜ και ΑΓΠ- > LAD
E′ s = 9 cm/s
E′ l = 12 cm/s

E′ s = 7 cm/s
E′ l = 9 cm/s

Em = 0.82 cm/s
E/e′ = 7.8

Em = 0.72 cm/s
E/e′ = 9

SUPINE

STANDING
**Figure 7** Diastolic Strain Rate

Spacelode tracking-based early diastolic longitudinal strain rate (red double arrow; 1/s) from the mid-septum (blue dot in left 4-chamber view) of a patient with normal left ventricular function. AVC = aortic valve closure.

**Figure 8** Left Ventricular Rotation

- **Male Patient - 62 Years**
  - LOCAL: Rotation (deg) = 9.95
  - T = 337 msec
  - Peak Rotation: 9.95° (337 ms)
  - Untwist at 25% of Untwist Duration: 9.25 = 7% (378 ms)

- **Female Patient - 61 Years**
  - LOCAL: Rotation (deg) = 23.36
  - T = 425 msec
  - Peak Rotation: 23.36° (425 ms)
  - Untwist at 25% of Untwist Duration: 18.68 = 28% (491 ms)

Rotation curve of patient with heart failure with preserved ejection fraction (left) and control patient (right) showing reduced magnitude of peak apical rotation and percentage of early untwist (25% of total untwist duration), derived from speckle tracking 2-dimensional echocardiography. AVC = aortic valve closure. Reproduced with permission from Tan et al. [46].
Estimation of left ventricular filling pressures by speckle tracking echocardiography in patients with idiopathic dilated cardiomyopathy

Meluzin et al.

European Journal of Echocardiography (2011)

### Table 6  Accuracy of echocardiographic parameters in predicting PCWP

<table>
<thead>
<tr>
<th>Parameter and cut-off</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>AUC (95% CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predicting PCWP &gt; 12 mmHg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conventional pulsed Doppler</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>E (cm s⁻¹) &gt; 57.5</td>
<td>97.4%</td>
<td>81.9%</td>
<td>0.912 (0.781−1.000)</td>
<td>0.001</td>
</tr>
<tr>
<td>DT (ms) &lt; 146</td>
<td>92.3%</td>
<td>81.6%</td>
<td>0.894 (0.804−0.983)</td>
<td>0.001</td>
</tr>
<tr>
<td>Doppler tissue imaging</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aₐ (cm s⁻¹) &lt; 5.1</td>
<td>92.3%</td>
<td>64.4%</td>
<td>0.853 (0.750−0.957)</td>
<td>0.001</td>
</tr>
<tr>
<td>2D-STE</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Strain A_long (%) &lt; 3.3</td>
<td>84.6%</td>
<td>80.0%</td>
<td>0.875 (0.768−0.981)</td>
<td>0.001</td>
</tr>
<tr>
<td>Strain A_short (%) &lt; 2.4</td>
<td>90.9%</td>
<td>88.9%</td>
<td>0.956 (0.880−0.990)</td>
<td>0.001</td>
</tr>
<tr>
<td>SR_A_orc (cm⁻¹ s⁻¹) &lt; 0.3</td>
<td>90.9%</td>
<td>84.1%</td>
<td>0.951 (0.870−0.989)</td>
<td>0.001</td>
</tr>
<tr>
<td>Echocardiographic indexes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>E/A &gt; 1.2</td>
<td>81.6%</td>
<td>92.3%</td>
<td>0.895 (0.793−0.996)</td>
<td>0.001</td>
</tr>
<tr>
<td>E/E orc &gt; 11.8</td>
<td>86.8%</td>
<td>92.3%</td>
<td>0.911 (0.809−1.000)</td>
<td>0.001</td>
</tr>
<tr>
<td>E/E orc &gt; 13.3</td>
<td>86.8%</td>
<td>92.3%</td>
<td>0.929 (0.855−1.000)</td>
<td>0.001</td>
</tr>
<tr>
<td>E/E orc &gt; 9.8</td>
<td>76.3%</td>
<td>84.6%</td>
<td>0.859 (0.742−0.977)</td>
<td>0.001</td>
</tr>
<tr>
<td>ESR_E orc (cm) &gt; 1000</td>
<td>88.2%</td>
<td>90.9%</td>
<td>0.894 (0.866−0.978)</td>
<td>0.001</td>
</tr>
<tr>
<td>E/Strain TE₉_long (cm s⁻¹ %⁻¹) &lt; −17.1</td>
<td>84.6%</td>
<td>97.1%</td>
<td>0.920 (0.820−0.964)</td>
<td>0.001</td>
</tr>
<tr>
<td>E/Strain TE₉_circ (cm s⁻¹ %⁻¹) &lt; −20.2</td>
<td>81.8%</td>
<td>94.3%</td>
<td>0.948 (0.840−0.954)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Parameter and cut-off</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>AUC (95% CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predicting PCWP &gt; 24 mmHg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conventional pulsed Doppler</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>DT (ms) &lt; 1140</td>
<td>90.0%</td>
<td>87.1%</td>
<td>0.885 (0.782−0.989)</td>
<td>0.001</td>
</tr>
<tr>
<td>2D-STE</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Strain A_orc (%) &lt; 1.1</td>
<td>88.9%</td>
<td>80.0%</td>
<td>0.900 (0.807−0.993)</td>
<td>0.001</td>
</tr>
<tr>
<td>Echocardiographic indexes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>E/E orc &gt; 13.7</td>
<td>85.0%</td>
<td>84.2%</td>
<td>0.890 (0.767−0.923)</td>
<td>0.001</td>
</tr>
<tr>
<td>E/Strain TE₉_long (cm s⁻¹ %⁻¹) &lt; −23</td>
<td>89.7%</td>
<td>72.4%</td>
<td>0.853 (0.744−0.962)</td>
<td>0.001</td>
</tr>
</tbody>
</table>
Ασθενής 68 ετών με ιστορικό αγγειοπλασθείσας ΣΝ, ΑΥΠ, ΣΔ προσέρχεται λόγω δύσπνοιας προσπάθειάς
Περιστατικό : Μέτρηση Ακ

La Vol = 44 ml/m2
Ασθενής 72 ετών με ιστορικό by-pass προ 7ετίας και ακτινοβολίας θώρακα προ 20 ετίας λόγω λεμφώματος Hodgkin προσέρχεται λόγω επιδεινούμενης δύσπνοιας και οιδήματος κάτω άκρων
Mixed restrictive cardiomyopathy and constrictive pericarditis

This diagnosis was made according to 3 well-defined criteria:

1. **Localized thickening of the pericardium** observed by TTE and/or TEE echocardiography and confirmed by MRI or CT.
2. **Restrictive Doppler findings** of transmitral and/or pulmonary venous flow velocity patterns and
3. **No significant respiratory variations** on early diastolic transmitral flow velocity (<25%).

Yanada et al. EJE 2007
ΣΥΜΠΕΡΑΣΜΑ

- Το υπερηχοκαρδιογράφημα αποτελεί την πρώτη (και ίσως μοναδική τεχνική) για την αξιολόγηση της διαστολικής λειτουργικότητας της ΑΚ.
- Κανένας δείκτης από μόνος του δεν μπορεί να θέσει την διάγνωση της διαστολικής ΚΑ.
- Η διάγνωση της θα τεθεί σε συνδυασμό με τα κλινικά-υπερηχογραφία (ανατομικά+ Doppler + ιστικές ταχύτητες) καθώς και τα δεδομένα στην άσκηση.