Imagen cardiovascular con un enfoque clínico

-2011 -
Ecocardiografía
EAE/ASE recommendations for the use of echocardiography in new transcatheter interventions for valvular heart disease

Jose L. Zamorano, Luigi P. Badano, Charles Bruce, Kwan-Leung Chan, Alexandra Gonçalves, Rebecca T. Hahn, Martin G. Keane, Giovanni La Canna, Mark J. Monaghan, Petros Nihoyannopoulos, Frank E. Silvestry, Jean-Louis Vanoverschelde, and Linda D. Gillam

Document Reviewers: European Association of Echocardiography (EAE): Alec Vahanian, Vito Di Bello, Thomas Buck; American Society of Echocardiography (ASE): The ASE Guidelines and Standards Committee and the ASE Board of Directors

1University Clinic San Carlos, Madrid, Spain; 2University of Padua, Padua, Italy; 3Mayo Clinic, Rochester, MN, USA; 4University of Ottawa Heart Institute, Ottawa, Ontario, Canada; 5University of Porto, Porto, Portugal; 6Columbia University Medical Center, New York, NY, USA; 7University of Pennsylvania School of Medicine, Philadelphia, PA, USA; 8San Raffaele Scientific Institute, Milan, Italy; 9King's College Hospital, London, UK; 10Imperial College London, Hammersmith Hospital, NHLI, London, UK; 11Cliniques Universitaires Saint-Luc, Brussels, Belgium; and 12Gagnon Cardiovascular Institute, Morristown, NJ, USA
Hemodynamic and Clinical Impact of Prosthesis–Patient Mismatch After Transcatheter Aortic Valve Implantation

See Hooi Ewe, MBBS,*† Manuela Muratori, MD,‡ Victoria Delgado, MD, PhD,* Mauro Pepi, MD,‡ Gloria Tamborini, MD,‡ Laura Fusini, MS,§ Robert J. M. Klautz, MD, PhD,* Paola Gripari, MD,‡ Jeroen J. Bax, MD, PhD,* Melissa Fusari, MD,‡ Martin J. Schalij, MD, PhD,* Nina Ajmone Marsan, MD*||

Leiden and Utrecht, the Netherlands; Singapore; and Milan, Italy

Objectives
This study examined the mid-term hemodynamic and clinical impact of prosthesis–patient mismatch (PPM) in patients undergoing transcatheter aortic valve implantation (TAVI) with balloon-expandable valves.

Background
PPM can be observed after aortic valve surgery. However, little is known about the incidence of PPM in patients undergoing TAVI.

Methods
Echocardiography and clinical assessment were performed in 165 patients at baseline, before hospital discharge, and at 6 months after TAVI. PPM was defined as an indexed effective orifice area ≤0.85 cm²/m².

Results
Thirty patients (18.2%) showed PPM before hospital discharge. At baseline, patients with PPM had a larger body surface area (1.84 ± 0.18 m² vs. 1.73 ± 0.18 m², p = 0.003) and a greater severity of aortic stenosis (indexed valve area 0.35 ± 0.09 cm²/m² vs. 0.40 ± 0.10 cm²/m², p = 0.005) than patients without PPM. Patients with PPM demonstrated a slower and smaller reduction in mean transaortic gradient, limited left ventricular (LV) mass regression, and left atrial volume reduction over 6 months compared with patients without PPM. LV filling pressure, measured by E/e′, tended to remain elevated in patients with PPM. Importantly, a higher proportion of patients with PPM did not improve in New York Heart Association functional class compared with patients without PPM (36.7% vs. 1.5%, p < 0.001), although major adverse valve-related and cardiovascular events did not differ between the 2 groups.

Conclusions
PPM may be observed after TAVI and when present may be accompanied by less favorable changes in transvalvular hemodynamics, limited LV mass regression, persistent elevated LV filling pressure, and less improvement in clinical functional status. (J Am Coll Cardiol 2011;58:1910–8) © 2011 by the American College of Cardiology Foundation
Figure 1  Impact of PPM

Comparison of changes in aortic valve effective orifice area index (cm²/m²)

The p value is for the group-by-time interaction. *p < 0.05 below each graph.

Figure 4  Kaplan-Meier Curves for Major Adverse Events After Transcatheter Aortic Valve Implantation

Kaplan-Meier probability of freedom from combined major adverse valve-related and cardiovascular events (MAVCE) in patients with and without prosthesis-patient mismatch (PPM).
Tissue Doppler Imaging and Plasma BNP Levels to Assess the Prognosis in Patients with Hypertrophic Cardiomyopathy

Hiroaki Kitaoka, MD, Toru Kubo, MD, Makoto Okawa, MD, Nana Takenaka, MD, Chiyo Sakamoto, MD, Yuichi Baba, MD, Kayo Hayashi, MD, Naohito Yamasaki, MD, Yoshihisa Matsumura, MD, and Yoshinori L. Doi, MD, Kochi, Japan

Background: In addition to sudden death, heart failure and stroke due to atrial fibrillation are important in patients with hypertrophic cardiomyopathy (HCM). The aim of the present study was to determine whether Doppler tissue imaging findings and plasma B-type natriuretic peptide (BNP) levels, which are widely used for risk stratification in several cardiovascular diseases, are useful for risk stratification in patients with HCM in a regional cohort.

Methods: One hundred thirty patients (82 men; mean age, 60 ± 16 years) with HCM were enrolled in this study.

Results: Twenty end points were observed during a mean follow-up period of 3.7 ± 1.7 years. Septal E/e' ratios and BNP levels in patients with events were higher than those in patients without events (17.4 ± 6.3 vs 10.6 ± 4.3, P < .0001, and 441 ± 304 vs 202 ± 174 pg/mL, P < .0001, respectively). By multivariate logistic regression analysis, a high septal E/e' ratio, in addition to a history of syncope and documentation of atrial fibrillation, was a significant predictor of combined end points. In contrast, plasma BNP levels were not a significant predictor of combined end points.

Conclusion: Assessment by Doppler tissue imaging is useful for further risk stratification of patients with HCM. (J Am Soc Echocardiogr 2011;24:1020-5.)
### Table 2  Echocardiographic findings of patients with and without events

<table>
<thead>
<tr>
<th>Variable</th>
<th>All patients</th>
<th>With events</th>
<th>Without events</th>
</tr>
</thead>
<tbody>
<tr>
<td>NYHA class (class III/IV = 1, I/II = 0)</td>
<td>0.02</td>
<td>–</td>
<td>.85</td>
</tr>
<tr>
<td>Syncope (yes = 1, no = 0)</td>
<td>4.31</td>
<td>6.8</td>
<td>.04</td>
</tr>
<tr>
<td>Atrial fibrillation (yes = 1, no = 0)</td>
<td>9.07</td>
<td>12.4</td>
<td>.003</td>
</tr>
<tr>
<td>Left atrial volume</td>
<td>0.18</td>
<td>1.0</td>
<td>.68</td>
</tr>
<tr>
<td>Septal E/e' ratio (≥15 = 1, &lt;15 = 0)</td>
<td>4.57</td>
<td>6.2</td>
<td>.03</td>
</tr>
<tr>
<td>Plasma BNP level (≥188.9 = 1, &lt;188.9 = 0)</td>
<td>1.49</td>
<td>3.1</td>
<td>.22</td>
</tr>
<tr>
<td>LV outflow tract gradient (yes = 1, no = 0)</td>
<td>0.04</td>
<td>1.22</td>
<td>.84</td>
</tr>
<tr>
<td>Maximum LV wall thickness</td>
<td>2.12</td>
<td>1.22</td>
<td>.15</td>
</tr>
</tbody>
</table>

### Table 3  Multivariate logistic regression analysis

<table>
<thead>
<tr>
<th>Variable</th>
<th>( \chi^2 )</th>
<th>Odds ratio</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>NYHA class (class III/IV = 1, I/II = 0)</td>
<td>0.02</td>
<td>–</td>
<td>.85</td>
</tr>
<tr>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>( \chi^2 )</th>
<th>Odds ratio</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Septal e' (mm)</td>
<td>8.2 ± 2.0</td>
<td>6 ± 2.1</td>
<td>7.2 ± 2.2</td>
</tr>
<tr>
<td>Septal E/e' ratio</td>
<td>11.7 ± 5.2</td>
<td>17.4 ± 6.3</td>
<td>10.6 ± 4.3</td>
</tr>
<tr>
<td>Septal E/e' ratio &gt; 15</td>
<td>24 (18%)</td>
<td>11 (54%)</td>
<td>13 (11%)</td>
</tr>
</tbody>
</table>

\( \text{LVDD}, \text{LV end-diastolic dimension}; \text{LVDs}, \text{LV end-systolic dimension}; \text{LVOT}, \text{LV outflow tract}; \text{MVO}, \text{midventricular obstruction}. \)

*Comparison between patients with and those without events.*
Diastolic Dysfunction in Patients Undergoing Cardiac Surgery

A Pathophysiological Mechanism Underlying the Initiation of New-Onset Post-Operative Atrial Fibrillation

Rowlens M. Melduni, MD,* Rakesh M. Suri, MD, DPHIL,† James B. Seward, MD,* Kent R. Bailey, PHD,* Naser M. Ammash, MD,* Jae K. Oh, MD,* Hartzell V. Schaff, MD,† Bernard J. Gersh, MB, CHB, DPHIL*

Rochester, Minnesota

Objectives
Our goal was to investigate whether left ventricular (LV) diastolic dysfunction was an important pathophysiological mechanism underlying the initiation of new-onset post-operative atrial fibrillation (POAF).

Background
Atrial fibrillation is a common complication after cardiac surgery. However, the precise mechanism underlying its development remains poorly understood. Pre-existing alterations of myocardial diastolic function may predispose patients to the development of POAF.

Methods
Patients were residents of Olmsted County, Minnesota, who underwent complete LV diastolic function assessment before coronary artery bypass grafting and/or valve surgery between January 1, 2000, and December 31, 2005. All were in sinus rhythm and had no history of atrial fibrillation, a pacemaker, mitral stenosis, or congenital heart disease. POAF was defined as any episode of atrial fibrillation within 30 days after surgery.

Results
POAF occurred in 135 of 351 patients (38.5%). Patients with POAF were older (mean age 72.5 ± 10.3 years vs. 63.1 ± 14.1 years; p < 0.001) and more likely to have abnormal diastolic function. The rate of POAF increased exponentially with diastolic function grade (DFG) severity (p < 0.001). By multivariate analysis, after adjusting for clinical and surgical risk factors, independent predictors of POAF were older age (odds ratio [OR]: 1.05; p < 0.001), higher body mass index (OR: 1.06; p = 0.03), and abnormal LV DFG (DFG 1, OR: 5.12 [p = 0.006]; DFG 2, OR: 9.87 [p < 0.001]; and DFG 3, OR: 28.52 [p < 0.001]).

Conclusions
LV diastolic dysfunction is a powerful, independent predisposing substrate for the initiation of POAF. Evaluation may be useful during risk stratification of patients undergoing cardiac surgery. (J Am Coll Cardiol 2011;58:953–61) © 2011 by the American College of Cardiology Foundation
Figure 2  Incidence of POAF as a Function of Age and Diastolic Function

The rate of post-operative atrial fibrillation (POAF) increased significantly across diastolic function grades (stratified by age). Grade 1 versus 0, p = 0.006; grade 2 versus 0, <0.001; grade 3 versus 0, <0.001.
<table>
<thead>
<tr>
<th>Covariates</th>
<th>OR</th>
<th>95% CI</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>1.05</td>
<td>1.02–1.08</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>1.06</td>
<td>1.01–1.11</td>
<td>0.03</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1.03</td>
<td>0.55–1.95</td>
<td>0.92</td>
</tr>
<tr>
<td>Mitral regurgitation (&gt;moderate)</td>
<td>1.85</td>
<td>0.83–4.12</td>
<td>0.13</td>
</tr>
<tr>
<td>Diastolic function grade 1</td>
<td>5.12</td>
<td>1.60–16.38</td>
<td>0.006</td>
</tr>
<tr>
<td>Diastolic function grade 2</td>
<td>9.87</td>
<td>3.23–30.13</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diastolic function grade 3</td>
<td>28.52</td>
<td>7.11–114.44</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Univalvular surgery</td>
<td>0.90</td>
<td>0.46–1.73</td>
<td>0.75</td>
</tr>
<tr>
<td>Multivalvular surgery</td>
<td>4.49</td>
<td>0.46–43.60</td>
<td>0.20</td>
</tr>
<tr>
<td>CABG + valvular surgery</td>
<td>1.42</td>
<td>0.69–2.91</td>
<td>0.34</td>
</tr>
<tr>
<td>Perfusion time</td>
<td>1.00</td>
<td>0.99–1.01</td>
<td>0.89</td>
</tr>
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</table>
Echocardiographic diastolic parameters and risk of atrial fibrillation: the Cardiovascular Health Study

Michael A. Rosenberg\textsuperscript{1*}, John S. Gottdiener\textsuperscript{2}, Susan R. Heckbert\textsuperscript{3}, and Kenneth J. Mukamal\textsuperscript{4}

\textsuperscript{1}Beth Israel Deaconess Medical Center, Cardiovascular Institute, 185 Pilgrim Road, Baker 4, Boston, MA 02215, USA; \textsuperscript{2}Department of Medicine, University of Maryland Hospital, Baltimore, MD, USA; \textsuperscript{3}Department of Epidemiology, University of Washington, Seattle, WA, USA; and \textsuperscript{4}Department of General Medicine and Primary Care, Beth Israel Deaconess Medical Center, Boston, MA, USA

Received 13 April 2011; revised 16 August 2011; accepted 13 September 2011

Aims

Atrial fibrillation (AF) is the most common sustained arrhythmia in the elderly, and shares several risk factors with diastolic dysfunction, including hypertension and advanced age. The purpose of this study is to examine diastolic dysfunction as a risk for incident AF.

Methods and results

We examined the association of echocardiographic parameters of diastolic function with the incidence of AF in 4480 participants enrolled in the Cardiovascular Health Study, an ongoing cohort of community-dwelling older adults from four US communities. Participants underwent baseline echocardiography in 1989–1990 and were followed for incident AF on routine follow-up and hospitalizations. After 50941 person-years of follow-up (median follow-up time 12.1 years), 1219 participants developed AF. In multivariable-adjusted age-stratified Cox models, diastolic echocardiographic parameters were significantly associated with the risk of incident AF. The most significant parameters were the Doppler peak E-wave velocity and left atrial diameter, which demonstrated a positive nonlinear association [HR 1.5 (CI 1.3–1.9) and HR 1.7 (CI 1.4–2.1) for highest vs. lowest quintile, respectively], and Doppler A-wave velocity time integral, which displayed a U-shaped relationship with the risk of AF [HR 0.7 (CI 0.6–0.9) for middle vs. lowest quintile]. Each diastolic parameter displayed a significant association with adjusted NT-proBNP levels, although the nature of the association did not entirely parallel the risk of AF. Further cluster analysis revealed unique patterns of diastolic function that may identify patients at risk for AF.

Conclusion

In a community-based population of older adults, echocardiographic measures of diastolic function are significantly associated with an increased risk of AF.
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Quantile/group</th>
<th>Adjusted HR</th>
<th>95% CI</th>
<th>P heterogeneity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak E velocity</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>1 (0.16–0.56 m/s)</td>
<td>1.0</td>
<td>–</td>
<td></td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>2 (0.57–0.65 m/s)</td>
<td>0.983</td>
<td>0.807–1.197</td>
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</tr>
<tr>
<td>3 (0.66–0.73 m/s)</td>
<td>1.095</td>
<td>0.900–1.334</td>
<td></td>
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</tr>
<tr>
<td>4 (0.74–0.83 m/s)</td>
<td>1.172</td>
<td>0.961–1.429</td>
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<td></td>
</tr>
<tr>
<td>5 (0.84–1.85 m/s)</td>
<td>1.549</td>
<td>1.275–1.883</td>
<td></td>
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</tr>
<tr>
<td>A wave VTI</td>
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<td></td>
<td></td>
<td>0.005</td>
</tr>
<tr>
<td>1 (1–6 cm)</td>
<td>1.0</td>
<td>–</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 (7–7 cm)</td>
<td>0.881</td>
<td>0.716–1.085</td>
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<tr>
<td>3 (8–9 cm)</td>
<td>0.742</td>
<td>0.622–0.886</td>
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<tr>
<td>4 (10–10 cm)</td>
<td>0.803</td>
<td>0.645–1.001</td>
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<tr>
<td>5 (11–80 cm)</td>
<td>0.946</td>
<td>0.772–1.160</td>
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<tr>
<td>Left atrial diameter</td>
<td></td>
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<td></td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>1 (1.5–3.3 cm)</td>
<td>1.0</td>
<td>–</td>
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</tr>
<tr>
<td>2 (3.3–3.7 cm)</td>
<td>1.002</td>
<td>0.818–1.228</td>
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<tr>
<td>3 (3.7–4.0 cm)</td>
<td>0.954</td>
<td>0.774–1.177</td>
<td></td>
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</tr>
<tr>
<td>4 (4.0–4.4 cm)</td>
<td>1.176</td>
<td>0.961–1.440</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 (4.4–6.5 cm)</td>
<td>1.696</td>
<td>1.386–2.075</td>
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<tr>
<td>LV systolic function</td>
<td></td>
<td></td>
<td></td>
<td>0.33</td>
</tr>
<tr>
<td>Normal</td>
<td>1.0</td>
<td>–</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mildly depressed</td>
<td>1.227</td>
<td>0.937–1.605</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severely depressed</td>
<td>1.077</td>
<td>0.694–1.670</td>
<td></td>
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</tr>
<tr>
<td>LV chamber size</td>
<td></td>
<td></td>
<td></td>
<td>0.55</td>
</tr>
<tr>
<td>Normal</td>
<td>1.0</td>
<td>–</td>
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</tr>
<tr>
<td>Mildly dilated</td>
<td>1.072</td>
<td>0.839–1.368</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severely dilated</td>
<td>1.212</td>
<td>0.844–1.741</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Adjusted for gender, height, BMI, any AV block on ECG, bradycardia on ECG, any Q waves on ECG, any history of ventricular conduction disease on ECG, history of congestive heart failure, diabetes, hypertension, or myocardial infarction, systolic, and diastolic blood pressure, random glucose and creatinine level, diuretic use, beta-blockers, calcium channel blockers, and ACE inhibitors, and peak FEV-1, as well as peak E velocity, A wave VTI, LA diameter, qualitative LV systolic function, and qualitative LV chamber size.
Figure 1  (A) Kaplan–Meier curve for quantiles of Doppler early mitral inflow velocity on survival free from atrial fibrillation, with adjustment for risk factors (see text). (B) KM curve for Doppler late mitral inflow velocity time integral. (C) KM curve for M-mode derived left atrial size.
Resonancia magnética
High-Resolution Magnetic Resonance Myocardial Perfusion Imaging at 3.0-Tesla to Detect Hemodynamically Significant Coronary Stenoses as Determined by Fractional Flow Reserve

Timothy Lockie, BSc, MBChB,∗ Masaki Ishida, MD, PhD,† Divaka Perera, MD,* Amedeo Chiribiri, MD,† Kalpa De Silva, MBBS,* Sebastian Kozerke, PhD,† Mike Marber, MD, PhD,* Eike Nagel, MD, PhD,† Reza Rezavi, MD,† Simon Redwood, MD,* Sven Plein, MD, PhD††

London and Leeds, United Kingdom

Objectives
The objective of this study was to compare visual and quantitative analysis of high spatial resolution cardiac magnetic resonance (CMR) perfusion at 3.0-T against invasively determined fractional flow reserve (FFR).

Background
High spatial resolution CMR myocardial perfusion imaging for the detection of coronary artery disease (CAD) has recently been proposed but requires further clinical validation.

Methods
Forty-two patients (33 men, age 57.4 ± 9.6 years) with known or suspected CAD underwent rest and adenosinestress k-space and time sensitivity encoding accelerated perfusion CMR at 3.0-T achieving in-plane spatial resolution of 1.2 × 1.2 mm². The FFR was measured in all vessels with >50% severity stenosis. Fractional flow reserve <0.75 was considered hemodynamically significant. Two blinded observers visually interpreted the CMR data. Separately, myocardial perfusion reserve (MPR) was estimated using Fermi-constrained deconvolution.

Results
Of 126 coronary vessels, 52 underwent pressure wire assessment. Of these, 27 lesions had an FFR <0.75. Sensitivity and specificity of visual CMR analysis to detect stenoses at a threshold of FFR <0.75 were 0.82 and 0.94 (p < 0.0001), respectively, with an area under the receiver-operator characteristic curve of 0.92 (p < 0.0001). From quantitative analysis, the optimum MPR to detect such lesions was 1.58, with a sensitivity of 0.80, specificity of 0.89 (p < 0.0001), and area under the curve of 0.89 (p < 0.0001).

Conclusions
High-resolution CMR MPR at 3.0-T can be used to detect flow-limiting CAD as defined by FFR, using both visual and quantitative analyses.

(J Am Coll Cardiol 2011:57:70–5) © 2011 by the American College of Cardiology Foundation
Figure 4  Scatter Plot Showing Distribution of MPR Values According to FFR

A dichotomous cut-off of 0.75 was used to signify a significant lesion. Abbreviations as in Figures 1 and 3.
Non-invasive estimation of pulmonary vascular resistance with cardiac magnetic resonance

Ana García-Alvarez¹,²,³, Leticia Fernández-Friera¹, Jesús G. Mirelis¹,², Simonette Sawit¹, Ajith Nair¹, Jill Kallman¹, Valentin Fuster¹,², and Javier Sanz¹*

¹The Zena and Michael A. Wiener Cardiovascular Institute and Marie-Josee and Henry R. Kravis Center for Cardiovascular Health, Mount Sinai School of Medicine, New York, USA; ²Centro Nacional de Investigaciones Cardiovasculares (CNIC), Madrid, Spain; and ³Thorax Institute. Cardiology Department, Hospital Clinic, Barcelona, Spain

Received 7 January 2011; revised 28 March 2011; accepted 9 May 2011; online publish-ahead-of-print 30 May 2011

Aim
To develop a cardiac magnetic resonance (CMR) method for non-invasive estimation of pulmonary vascular resistance (PVR).

Methods and results
The study comprised 100 consecutive patients with known or suspected pulmonary hypertension (PH; 53 ± 16 years, 73% women) who underwent same-day right heart catheterization (RHC) and CMR. Increased PVR was defined from RHC as >3 WU (n = 66, 66%). From CMR cine and phase-contrast images, right ventricular (RV) volumes and ejection fraction (RVEF), pulmonary artery (PA) flow velocities and areas, and cardiac output were quantified. The best statistical model to estimate PVR was obtained from a derivation cohort (n = 80) based on physiological plausibility and statistical criteria. Validity of the model was assessed in the remaining 20 patients (validation cohort). The CMR-derived model was: estimated PVR (in WU) = 19.38 − [4.62 × Ln PA average velocity (in cm/s)] − [0.08 × RVEF (in %)]. In the validation cohort, the correlation between invasively quantified and CMR-estimated PVR was 0.84 (P < 0.001). The mean bias between the RHC-derived and CMR-estimated PVR was −0.54 (agreement interval −6.02 to 4.94 WU). The CMR model correctly classified 18 (90%) of patients as having normal or increased PVR (area under the receiver operator characteristics curve 0.97; 95% confidence interval: 0.89–1.00).

Conclusions
Non-invasive estimation of PVR using CMR is feasible and may be valuable for PH diagnosis and/or follow-up.
Figure 2  Diagnostic accuracy of the model in the derivation cohort. (A) Correlation between pulmonary vascular resistance estimated by cardiac magnetic resonance and pulmonary vascular resistance quantified with right heart catheterization. (B) Bland–Altman analysis of pulmonary vascular resistance obtained using cardiac magnetic resonance and right heart catheterization. Continuous line represents the mean difference and dashed lines the limits of agreement. (C) Area under the receiver operator characteristics curve (AUC) with 95% confidence interval for the cardiac magnetic resonance model to detect increased pulmonary vascular resistance (>3 WU by right heart catheterization).

Figure 3  Diagnostic accuracy of the model in the validation cohort. (A) Correlation between pulmonary vascular resistance estimated by cardiac magnetic resonance and cardiac magnetic resonance quantified with right heart catheterization. (B) Bland–Altman analysis of pulmonary vascular resistance obtained using cardiac magnetic resonance and right heart catheterization. Continuous line represents the mean difference and dashed lines the limits of agreement. (C) Area under the receiver operator characteristics curve with 95% confidence interval for the cardiac magnetic resonance model to detect increased pulmonary vascular resistance (>3 WU by right heart catheterization).
Myocardial Fibrosis Predicts Appropriate Device Therapy in Patients With Implantable Cardioverter-Defibrillators for Primary Prevention of Sudden Cardiac Death

Leah Iles, MBChB, Heinz Pfluger, MD, Lisa Lefkovits, MBBS, Michelle J. Butler, MBBS, Peter M. Kistler, PhD, David M. Kaye, PhD, Andrew J. Taylor, PhD

*Melbourne, Australia*

**Objectives**
The purpose of this study was to evaluate the association between regional myocardial fibrosis and ventricular arrhythmias in patients with cardiomyopathy.

**Background**
Patients with heart failure are at risk of sudden cardiac death (SCD). Current guidelines recommend implantable cardioverter-defibrillator (ICD) devices for a subgroup based on impaired left ventricular function. A significant proportion of devices never discharge, hence a more accurate method for targeting those at risk is desirable.

**Methods**
We prospectively enrolled 103 patients meeting criteria for ICD implantation for primary prevention of SCD. Cardiac magnetic resonance imaging was performed before device implantation. Regional fibrosis was identified with late gadolinium enhancement (LGE).

**Results**
Median follow-up was 573 days (interquartile range: 379 to 863 days). The LGE identified regional fibrosis in 31 of 61 (51%) patients with nonischemic cardiomyopathy (NICM) and in all 42 patients with ischemic cardiomyopathy (ICM). There was a 29% (9 of 31) discharge rate in the NICM group with LGE compared with a 14% (6 of 42) discharge rate in the ICM group (p = NS). There were no ICD discharges in the NICM group without LGE, which was significantly lower than the rate observed in both the ICM patients (p = 0.04) and the NICM patients with LGE (p < 0.01). Left ventricular ejection fraction was similar in patients with and without device therapy (24 ± 12% vs. 26 ± 8%, p = NS) and those with or without LGE (25 ± 9% vs. 26 ± 9%, p = NS).

**Conclusions**
Patients with advanced cardiomyopathy and myocardial fibrosis demonstrated by LGE on cardiac magnetic resonance imaging have a high likelihood of appropriate ICD therapy. Correspondingly, absence of LGE may indicate a lower risk for malignant ventricular arrhythmias. (*J Am Coll Cardiol* 2011;57:821–8) © 2011 by the American College of Cardiology Foundation
Figure 4  Event-Free Survival

Kaplan-Meier analysis using the composite end point of appropriate device therapy, all-cause mortality, and cardiac transplantation for patients with ischemic cardiomyopathy (ICM) (green line), nonischemic cardiomyopathy (NICM) with (NICM+) (red line) and without (NICM−) (blue line) late gadolinium enhancement (LGE) on cardiac magnetic resonance imaging shows improved event-free survival for patients without LGE (p < 0.01).
TC multidetector
Mortalidad en individuos sin enfermedad coronaria conocida pero con discordancia entre el puntuaje de riesgo Framingham y el calcio coronario

Comparación riesgo en caso discordancia FRCV y SC

730
Sujetos asintomáticos
Seguidos 4 años

Estudio FRCV

Estudio SC

Riesgo alto según FR pero SC cero
Riesgo bajo según FR pero SC >100

Ahmadi et al. Am J Cardiol 2011; 107-799-804
Comparación TC 64x2 frente Coronariografía e IVUS

Table 1
Comparison of DSCT, IVUS and QCA for quantification of stenosis: The highest correlation was found for the minimal lumen area (MLA) between DSCT and IVUS (r = 0.90).

<table>
<thead>
<tr>
<th>Methods</th>
<th>% diameter stenosis</th>
<th>% area stenosis</th>
<th>MLD (mm)</th>
<th>MLA (mm²)</th>
<th>CSA (mm²)</th>
<th>% total vessel stenosis (MLA/CSA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>DSCT vs. IVUS</td>
<td>0.69</td>
<td>0.73</td>
<td>0.78</td>
<td>0.90</td>
<td>0.67</td>
<td>0.57</td>
</tr>
<tr>
<td>DSCT vs. QCA</td>
<td>0.60</td>
<td>0.60</td>
<td>0.59</td>
<td>0.59</td>
<td>0.59</td>
<td>0.57</td>
</tr>
<tr>
<td>QCA vs. IVUS</td>
<td>0.83</td>
<td>&lt;0.001</td>
<td>0.02</td>
<td>0.02</td>
<td>0.02</td>
<td>0.02</td>
</tr>
<tr>
<td>DSCT vs. QCA</td>
<td>0.73</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>DSCT vs. IVUS</td>
<td>0.78</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

10% de más
Mejor correlación
Con IVUS que QCA

Feuchtnet et al. Eur J Radiol 2011, in press
Comparison of In Vivo Assessment of Vulnerable Plaque by 64-Slice Multislice Computed Tomography Versus Optical Coherence Tomography

### Datos CT asociados placa vulnerable en OCT

<table>
<thead>
<tr>
<th>Pacientes con TC</th>
<th>Lesiones con pared fina (&lt;65 μm)</th>
<th>KT</th>
<th>OCT</th>
<th>Pacientes</th>
<th>CT densidad &lt; 60 HU</th>
<th>Remodelado positivo &lt; 1.08</th>
<th>Forma anillo</th>
</tr>
</thead>
<tbody>
<tr>
<td>3,320</td>
<td>122</td>
<td>528</td>
<td>90</td>
<td>81</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Ito et al. Am J Cardiol 2011; 107:1270-1277
Diagnosis of Ischemia-Causing Coronary Stenoses by Noninvasive Fractional Flow Reserve Computed From Coronary Computed Tomographic Angiograms

Results From the Prospective Multicenter DISCOVER-FLOW (Diagnosis of Ischemia-Causing Stenoses Obtained Via Noninvasive Fractional Flow Reserve) Study

Bon-Kwon Koo, MD, PhD,* Andrejs Erglis, MD, PhD,† Joon-Hyung Doh, MD, PhD,*‡ David V. Daniels, MD,§ Sanda Jegere, MD,∥ Hyo-Soo Kim, MD, PhD,* Allison Dunning, MD,¶ Tony DeFrance, MD,# Alexandra Lansky, MD,** Jonathan Leipsic, BSc, MD,†† James K. Min, MD‡‡ Seoul and Goyang, South Korea; Riga, Latvia; Palo Alto, San Francisco, and Los Angeles, California; New York, New York; New Haven, Connecticut; and Vancouver, British Columbia, Canada

Objectives

The aim of this study was to determine the diagnostic performance of a new method for quantifying fractional flow reserve (FFR) with computational fluid dynamics (CFD) applied to coronary computed tomography angiography (CCTA) data in patients with suspected or known coronary artery disease (CAD).

Background

Measurement of FFR during invasive coronary angiography is the gold standard for identifying coronary artery lesions that cause ischemia and improves clinical decision-making for revascularization. Computation of FFR from CCTA data (FFR_{CCTA}) provides a noninvasive method for identifying ischemia-causing stenosis; however, the diagnostic performance of this new method is unknown.

Methods

Computation of FFR from CCTA data was performed on 159 vessels in 103 patients undergoing CCTA, invasive coronary angiography, and FFR. Independent core laboratories determined FFR_{CCTA} and CAD stenosis severity by CCTA. Ischemia was defined by an FFR_{CCTA} of FFR ≤0.80, and anatomically obstructive CAD was defined as a CCTA with stenosis >50%. Diagnostic performance of FFR_{CCTA} and CCTA stenosis was assessed with invasive FFR as the reference standard.

Results

Fifty-six percent of patients had ≥1 vessel with FFR ≤0.80. On a per-vessel basis, the accuracy, sensitivity, specificity, positive predictive value, and negative predictive value were 84.3%, 87.9%, 82.2%, 73.9%, and 92.2%, respectively, for FFR_{CCTA} and were 58.5%, 91.4%, 39.6%, 46.5%, 88.9%, respectively, for CCTA stenosis. The area under the receiver-operator characteristics curve was 0.50 for FFR_{CCTA} and 0.75 for CCTA (p = 0.001). The FFR_{CCTA} and FFR were well correlated (r = 0.717, p < 0.001) with a slight underestimation by FFR_{CCTA} (0.022 ± 0.116, p = 0.016).

Conclusions

Noninvasive FFR derived from CCTA is a novel method with high diagnostic performance for the detection and exclusion of coronary lesions that cause ischemia. (The Diagnosis of Ischemia-Causing Stenoses Obtained Via Noninvasive Fractional Flow Reserve; NCT01189331) (J Am Coll Cardiol 2011:58:1989–97) © 2011 by the American College of Cardiology Foundation

* Department of Radiology, Seoul National University College of Medicine, Seoul, South Korea

† Department of Cardiology, Seoul National University College of Medicine, Seoul, South Korea

‡ Department of Radiology, Seoul National University College of Medicine, Seoul, South Korea

§ Department of Radiology, Riga Latvia University College of Medicine, Riga, Latvia

∥ Department of Radiology, San Francisco Veterans Affairs Medical Center, San Francisco, California

¶ Department of Radiology, Los Angeles VA Medical Center, Los Angeles, California

# Department of Cardiology, New York Presbyterian Hospital, New York, New York

** Department of Cardiology, Yale University School of Medicine, New Haven, Connecticut

†† Department of Cardiology, University of British Columbia, Vancouver, British Columbia, Canada

‡‡ Department of Radiology, Seoul National University College of Medicine, Seoul, South Korea
### Table 2: Diagnostic Performance of FFR\textsubscript{CT} and CTA on a Per-Vessel and Per-Patient Basis

<table>
<thead>
<tr>
<th>Measure</th>
<th>FFR\textsubscript{CT} ≤0.80 (95% CI)</th>
<th>CCTA Stenosis ≥50% (95% CI)</th>
<th>FFR\textsubscript{CT} ≤0.80 (95% CI)</th>
<th>CCTA Stenosis ≥50% (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accuracy</td>
<td>84.3 (77.7–90.0)</td>
<td>58.5 (50.4–66.2)</td>
<td>87.4 (79.4–93.1)</td>
<td>61.2 (51.1–70.6)</td>
</tr>
<tr>
<td>Sensitivity</td>
<td>87.9 (76.7–95.0)</td>
<td>91.4 (81.0–97.1)</td>
<td>92.6 (82.1–97.9)</td>
<td>94.4 (84.6–98.8)</td>
</tr>
<tr>
<td>Specificity</td>
<td>82.2 (73.3–89.1)</td>
<td>39.6 (30.0–49.8)</td>
<td>81.6 (68.0–91.2)</td>
<td>24.5 (13.3–38.9)</td>
</tr>
<tr>
<td>PPV</td>
<td>73.9 (61.9–83.7)</td>
<td>46.5 (37.1–56.1)</td>
<td>84.7 (73.0–92.8)</td>
<td>58.0 (47.0–68.4)</td>
</tr>
<tr>
<td>NPV</td>
<td>92.2 (84.6–96.8)</td>
<td>88.9 (75.9–96.3)</td>
<td>90.9 (78.3–97.5)</td>
<td>80.0 (51.9–95.7)</td>
</tr>
<tr>
<td>LR (+)</td>
<td>4.94 (3.54–6.89)</td>
<td>1.51 (1.33–1.73)</td>
<td>5.03 (3.34–7.59)</td>
<td>1.25 (1.11–1.41)</td>
</tr>
</tbody>
</table>

### Figure 3: ROC Demonstrating the AUC for FFR\textsubscript{CT} and CTA Stenosis for the Discrimination of Lesions That Cause Ischemia on a Per-Vessel and Per-Patient Level

Areas under the receiver-operator characteristics curve (AUC) on a per-patient (right) and per-vessel (left) level for ischemia by fractional flow reserve ≤0.80 by coronary computed tomography angiography (CTA) stenosis ≥50% or computation of fractional flow reserve from coronary computed tomographic angiography data (FFR\textsubscript{CT}) ≤0.80. ROC = receiver-operator characteristic.
A Prospective Study for Comparison of MR and CT Imaging for Detection of Coronary Artery Stenosis

Estudio Coronarias con RMN y CT frente a coronariografía

Hamdam et al. J Am Coll Cardiol Img 2011; 4: 50-61
Estudio Coronarias con RMN y CT frente a coronariografía

120 pacientes
- Sospecha enfermedad coronaria
- Dolor torácico
- Indicación clínica de cateterismo
- Edad superior a 50 años

Exclusión
- Pacientes inestables
- Síndrome coronario agudo
- Fibrilación auricular
- Obesidad mórbida
- MRI: Marcasos/Claustrofobia
- CT: Insuf renal/Alergia contraste

CT 64
- 14 segundos
- 5 no se puede hacer
- 3 no se puede interpretar
- S 90%
- E 83%
- VPP 88%
- VPN 87%
- Radiación 15-20 mSv

3T
- 17 minutos
- 5 no se puede hacer
- 5 no se puede interpretar
- S 87%
- E 77%
- VPP 83%
- VPN 82%
- Radiación CERO

Hamdam et al. J Am Coll Cardiol Img 2011; 4: 50-61
Age- and Sex-Related Differences in All-Cause Mortality Risk Based on Coronary Computed Tomography Angiography Findings

Results From the International Multicenter CONFIRM (Coronary CT Angiography Evaluation for Clinical Outcomes: An International Multicenter Registry) of 23,854 Patients Without Known Coronary Artery Disease

James K. Min, MD,* Allison Dunning, MS,‡ Fay Y. Lin, MD,† Stephan Achenbach, MD,§ Mouaz Al-Mallah, MD,¶ Matthew J. Budoff, MD,¶ Filippo Cademartiri, MD,# Tracy Q. Callister, MD,** Hyuk-Jae Chang, MD,†† Victor Cheng, MD,‡‡ Kavitha Chinnaiyan, MD,§§ Benjamin J. W. Chow, MD,|| Augustin Delago, MD,¶¶ Martin Hadamitzky, MD,## Joerg Hausleiter, MD,### Philipp Kaufmann, MD,*** Erica Maffei, MS,# Gilbert Raff, MD,§§ Leslee J. Shaw, PHD,††† Todd Villines, MD,‡‡‡ Daniel S. Berman, MD,‡‡‡ for the CONFIRM Investigators

New York and Albany, New York; Erlangen and Munich, Germany; Detroit and Royal Oaks, Michigan; Los Angeles, California; Parma, Italy; Hendersonville, Tennessee; Seoul, Korea; Ottawa, Ontario, Canada; Zurich, Switzerland; Atlanta, Georgia; and Washington, DC
Objectives
We examined mortality in relation to coronary artery disease (CAD) as assessed by ≥64-detector row coronary computed tomography angiography (CCTA).

Background
Although CCTA has demonstrated high diagnostic performance for detection and exclusion of obstructive CAD, the prognostic findings of CAD by CCTA have not, to date, been examined for age- and sex-specific outcomes.

Methods
We evaluated a consecutive cohort of 24,775 patients undergoing ≥64-detector row CCTA between 2005 and 2009 without known CAD who met inclusion criteria. In these patients, CAD by CCTA was defined as mild (1% to 49% stenosis), moderate (50% to 69% stenosis), or severe (70% to 100% stenosis). The presence of obstructive CAD was judged on a per-patient, per-vessel, and per-segment basis. Time to mortality was modeled using Cox proportional hazards models.

Results
At a 2.3 ± 1.1-year follow-up, 404 deaths had occurred. In risk-adjusted analysis, both per-patient obstructive (hazard ratio [HR]: 2.60; 95% confidence interval [CI]: 1.94 to 3.49; p < 0.0001) and nonobstructive (HR: 1.60; 95% CI: 1.18 to 2.16; p = 0.002) CAD conferred increased risk of mortality compared with patients without evidence of CAD. Incident mortality was associated with a dose-response relationship to the number of coronary vessels exhibiting obstructive CAD, with increasing risk observed for nonobstructive (HR: 1.62; 95% CI: 1.20 to 2.19; p = 0.002), obstructive 1-vessel (HR: 2.00; 95% CI: 1.43 to 2.82; p < 0.0001), 2-vessel (HR: 2.92; 95% CI: 2.00 to 4.25; p < 0.0001), or 3-vessel or left main (HR: 3.70; 95% CI: 2.58 to 5.29; p < 0.0001) CAD. Importantly, the absence of CAD by CCTA was associated with a low rate of incident death (annualized death rate: 0.28%).

When stratified by age <65 years versus ≥65 years, younger patients experienced higher hazards for death for 2-vessel (HR: 4.00; 95% CI: 2.16 to 7.40; p < 0.0001 vs. HR: 2.46; 95% CI: 1.51 to 4.02; p = 0.0003) and 3-vessel (HR: 6.19; 95% CI: 3.43 to 11.2; p < 0.0001 vs. HR: 3.10; 95% CI: 1.95 to 4.92; p < 0.0001) CAD. The relative hazard for 3-vessel CAD (HR: 4.21; 95% CI: 2.47 to 7.18; p < 0.0001 vs. HR: 3.27; 95% CI: 1.96 to 5.45; p < 0.0001) was higher for women as compared with men.

Conclusions
Among individuals without known CAD, nonobstructive and obstructive CAD by CCTA are associated with higher rates of mortality, with risk profiles differing for age and sex. Importantly, absence of CAD is associated with a very favorable prognosis. (J Am Coll Cardiol 2011;58:849–60) © 2011 by the American College of Cardiology Foundation.
Figure 2  Unadjusted All-Cause 3-Year Kaplan-Meier Survival by the Presence, Extent, and Severity of CAD by CCTA

Note the dose-response relationship of mortality to increasing numbers of vessels with obstructive coronary artery disease (CAD).

CCTA = coronary computed tomography angiography.
Detection of Coronary Artery Stenoses by Low-Dose, Prospectively ECG-Triggered, High-Pitch Spiral Coronary CT Angiography

75 pacientes

Estudio con DOBLE TUBO
2x128

β agresivo para FC<60
5-30 mg metoprolol iv

Coronariografía

16 Pacientes S 100% E 82%
con lesiones VPP 72% VPN 100%

Estudios diagnósticos

0.76 ± 0.08 mSv

50 <60 lpm

Achenbach et al J Am Coll Cardiol Img 2011; 4:328-37