Background: The aim of the present study was to analyze epicardial (EPI) and endocardial (ENDO) strain (S) in patients with transthyretin-related cardiac amyloidosis (TTR-CA) and hypertrophic cardiomyopathy (HCM) using echocardiography (TTE) with 2-dimensional feature tracking imaging (FTI).

Methods and Results: Thirty-three subjects (11 with HCM, 11 with TTR-CA, and 11 healthy subjects as controls) with a New York Heart Association functional class ≤ II underwent conventional TTE and FTI. TTE was used for the evaluation of left ventricle (LV) wall thickness, mass, systolic and diastolic function. FTI was used for the evaluation of EPI and ENDO longitudinal, and circumferential, and radial S. LV wall thickness and mass were higher in both TTR-CA and HCM in comparison with controls (P<0.001), but ejection fraction (EF) was similar among patients with TTR-CA, HCM and controls (63±6%, 64±6%, 61±5%, respectively). ENDO and EPI longitudinal and circumferential S and radial S were significantly lower in HCM and TTR-CA when compared with controls (P<0.01). No differences in EPI and ENDO longitudinal S, ENDO circumferential S and radial S were found between TTR-CA and HCM groups, while EPI circumferential S was significantly lower in the TTR-CA group (6±3.3%) than in the HCM group (8.1±4.3%; P<0.0001).

Conclusions: Longitudinal, circumferential and radial LV deformations are impaired in patients with TTR-CA and HCM with a preserved EF. Impairment of EPI circumferential strain is greater in TTR-CA than in HCM. (Circ J 2011; 75: 1200–1208)

Key Words: Cardiac amyloidosis; 2-D Feature strain echocardiography; Hypertrophic cardiomyopathy; Myocardial strain

Cardiac amyloidosis (CA) and hypertrophic cardiomyopathy (HCM) are 2 known causes of ventricular thickening, due to interstitial infiltration in CA and myocardial cell hypertrophy with disarray in HCM. They are difficult to distinguish on conventional echocardiography, sharing similar signs, such as concentric parietal thickening, small left ventricle (LV) volumes, atrial dilatation and diastolic dysfunction.

Myocardial strain echocardiography derived from tissue Doppler imaging (TDI) or from 2-dimensional (D) images permits quantification of longitudinal, radial and circumferential myocardial deformations of LV. 2-D myocardial strain echocardiography has shown a high level of accuracy in detecting early impairment of systolic function in many cardiovascular diseases. Furthermore, strain imaging abnormalities have been identified in hypertensive cardiomyopathy, athlete’s heart, CA and HCM.

New 2-D imaging-based echocardiographic methods have been recently introduced in myocardial deformation quantification imaging to overcome the well-known limitations of the Doppler-derived measurements due to the angle dependency. Among them, 2 of the most widely used 2-D-imaging-based algorithms for the quantification of the regional and local myocardial deformation are speckle tracking imaging (STI), and feature tracking imaging (FTI).

FTI technology has an interesting technical advantage.
Myocardial Deformations in Amyloidosis and HCM compared to standard STI technology in that it has a smaller region of interest and combines ultrasound speckle with border tracking, thereby enabling the tracking of endocardial (ENDO) and epicardial (EPI) motion separately. This capability allows independent quantification of myocardial deformation in ENDO and in EPI layers of LV, providing the opportunity to evaluate and estimate the physiological ENDO to EPI functional gradient.

Previous studies using STI have shown impaired longitudinal, circumferential and radial myocardial deformations in symptomatic patients with CA as well as in patients with HCM. To the best of our knowledge, however, no previous study has analyzed the ENDO and EPI deformations of LV in patients with CA and HCM. The aim of the present study was therefore to analyze EPI and ENDO myocardial deformations, and their differences, in patients with CA and HCM with none–mild impairment of cardiac functional status.

**Methods**

**Patients**

Between September 2008 and March 2010, 35 consecutive patients with HCM (LV thickness ≥15 mm) and 16 consecutive patients with familial transthyretin-related CA (TTR-CA; LV thickness ≥15 mm and positive genetic test for transthyretin [TTR] gene mutation) were considered for enrollment in the present study; 29 patients who had at least 1 of the following exclusion criteria were excluded: arterial hypertension (14 patients), LV outflow obstruction (8 patients), significant valve disease (3 patients), exclusively apical hypertrophy (1 patient), previous myocardial infarction (2 patients), significant atrial and ventricular arrhythmias (5 patients), EF<50% (3 patients), and New York Heart Association (NYHA) functional class >II (6 patients). In addition, 11 gender- and age-matched healthy subjects were enrolled in the study as a control group.

Thus, the final population consisted of 33 subjects with preserved EF: 11 patients with HCM (8 of whom had asymmetric septal HCM and 3, symmetrical HCM), 11 patients with familial TTR-CA and 11 healthy subjects as the control group. All subjects underwent conventional and FTI echocardiographic study in the same examination. Clinical and instrumental patient data were collected at the time of examination. The local ethics review committee approved the study and the investigation conformed to the principles outlined in the Declaration of Helsinki. Written informed consent was obtained from all subjects.

**Scintigraphy Data Acquisition and Analysis**

Patients with mutations of the TTR gene underwent $^{99m}$Tc-DPD scintigraphy to ensure cardiac deposition of TTR and to evaluate its distribution, no more than 1 month before/after echocardiography.

Thorax planar scans (anterior and posterior projections) and thoracic single-photon emission computed tomography were obtained 3h after the i.v. injection of 740MBq $^{99m}$Tc-DPD using a dual-headed gamma camera (Odyssey; Picker International). The $^{99m}$Tc-DPD examinations were visually categorized by an experienced observer as: negative (no cardiac localization of the radiotracer) or positive (cardiac localization of the radiotracer). When positive, $^{99m}$Tc-DPD accumulation was categorized as diffuse (if the entire LV was recognizable)
or not diffuse.

2-D Echocardiographic Data Acquisition and Analysis
Echocardiographic images were obtained using a commercial ultrasound machine (My Lab 30 gold, Esaote, Florence, Italy), equipped with a 2.5-MHz phased array transducer. Parasternal long axis view, parasternal short axis views at the basal, middle, and apical levels, and 3 standard apical views (4-chamber, 2-chamber, and 3-chamber long-axis) were acquired. LV end-diastolic volume (EDV, ml), end-systolic volume (ESV, ml), and ejection fraction (EF, %) were calculated using Simpson’s biplane method.

Septal thickness (mm), posterior wall thickness (mm) and LV mass were quantified using the Teicholz method; furthermore, the thickness (mm) of each segment was determined: a segment was considered to be hypertrophic when its thickness was ≥15 mm. A 16-segment model was used to divide the LV.25

LV diastolic function was quantified using the ratio between the E wave velocity of the pulsed wave Doppler mitral flow and the early diastolic velocity of the septum at mitral annulus level (E’ wave) on TDI. The tracking quality was verified either or not diffuse.

<table>
<thead>
<tr>
<th>Table 1. Clinical and Conventional Echocardiographic Subject Characteristics</th>
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<tbody>
<tr>
<td><strong>Control (n=11)</strong></td>
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<tr>
<td>Age (years)</td>
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<tr>
<td>M/F</td>
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<tr>
<td>Septal thickness (mm)</td>
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<tr>
<td>Posterior wall thickness (mm)</td>
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<tr>
<td>LV mass (g)</td>
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<tr>
<td>Hypertrophic segments (%)</td>
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<tr>
<td>EDV (ml)</td>
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<tr>
<td>ESV (ml)</td>
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<td>EF (%)</td>
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</table>

P value, control group vs. HCM group; P value<sup>1</sup>, control group vs. TTR-CA group; P value<sup>2</sup>, HCM group vs. TTR-CA group.

HCM, hypertrophic cardiomyopathy; TTR-CA, transthyretin-related cardiac amyloidosis; LV, left ventricle; EDV, end-diastolic volume; ESV, end-systolic volume; EF, ejection fraction.

Continuously variables are expressed as mean±1SD and cate-

and ENDO circumferential and radial strain (%) as well as radial strain (%), we recorded digitalized 2-D video clips from parasternal short axis views at basal, mid- and apical LV levels. From each short axis view we obtained 3 strain values (ENDO and EPI circumferential strain and radial strain) for each segment in the corresponding level. Basal, mid-LV and apical strains (ENDO and EPI circumferential strain and radial strain) were obtained by averaging the corresponding strain values of all segments at each level. Mean LV ENDO and EPI circumferential strain as well as mean LV radial strain were obtained by averaging the corresponding strain values of all 16 segments.

Video clips from apical views (4-chamber, 2-chamber and 3-chamber long-axis) were recorded for the evaluation of EPI and ENDO longitudinal strain (%).28 ENDO and EPI longitudinal strain values for each segment were obtained from apical views. Basal, mid-LV and apical ENDO longitudinal strain were obtained by averaging the corresponding strain values of all segments at each level. Basal, mid-LV and apical EPI longitudinal strain were obtained in a similar manner. Mean LV ENDO and EPI longitudinal strain were obtained by averaging the corresponding strain values of all 16 segments.

Longitudinal, circumferential and radial strain in HCM patients, CA patients and controls were compared; when appropriate, cut-offs of strain for optimal sensitivity and specificity in differentiating CA from HCM were determined. Finally, the variability of strain data among segments was evaluated for each patient.

All images used for the analysis of longitudinal, circumferential and radial strain were analyzed off-line by a cardiologist (G.D.B.) and these results were used for statistical analysis; moreover, the 4-chamber view (ENDO and EPI longitudinal strain) and mid-short axis view (ENDO and EPI circumferential strain) of 12 randomly chosen subjects (4 controls, 4 CA and 4 HCM) were evaluated by a second cardiologist (C.Z.), in order to evaluate inter-observer variability, and by G.D.B., 1 month after the first evaluation, to determine intra-observer variability.

**Statistical Analysis**
Continuous variables are expressed as mean±1SD and cate-
Table 2. ENDO and EPI Longitudinal Function

<table>
<thead>
<tr>
<th></th>
<th>Longitudinal strain</th>
<th>Control (n=11)</th>
<th>HCM (n=11)</th>
<th>TTR-CA (n=11)</th>
<th>P value</th>
<th>P value^1</th>
<th>P value^2</th>
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<tbody>
<tr>
<td></td>
<td>Basal LV</td>
<td></td>
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<tr>
<td>ENDO S (%)</td>
<td>–17.4±6</td>
<td>–12.9±7.6</td>
<td>–11.1±4.8</td>
<td>0.002</td>
<td>&lt;0.001</td>
<td>NS</td>
<td></td>
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<tr>
<td>EPI S (%)</td>
<td>–22.7±9</td>
<td>–15.3±8.7</td>
<td>–14.2±5</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>NS</td>
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<tr>
<td></td>
<td>Mid-LV</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>ENDO S (%)</td>
<td>–17.9±5</td>
<td>–13±6.6</td>
<td>–11.4±3.9</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>NS</td>
<td></td>
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<tr>
<td>EPI S (%)</td>
<td>–18.9±7</td>
<td>–11.9±6.9</td>
<td>–11.8±4.1</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>NS</td>
<td></td>
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<td></td>
<td>Apical LV</td>
<td></td>
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<tr>
<td>ENDO S (%)</td>
<td>–19.7±7.7</td>
<td>–17±7.5</td>
<td>–15.7±5.6</td>
<td>NS</td>
<td>0.04</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>EPI S (%)</td>
<td>–13.6±6.1</td>
<td>–8.8±5</td>
<td>–8.8±3.8</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>NS</td>
<td></td>
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</tbody>
</table>

ENDO S, endocardial strain; EPI S, epicardial strain. Other abbreviations see in Table 1.
P value, control group vs. HCM group; P value^1, control group vs. TTR-CA group; P value^2, HCM group vs. TTR-CA group.

Figure 2. Hypertrophic cardiomyopathy (HCM) and transthyretin-related cardiac amyloidosis (TTR-CA): (A) longitudinal endocardial (ENDO) and (B) epicardial (EPI) strain in segments with and without left ventricle hypertrophy.
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gorical variables as percentages. The unpaired t-test was used to assess the differences among HCM, TTR-CA and control groups. Receiver operating characteristic (ROC) curves were constructed, and areas under the curves were measured to determine cut-offs of strain for optimal sensitivity and specificity. Inter-observer and intra-observer variability were assessed using the Bland–Altman method. For any statistical comparison, P<0.05 was considered to be significant. Statistical analyses were performed using SPSS version 12 (SPSS, Chicago, IL, USA) and MedCalc™ 6.00.014 (MedCalc Software, Mariakerke, Belgium).

Results

99mTc-DPD examination demonstrated a diffuse cardiac uptake of the radiotracer in all the patients with TTR-CA.

Clinical Characteristics and Conventional Trans-Thoracic Echocardiographic Parameters

Septal and posterior wall thickness and LV mass were higher in TTR-CA and HCM groups in comparison with controls; in contrast, EDV and ESV were lower in the TTR-CA and HCM groups in comparison with controls (Table 1).

As reported in Table 1, many echocardiographic parameters were similar in the TTR-CA and HCM groups. In contrast, ESV was significantly lower in the TTR-CA than in the HCM group; however, septal thickness was significantly higher in the HCM than in the TTR-CA group, but the prevalence of hypertrophic segments was higher in the TTR-CA than in the HCM group (P<0.0001).

Longitudinal Strain

ENDO longitudinal strain was measured in 480/528 segments (91%), while EPI longitudinal strain was measured in 433/528 segments (82%). The remaining segments were excluded because of poor echocardiographic image quality. When comparing controls, HCM and TTR-CA, we found that mean ENDO and EPI longitudinal strain were significantly lower in the HCM and TTR-CA groups than in the controls (P<0.0001), whereas no difference of mean longitudinal strain was found between the TTR-CA and HCM groups. Analysis of END0 and EPI longitudinal function, performed

<table>
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<th>Table 3. Mean Longitudinal, Circumferential and Radial Strain</th>
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<th>Table 4. Analysis of ENDO and EPI Circumferential Strain and Radial Strain</th>
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<td><img src="image" alt="Table 4" /></td>
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Abbreviations see in Tables 1, 2.
P value, control group vs. HCM group; P value1, control group vs. TTR-CA group; P value2, HCM group vs. TTR-CA group.
separately at basal, mid- and apical LV levels (Table 2), indicated that (1) longitudinal ENDO strain increased in each group from base to apex; (2) longitudinal EPI strain decreased in each group from base to apex; and (3) there were no differences in EPI and ENDO longitudinal strain between the TTR-CA and HCM groups at each LV level.

In more detail, considering the different patterns of deformation in the HCM group, patients with symmetrical HCM had greater impairment of mean ENDO (–9.5±4 vs. –12.2±2) and mean EPI (–7.1±4 vs. –11±6) longitudinal strain than patients with asymmetrical HCM (P=0.03 and P=0.002, respectively). Furthermore, comparing longitudinal strain values of hypertrophic segments with those of non-hypertrophic segments in the HCM group, hypertrophic segments had a lower longitudinal ENDO (–10.2±4 vs. –13.5±6) and EPI (–9.7±4 vs. –11.8±2) strain than those without hypertrophy (P=0.002 and P=0.04, respectively; Figure 2A). When the same analysis is made considering hypertrophic and non-hypertrophic segments separately at basal, mid- and apical LV levels, the only significant difference was seen in impairment of ENDO longitudinal strain in hypertrophic segments (–8.1±3 vs. –12.2±7, P=0.04) at the basal level.

Comparing the longitudinal strain of hypertrophic segments with that of non-hypertrophic segments in the TTR-CA group, only mean EPI longitudinal strain was significantly lower in hypertrophic segments than in non-hypertrophic segments (–11.2±5 vs. –13.3±5, P=0.04; Figure 2B).

**Circumferential Strain**

ENDO circumferential strain was measured in 427/528 segments (81%), while EPI circumferential strain was measured in 391/528 segments (74%). The remaining segments were

![Figure 3.](image-url)
Mean ENDO and EPI circumferential strain were significantly impaired in the HCM and TTR-CA groups in comparison with controls (P<0.0001; Table 3), and mean EPI circumferential strain was significantly lower in the TTR-CA group than in the HCM group (P<0.0001). Analysis of ENDO and EPI circumferential strain, performed separately at basal, mid- and apical LV levels (Table 4) indicated that (1) circumferential ENDO strain increased in each group from base to apex; (2) circumferential ENDO strain was significantly different (lower) between the TTR-CA and controls and between HCM and controls only at mid-LV level; (3) no difference in circumferential ENDO strain was found between TTR-CA and HCM groups; (4) circumferential EPI strain increased, in normal subjects and patients with HCM, from base to apex while it decreased in the same direction in TTR-CA patients; and (5) a significantly lower mid- and apical LV circumferential EPI strain was found in TTR-CA patients than in HCM patients.

Mean EPI circumferential strain had a low–moderate predictive value (area under the ROC curve was 0.65; 95% confidence interval: 0.59–0.71) in distinguishing HCM from CA. As a cut-off, EPI circumferential strain ≤–8.1% yielded the best result in terms of combined sensitivity (46%) and specificity (81%). Moreover, using a mean EPI circumferential strain of ≤–8.1% in >20% of LV segments as a marker for CA, the sensitivity, specificity, positive predictive value and negative predictive value in differentiating CA from HCM were 64%, 82%, 78% and 69%, respectively.

Patients with symmetrical HCM had a greater impairment of mean ENDO (–15.4±5 vs. –20±7, P<0.0001) and mean EPI (–5.3±4 vs. –8.7±4, P<0.0001) circumferential strain than patients with asymmetrical HCM. Furthermore, comparing the circumferential strain of hypertrophic segments with that of non-hypertrophic segments in the HCM group, a greater impairment (P<0.01) of EPI circumferential strain was found in hypertrophic segments (–5.5±4) than in non-hypertrophic segments (–7.8±4; Figure 3B).

When the same analysis was done considering hypertrophic and non-hypertrophic segments separately at the basal, mid- and apical LV levels, the only significant difference was found for lower EPI circumferential strain in hypertrophic segments (–5.8±3 vs. –8±4, P=0.04) at the basal level.

Comparing the circumferential strain of hypertrophic segments with that of non-hypertrophic segments in the TTR-CA group, hypertrophic segments had a lower EPI circumferential strain (–5.6±3 vs. –7.4±5, P=0.04) than non-hypertrophic segments (–6.6±3 vs. –14.2±4, P<0.0001) at the basal level only.

Radial Strain
Radial strain was measured in 389/528 segments (74%). The remaining segments were excluded because of poor echocardiographic image quality. Mean radial strain was significantly lower in the HCM and TTR-CA groups than in controls, whereas no difference in mean radial strain was found between the HCM and TTR-CA groups (Table 3). Analysis of radial strain, performed separately at the basal, mid- and apical LV levels showed that radial strain was lower in the HCM and TTR-CA groups than in the controls only at the mid-LV level (Table 4). No significant difference between radial strain in the HCM and TTR-CA groups was found at each level.

No difference in mean radial strain was found between patients with symmetrical HCM and patients with asymmetrical HCM; in HCM patients, radial strain was lower in hypertrophic segments than in non-hypertrophic segments (17.3±10 vs. 23±13; P=0.04; Figure 4); namely, radial strain was lower in hypertrophic segments than in non-hypertrophic segments (14.9±9 vs. 27±12, P=0.002) at the basal LV level.

In the TTR-CA group, radial strain was lower in hypertrophic segments than in non-hypertrophic segments (30±15 vs. 22.5±14; P=0.02). Considering hypertrophic and non-hypertrophic segments separately at the basal, mid- and apical LV levels, radial strain was lower in hypertrophic segments than in non-hypertrophic segments only at the mid-LV level (40±18 vs. 25±12, P=0.005).
Reproducibility
There was good inter-observer and intra-observer agreement concerning longitudinal ENDO strain (−1.3±22.3% and 1.4±17.4%, respectively), and longitudinal EPI strain (−1.9±5.8% and −1.7±7.2%, respectively), circumferential ENDO strain (−1.8±12.4% and −1.4±11.4%, respectively) and circumferential EPI strain (−2.8±16.3% and −2.4±14.2%, respectively).

Discussion
The main findings can be summarized as follows: (1) LV hypertrophy due to HCM and TTR-CA results in an impairment of LV deformation, particularly in the EPI layer; (2) longitudinal, radial and circumferential LV strains were lower in TTR-CA and in HCM than in controls; and (3) a greater dysfunction of circumferential EPI strain occurs in TTR-CA than in HCM, namely at the mid- and apical LV levels.

Interestingly, these results were obtained in patients without overt regional contractile dysfunction and with normal EF, highlighting the evidence that alterations in myocardial strain are earlier, preceding contractile abnormalities both in TTR-CA and in HCM patients.32,33

In HCM patients, a heterogeneous reduction in longitudinal strain has also been documented on magnetic resonance imaging tagging34 and, similarly, an early impairment of longitudinal strain, despite normal standard 2-D echocardiographic parameters and normal diastolic function, has been shown in patients with CA.30,31 Longitudinal EPI and ENDO strains were similar in TTR-CA and HCM, whereas a greater dysfunction of circumferential EPI strain was measured in TTR-CA compared to HCM patients. When a cut-off of ≤−8.1% in >20% of LV segments, for mean circumferential EPI strain was used, the sensitivity for CA was 64% and the specificity was 82%. Therefore, mean circumferential EPI strain may be considered a potential new echocardiographic parameter for distinguishing CA and HCM, 2 different entities that share conventional echocardiographic findings, such as ventricular symmetric thickening with tissue granular sparkling, inter-atrial septal thickening, biaxial dilatation, pericardial effusion and a restrictive pattern of diastolic function.

We think that the greater reduction in circumferential EPI strain in CA than in HCM could be due to the diffuse transmural myocardial deposition, mainly perivasculare, of amyloid material,32,33 in contrast to patchy and mainly subendocardial disarray occurring in HCM; this hypothesis can be confirmed, in the present series, by the diffuse 99mTc-DPD cardiac accumulation, reflecting amyloid deposition, that was found in all CA patients. This scintigraphic evidence, indeed, fits with the higher number of hypertrophic segments in TTR-CA than in HCM patients, although the degree of wall thickness in hypertrophic segments was higher in HCM than in TTR-CA.

In contrast, interstitial fibrosis in HCM is patchy and regionally distributed in the hypertrophic segments.34 Interestingly, regional non-uniformities in peak systolic strain have been reported in HCM patients and, more importantly, it has been demonstrated that their localization matches areas of delayed magnetic resonance enhancement due to fibrosis.35–37 Accordingly, in the present study, ENDO and EPI circumferential and longitudinal strain measurements were more heterogeneously distributed in HCM than in CA patients.

The present results are different from those of Sun et al, who, using STI echocardiography, reported that CA had lower values of longitudinal, circumferential and radial deformations than HCM.22 These differences can be explained as follows. First, the present series included patients with early phase CA with preserved LVEF and an NYHA functional class ≤II; moreover, LVEF was similar to that of HCM patients (63% vs. 64% respectively), whereas in the Sun et al. series the CA subjects had a much lower LVEF (39±10%), which was different from that of HCM patients (57±9%). Second, we quantified EPI and ENDO function using an FTI algorithm whereas in the Sun et al study an STI algorithm was used. As aforementioned, STI utilizes a larger region of interest traced on myocardial tissue in determining the displacement of speckles in relation to each other,10,11 whereas FTI uses a smaller region of interest (3–4 pixels) to track ENDO as well as EPI motion through a combination of tracking of ultrasound speckle, myocardial blood interface, and myocardial shape.12,13

Study Limitations
The major limitations of the present study were the small number of subjects and the lack of biopsy specimens available for histopathological analysis, useful for correlating imaging to histopathological findings. Because endomyocardial biopsy is invasive, however, it was not appropriate in the asymptomatic patients with preserved systolic function.

Conclusion
Myocardial deformation is impaired in LV hypertrophy due to HCM and TTR-CA. Longitudinal, circumferential and radial deformations are impaired in CA and HCM with preserved global systolic function. EPI circumferential strain is more impaired in CA than in HCM, and it can be considered as a further echocardiographic marker of early phase CA. These findings suggest that FTI echocardiography provides, in addition to traditional echocardiographic findings, a helpful tool in distinguishing LV thickening due to CA or HCM. Further studies involving a larger series of patients with CA and HCM are needed to confirm the present data.

Acknowledgment
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Disclosure
All the authors declare no conflict of interest.

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