Myocardial contraction properties along the long and short axes of the left ventricle in isolated left ventricular non-compaction: pulsed tissue Doppler echocardiography

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Aim Our objective was to evaluate regional systolic myocardial contraction properties along the long and short axes of the left ventricle (LV) in patients with isolated LV non-compaction (IVNC). 

Methods and results Pulsed tissue Doppler imaging (TDI) was used to record myocardial velocities along these axes in 25 patients with IVNC (10 asymptomatic–LV ejection fraction [LVEF] ≥ 50%; 15 symptomatic–LVEF < 50%) and 15 healthy controls. In all cases, the systolic velocity pattern featured 2 distinct peaks (SW1, SW2). These peak velocities and the intervals from the electrocardiographic Q wave to each peak (Q-SW1, Q-SW2) were recorded for each axis, and group means were calculated. The asymptomatic group displayed significantly higher long axis SW2 and significantly longer long axis Q-SW1 than the controls. The symptomatic group had significantly lower SW1 and SW2 on both axes and significantly longer short axis Q-SW1 than the asymptomatic group and the controls. Long axis Q-SW1 and short axis Q-SW1 in the symptomatic group were significantly longer than the corresponding control findings, but were not significantly different from the corresponding asymptomatic group findings.

Conclusion In conclusion, patients with IVNC exhibit distinct systolic myocardial shortening velocities along the long and short axes of the LV. Further, these pulsed TDI findings suggest that asymptomatic patients (those with normal LVEF) have subclinical myocardial dysfunction.

KEYWORDS Tissue Doppler imaging; Left ventricular non-compaction

Introduction

Isolated left ventricular non-compaction (IVNC) is a rare congenital form of cardiomyopathy. Myocardial non-compaction occurs when the early, normal intrauterine process of endomyocardial morphogenesis is arrested, resulting in abnormal segmental hypertrabeculation (spongy myocardium) of the left ventricle (LV), and particularly at the apex. The main causes of mortality and morbidity in this patient group are heart failure (HF), embolic events and lethal arrhythmias. Although HF is common in the setting of IVNC, this condition is not always fatal; patients can recover LV systolic function for variable periods of time before they deteriorate further. Reports state that 17–40% of patients with IVNC have preserved LV systolic function at the time of initial diagnosis. Previous studies have shown that abnormal myocardial contraction in non-compacted regions, microcirculatory dysfunction, and contraction abnormalities along the long and short axes of the LV are 3 factors that might be involved.

Normal LV myocardium comprises 2 main categories of contractile fibers. One type runs circumferentially in the mid-wall, and the second type runs longitudinally in the sub-endocardial and subepicardial layers of the entire LV wall from base to apex. LV ejection results from coordinated contraction of these fibers along the long and short axes of the LV. Abnormal myocardial contraction along these axes leads to distinct disturbances in various cardiac disorders. Pulsed tissue Doppler imaging (TDI) has proven valuable for evaluating myocardial shortening properties along the long and short axes of the LV in healthy subjects and those with cardiac disease. However, no study to date has investigated these specific properties in patients with IVNC. Our purpose was to assess systolic myocardial
shortening properties along the long and short axes of the LV using pulsed TDI in this patient group, and to consider the clinical significance of the findings.

Methods

Study population

Twenty-five consecutive, eligible (criteria defined below) adults with IVNC (13 men and 12 women; mean age, 30.5 ± 11.2 years) and 15 healthy hospital-staff volunteers (controls; 8 men and 7 women; mean age, 38 ± 14 years) were enrolled in the study. The groups were matched for age and sex distribution, and the subgroup with asymptomatic IVNC (defined below) and the control group were also matched for echocardiographically determined LV chamber size. None of the controls had cardiovascular symptoms or evidence of any systemic disease, as assessed by physical examination, chest radiography, electrocardiography (ECG), and echocardiography.

The diagnostic criteria for IVNC were as follows: (1) absence of coexisting cardiac anomalies; (2) presence of excessive numbers of large trabeculae; (3) multiple deep intertrabecular recesses filled with blood from the ventricular cavity, as demonstrated by color Doppler imaging; and (4) ratio of the thickness of the non-compacted endocardial layer to that of the compacted epicardial layer (NC/C ratio) ≥ 2 (Figure 1).9 Individuals with IVNC were excluded from the study if they had 1 or more of the following: rhythm other than sinus, complete bundle branch block, any pre-excitation syndrome, significant valvular regurgitation or valvular stenosis, any systemic disease (diabetes, hypertension, goiter, or others), neuromuscular disease at time of presentation, clinical and ECG evidence of ischemic heart disease, history of cardiotoxic agent use (chemotherapeutics or long-term alcohol consumption), and hypertrabeculation on the site that the sample gate of the pulsed TDI would be placed.

For analysis, the 25 patients with IVNC were divided into 2 subgroups: an asymptomatic group (n = 10; 5 men and 5 women; mean age 39 ± 11 years; New York Heart Association [NYHA] functional class I or IIa; LV ejection fraction [LVEF] ≥ 50%) and a symptomatic group (n = 15; 8 men and 7 women; mean age 41.2 ± 12.4 years; NYHA functional class > IIa; LVEF < 50%). The 10 asymptomatic patients exhibited no clinical evidence of HF (8 cases diagnosed during family screening, 2 diagnosed incidentally during echocardiography for other reasons). The 15 symptomatic patients had clinical HF and each had been hospitalized at least once for decompensated HF. These individuals (symptomatic group) were all taking appropriate medical therapy, including diuretics, β-blockers, digitalis, anticoagulants and angiotensin-converting enzyme inhibitors. Patients who were on medications that affect LV performance (β-blockers or digoxin) had these drugs withdrawn at least 4 times each agent’s half-life before the echocardiographic examinations were done.

All participants were informed about the study and each gave written consent to participate.

Transthoracic echocardiography

Each subject was examined with conventional transthoracic 2-dimensional echocardiography and color Doppler echocardiography. Echocardiographic examinations were done with the individual in left lateral decubitus position and in a calm state, breathing spontaneously. Each set of exams was done using the same commercially available echocardiography system (GE Vivid 7 Pro, Horten, Norway) with a 1.5–3.6 MHz multi-frequency phased array probe. Parasternal and apical windows were used for conventional M-mode, 2-dimensional and color Doppler imaging. LV end-diastolic diameter (LVEDD), LV end-systolic diameter (LVESD), thickness of the interventricular septum during diastole, thickness of the posterior wall of the LV during diastole, and maximum left atrial diameter (LAD)

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Figure 1  Modified apical view of the left ventricular apex in a patient with isolated left ventricular non-compaction. Two-dimensional echocardiography reveals the 2 layers of myocardium (compacted and non-compacted), excessive numbers of large trabeculae, and multiple deep intertrabecular recesses filled with blood from the ventricular cavity, as shown on color Doppler.
were measured from M-mode tracings according to the recommendations of The American Society of Echocardiography.

For each patient with IVNC, we recorded the number of regions/segments exhibiting noncompaction, the specific locations of these, and the NC/C ratio. As noted, the NC/C ratio was calculated based on the thickness of the 2 layers of myocardium (compacted and non-compacted). These measurements were made at the site of the most prominent trabecular meshwork in end-systole as this allowed best visualization of the layers. The locations/sites of non-compaction were recorded by dividing the LV wall into 16 regions/segments: the inferior wall, lateral wall, anterior wall, and septum on the short-axis apical view; the anterior septum, posterior septum, and the posterior, inferior, lateral and anterior LV walls on the short-axis mid-ventricular view and the short-axis basal view.

LV systolic function was assessed by calculating ejection fraction and fractional shortening (FS). LVEF was calculated from apical views using the biplane area length method. FS was calculated from M-mode tracings of the parasternal long axis using the formula, FS (%) = ([LVEDD - LVESD] ÷ LVEDD) × 100.

**Pulsed TDI**

Pulsed TDI recordings of systolic myocardial velocities along the long and short axes of the LV were obtained as previously described.26,27 Pulsed TDI was performed using a presenting program on the same echocardiography system that was used for transthoracic echocardiography. ECG tracings were obtained simultaneously. The pulsed TDI recordings were obtained through the apical and parasternal windows. To record velocities along the short axis, the sample volume was placed in the mid-portion of the middle region of the posterior LV wall as seen on a parasternal long-axis view. To record velocities along the long axis, the sample volume was placed in the subendocardial portion of the middle region of the posterior LV wall as seen on an apical long-axis view. To avoid angle correction, the transducer was aligned as close to parallel as possible to the segment of interest. Meticulous care was taken to place the sample volume in normal myocardial segments (those without non-compaction). The imaging depth was set at 16–18 cm with a sector angle of less than 60 degrees. The sweep rate was set at 100 mm/s and the width of the sample gate was 5 mm. Velocities were measured in centimeters per second, and time intervals in milliseconds.

In each case, pulsed-TDI showed 2 separate peaks in the systolic myocardial velocity patterns (Figure 2). The first peak (SW1) was recorded as the first systolic myocardial velocity and the second peak (SW2) was recorded as the second systolic myocardial velocity.

Peak velocities were measured in centimeters per second, and the intervals from the electrocardiographic Q wave to SW1 (QSW1) and SW2 (QSW2) were recorded in milliseconds.

All transthoracic echocardiographic and pulsed TDI examinations were conducted by the same observer (O.T.) and the mean parameter values calculated from 3 consecutive heartbeats were used.

**Statistical analysis**

Descriptive data for continuous variables are presented as mean ± 1 standard deviation. The Mann-Whitney U-test was used as appropriate, and Spearman’s correlation coefficient was used for correlation analysis. A P value <0.05 was considered statistically significant.

**Results**

**Clinical and transthoracic echocardiography findings**

Table 1 summarizes the results for clinical and transthoracic echocardiographic variables in the 3 groups (asymptomatic IVNC, symptomatic IVNC, healthy controls). There were no significant differences among the groups with respect to mean age, sex distribution, or mean blood pressure and heart rate findings. The symptomatic IVNC group had significantly higher mean LAD, LVESD and LVEDD values than the asymptomatic IVNC and control groups, and had significantly lower mean FS and mean LVEF than the asymptomatic and control groups.

There were no significant differences between the symptomatic IVNC and asymptomatic IVNC groups with respect to mean NC/C ratio (3.2 ± 0.7 vs. 3.2 ± 0.6, respectively; P > 0.05) or mean number of LV segments exhibiting non-compaction (6.1 ± 2 vs. 5.8 ± 3, respectively; P > 0.05). All 25 patients with IVNC had non-compaction in at least 1 segment at the apex and 1 segment in the mid-portion of the LV. All individuals with IVNC exhibited non-compaction in the inferior segment of the apex, and 9 patients (60%) in the symptomatic group and 7 (70%) in the asymptomatic group showed non-compaction in all segments of the apex. None of the patients in either IVNC subgroup exhibited non-compaction in any segment of the basal wall.

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Figure 2  Pulsed tissue Doppler image recordings of systolic myocardial contraction properties of the axes of the left ventricle along with simultaneous electrocardiography tracings. SW1, first peak of the systolic myocardial velocity pattern; SW2, second peak of the systolic myocardial velocity pattern.
Pulsed TDI findings

Table 1 summarizes the group results for the pulsed TDI variables. Compared to the symptomatic IVNC group, the asymptomatic and control groups both had significantly higher mean SW1 and mean SW2 on the long and short axes of the LV. In the symptomatic IVNC group, there was no significant difference between mean SW1 and mean SW2 on each axis. In the asymptomatic IVNC group, mean SW1 on the long axis was significantly higher than mean SW1 and SW2 on the short axis, and the mean SW1 and SW2 on the long axis were statistically similar. As well, in the asymptomatic IVNC group, mean SW2 on the long axis was significantly higher than mean SW2 on the short axis (9.1 ± 1.6 cm/s vs. 7.5 ± 2.2 cm/s, respectively; \( P < 0.01 \)). In the control group, mean SW1 on the long axis was significantly higher than mean SW2 on the long axis, and mean SW1 and SW2 on the short axis. Comparisons of peak velocities among the groups revealed that mean SW2 on the long axis in the asymptomatic IVNC group was significantly higher than the corresponding means in the control group (9.1 ± 1.6 cm/s vs. 7.0 ± 1.4 cm/s, respectively; \( P < 0.01 \)) and the symptomatic IVNC group (9.1 ± 1.6 cm/s vs. 4.7 ± 1.8 cm/s, respectively; \( P < 0.0001 \)).

Within each of the 3 groups, mean Q-SW1 on the long axis was significantly shorter than mean QSW2 on the long axis, and also significantly shorter than mean Q-SW1 and Q-SW2 on the short axis. However, in each of the groups, mean Q-SW2 on the long axis was not significantly different from mean Q-SW2 on the short axis. The mean Q-SW1 on the long axis in the control group was significantly shorter than the corresponding intervals in the symptomatic IVNC group (134.2 ± 66.6 ms vs. 169.5 ± 25.8 ms, respectively; \( P < 0.01 \)) and the asymptomatic IVNC group (134.2 ± 66.6 ms vs. 152.5 ± 37.4 ms, respectively; \( P < 0.01 \)). The mean Q-SW1 on the short axis in the control group was also

Table 1  Group results for clinical and transthoracic echocardiography variables

<table>
<thead>
<tr>
<th>Clinical characteristics</th>
<th>Symptomatic IVNC group ((n = 15))</th>
<th>Asymptomatic IVNC group ((n = 10))</th>
<th>Healthy controls ((n = 15))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>(41.2 ± 12.4)</td>
<td>(39 ± 11)</td>
<td>(38 ± 14)</td>
</tr>
<tr>
<td>Sex (male/female)</td>
<td>8/7</td>
<td>5/5</td>
<td>8/7</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>(72 ± 11)</td>
<td>(70 ± 12)</td>
<td>(71 ± 8)</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>(111 ± 15)</td>
<td>(113 ± 11)</td>
<td>(108 ± 12)</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>(78 ± 5)</td>
<td>(77 ± 10)</td>
<td>(79 ± 12)</td>
</tr>
<tr>
<td>Medication use, n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACE inhibitor</td>
<td>(15 (100%))</td>
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<td>0</td>
</tr>
<tr>
<td>β-blocker</td>
<td>(6 (40%))</td>
<td>2 (20%)</td>
<td>0</td>
</tr>
<tr>
<td>Digoxin</td>
<td>(9 (60%))</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Diuretics</td>
<td>(10 (67%))</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Anticoagulant</td>
<td>(1 (7%))</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Echocardiographic data</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IVS (mm)</td>
<td>(9.0 ± 1.0)</td>
<td>(9.0 ± 1.1)</td>
<td>(9.0 ± 0.9)</td>
</tr>
<tr>
<td>PW (mm)</td>
<td>(9.0 ± 1.1)</td>
<td>(10.0 ± 1.0)</td>
<td>(9.0 ± 0.8)</td>
</tr>
<tr>
<td>LVEDD (mm)</td>
<td>(59.6 ± 6.7)</td>
<td>(45.9 ± 3.1)</td>
<td>(46 ± 6)</td>
</tr>
<tr>
<td>LVESE (mm)</td>
<td>(48.4 ± 7.2)</td>
<td>(30.4 ± 2.9)</td>
<td>(30 ± 4)</td>
</tr>
<tr>
<td>LAD (mm)</td>
<td>(42.1 ± 3.9)</td>
<td>(31.8 ± 2.0)</td>
<td>(32 ± 5)</td>
</tr>
<tr>
<td>FS (%)</td>
<td>(21.5 ± 3.9)</td>
<td>(33.3 ± 2.9)</td>
<td>(36 ± 4)</td>
</tr>
<tr>
<td>EF (%)</td>
<td>(35.5 ± 7.9)</td>
<td>(63 ± 4.8)</td>
<td>(66 ± 8)</td>
</tr>
<tr>
<td>NC/C ratio</td>
<td>(3.2 ± 0.7)</td>
<td>(3.2 ± 0.6)</td>
<td>NA</td>
</tr>
<tr>
<td>NSN</td>
<td>(6.1 ± 2)</td>
<td>(5.8 ± 3)</td>
<td>NA</td>
</tr>
</tbody>
</table>

IVNC, isolated left ventricular non-compaction; NA, not applicable; ACE, angiotensin-converting enzyme; IVS, interventricular septum; PW, posterior wall of the left ventricle; LVEDD, left ventricular end-diastolic diameter; LVESE, left ventricular end systolic diameter; F1, left ventricular fractional shortening; EF, left ventricular ejection fraction; NC/C, non-compaction to compaction; NSN, number of segments with non-compaction. Values are expressed as mean ± standard deviation. \( * P < 0.0001 \) vs. control group and asymptomatic group.

Table 2  Group results for pulsed tissue Doppler imaging variables

<table>
<thead>
<tr>
<th>Long axis of LV</th>
<th>Symptomatic IVNC group ((n = 15))</th>
<th>Asymptomatic IVNC group ((n = 10))</th>
<th>Healthy controls ((n = 15))</th>
</tr>
</thead>
<tbody>
<tr>
<td>SW1 (cm/s)</td>
<td>(5.0 ± 1.7) ,( *)</td>
<td>(10.4 ± 2.1),( *)</td>
<td>(10.4 ± 2.9),( **)</td>
</tr>
<tr>
<td>SW2 (cm/s)</td>
<td>(4.7 ± 1.8),( †)</td>
<td>(9.1 ± 1.6),( **)</td>
<td>(7.0 ± 1.4)</td>
</tr>
<tr>
<td>Q-SW1 (ms)</td>
<td>(169.5 ± 25.8),( *)</td>
<td>(152.5 ± 37.4),( *)</td>
<td>(134.2 ± 66.6),( *)</td>
</tr>
<tr>
<td>Q-SW2 (ms)</td>
<td>(261.3 ± 50.1)</td>
<td>(250 ± 61.6)</td>
<td>(266.3 ± 96.7)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Short axis of LV</th>
<th>Symptomatic IVNC group ((n = 15))</th>
<th>Asymptomatic IVNC group ((n = 10))</th>
<th>Healthy controls ((n = 15))</th>
</tr>
</thead>
<tbody>
<tr>
<td>SW1 (cm/s)</td>
<td>(4.3 ± 1.4),( †)</td>
<td>(7.9 ± 1.4)</td>
<td>(8.0 ± 2.2)</td>
</tr>
<tr>
<td>SW2 (cm/s)</td>
<td>(4.2 ± 1),( †)</td>
<td>(7.5 ± 2.2)</td>
<td>(7.8 ± 1.9)</td>
</tr>
<tr>
<td>Q-SW1 (ms)</td>
<td>(190.4 ± 40),( †)</td>
<td>(173.6 ± 38.4)</td>
<td>(167.3 ± 74.6)</td>
</tr>
<tr>
<td>Q-SW2 (ms)</td>
<td>(269.3 ± 44.6)</td>
<td>(257.5 ± 42.3)</td>
<td>(282.7 ± 96.3)</td>
</tr>
</tbody>
</table>

IVNC, isolated left ventricular non-compaction; LV, left ventricle; SW1-2, first and second peaks of the systolic myocardial velocity pattern; Q-SW1-2, time from the electrocardiographic Q wave to the first and second peaks of the systolic myocardial velocity pattern. Values are expressed as mean ± standard deviation. \( * P < 0.0001 \) vs. asymptomatic and control groups, \( ** P < 0.0001 \) vs. asymptomatic group, \( † P < 0.001 \) vs. control group, \( †† P < 0.01 \) vs. control group, \( P < 0.01 \) vs. asymptomatic group, \( * P < 0.05 \) vs. long axis Q-SW2, short axis Q-SW1-2 in the same group, \( ** P < 0.01 \) vs. short axis SW1–2 in the asymptomatic group, \( †† P < 0.01 \) vs. long axis SW2, short axis SW1–2 in the control group.
significantly shorter than the mean Q-SW1 on the short axis in the symptomatic IVNC group.

Analysis of data from the asymptomatic IVNC patients revealed no significant correlations among any of the conventional echocardiography variables studied, or between number of segments with non-compaction and any of the pulsed TDI variables. There were also no significant correlations between NC/C ratio and any of these variables. In the symptomatic IVNC group, there were significant positive correlations between SW1 on the short axis and FS ($r_s = 0.53$, $P < 0.01$) and LVEF ($r_s = 0.52$, $P < 0.01$), and significant negative correlations between SW1 on the short axis and LVEDD ($r_s = -0.53$, $P < 0.01$) and LVESD ($r_s = -0.67$, $P < 0.01$). However, there were no relationships among any of the other conventional echocardiography variables studied, nor was NC/C ratio or number of affected segments correlated with any of the pulsed TDI variables.

Discussion

In recent years, advancements in cardiac imaging have led to greater awareness of IVNC. Previous studies suggested that the prognosis for patients with LV non-compaction is grave owing to high risk for HF, embolic events, and malignant arrhythmia. However, subsequent research has indicated that this patient group actually has a better prognosis. Individuals with IVNC present with a broad spectrum of clinical pictures ranging from preserved LV function to HF.

Studies involving positron emission tomography, magnetic resonance imaging and myocardial contrast echocardiography have identified coronary microcirculatory dysfunction in patients with IVNC. Williams et al. reported that the curved M-mode (C-mode) strain rate profile of non-compacted myocardium reveals that these individuals exhibit alternating regions of regional compression and expansion throughout the cardiac cycle, resulting in physiologic disarray. Microcirculatory dysfunction and uncoordinated contraction in areas of the LV that exhibit non-compaction result in regional ventricular contraction abnormalities. Such abnormalities are likely the main explanation for progressive deterioration of LV function in patients with IVNC.

The normal LV is thickest at the base, and the number of circumferential fibers is greatest in this region. The ventricular wall becomes thinner towards the apex as the number of circumferential fibers diminishes. From the base to the apex, the circumferential radii of curvature decrease and the longitudinal radii of curvature increase. LV strain is somewhat different at the apex compared to the base. There are also different levels of strain at the endocardial and epicardial aspects of the myocardium. During the cardiac cycle, myocardial strain increases from base to apex, resulting in higher EF at the apex than at the base. However, non-compaction causes structural abnormalities, particularly at the apex, and these alter the normal contraction and strain properties of the LV.

Oki et al. reported that pulsed-TDI recordings for healthy subjects display 2 separate velocity peaks (SW1 and SW2) along the long axis and along the short axis of the LV during systolic contraction. Our testing also revealed these 2 separate systolic velocity peaks in patients with IVNC and in healthy volunteers. In line with the findings of Oki et al., in our control group we found that the mean SW1 on the long axis was significantly higher than the mean long axis SW2 and the mean short axis SW1 and SW2. Further, the mean Q-SW1 for the long axis in this group was significantly shorter than the long axis Q-SW2, and significantly shorter than the short axis Q-SW1 and Q-SW2. Interestingly, we found that both the symptomatic IVNC group (patients with low LVEF) and the asymptomatic IVNC group (patients with normal LVEF) had different peak velocity (SW) and interval (Q-SW) findings from the control group. Moreover, the peak velocity and interval findings in the symptomatic IVNC group were distinct from those in asymptomatic IVNC group.

Previous research has demonstrated that the long axis SW1 occurs at the isovolumic contraction phase of the LV in healthy subjects and in patients with cardiac disorders. Mishiro et al. suggested that SW1 on this axis, as measured with pulsed TDI, may be useful for understanding LV contraction abnormalities in patients who are in the early stages of myocardial disease. In our study, we found that the asymptomatic IVNC group had a significantly longer mean long axis Q-SW1 than the healthy subjects (152.5 ± 37.4 ms vs. 134.2 ± 66.6 ms, for asymptomatic vs. controls, respectively; $P < 0.01$). Prolongation of long axis Q-SW1 in the asymptomatic IVNC group suggests that there is delayed shortening of the long axis in the isovolumic contraction phase, leading to asynchronous systolic contraction. The above-normal long axis SW2 in this group (9.1 ± 1.6 cm/s vs. 7.0 ± 1.4 cm/s for asymptomatic vs. controls, respectively; $P < 0.01$) is likely a result of this asynchrony.

Previous research has demonstrated that the long axis SW1 occurs at the isovolumic contraction phase of the LV in healthy subjects and in patients with cardiac disorders. Mishiro et al. suggested that SW1 on this axis, as measured with pulsed TDI, may be useful for understanding LV contraction abnormalities in patients who are in the early stages of myocardial disease. In our study, we found that the asymptomatic IVNC group had a significantly longer mean long axis Q-SW1 than the healthy subjects (152.5 ± 37.4 ms vs. 134.2 ± 66.6 ms, for asymptomatic vs. controls, respectively; $P < 0.01$). Prolongation of long axis Q-SW1 in the asymptomatic IVNC group suggests that there is delayed shortening of the long axis in the isovolumic contraction phase, leading to asynchronous systolic contraction. The above-normal long axis SW2 in this group (9.1 ± 1.6 cm/s vs. 7.0 ± 1.4 cm/s for asymptomatic vs. controls, respectively; $P < 0.01$) is likely a result of this asynchrony.

Oki et al. reported that, in healthy subjects, SW2 on the short axis is greater than SW2 on the long axis. This finding suggests that the shortening of circumferential fibers predominates over shortening of longitudinal fibers during the systolic ejection phase. However, in the asymptomatic IVNC group in our study, mean SW2 on the long axis was significantly greater than that along the short axis (9.1 ± 1.6 cm/s vs. 7.5 ± 2.2 cm/s, for long axis vs. short axis, respectively; $P < 0.01$). Based on this, we suspect that there is uncoordinated shortening of circumferential and longitudinal fibers during the systolic ejection phase in patients with asymptomatic IVNC.

LV dilatation is the main echocardiographic hallmark of idiopathic dilated cardiomyopathy (DCMP). Sengupta et al. described a distinct form of spherical LV remodeling in IVNC in which the ventricle walls remain thick and chambers do not dilate in proportion to the degree of LV remodeling and dysfunction. A previous report by our group documented that, given comparable degrees of spherical remodeling and comparable LV dysfunction, patients with IVNC and patients with DCMP show similar regional contraction properties along the long and short axes of the LV. Mishiro et al. also reported that the remodeling and systolic dysfunction of the LV in patients with DCMP are related to decreased systolic myocardial velocities (SW1 and SW2) and prolongation of Q-SW1 and Q-SW2 on the long and short axes of the LV. In this study, our patients with symptomatic IVNC exhibited markedly reduced (compared to control group) systolic myocardial velocities and prolonged Q-SW1 along the long and short axes of the LV. Therefore, the pulsed TDI findings in the symptomatic IVNC group can be attributed to the LV remodeling and impaired LV
function. On the other hand, our findings in the asymptomatic IVNC patients indicated distinct myocardial contraction along the long and short axes of the LV. It is likely that regional shortening of LV myocardium becomes altered as the disease progresses, and that this results in systolic LV dysfunction. However, the precise steps in this process still need to be clarified.

Relationships between pulsed TDI variables and long-term prognosis for patients with IVNC were beyond the scope of this study. In the symptomatic IVNC group, we detected significant positive correlations between short axis SW1 and FS and EF (rs = 0.53, P < 0.01 for FS; and rs = 0.52, P < 0.01 for EF), and significant negative correlations between short axis SW1 and LVEDD and LVESD (rs = -0.53, P < 0.01 for LVEDD; rs = -0.67, P < 0.01 for LVESD); however, our findings revealed no such correlations in the asymptomatic group. None of the pulsed TDI variables we studied were significantly correlated with NC/C ratio or number of segments with non-compaction. Overall, these statistical findings suggest that certain pulsed TDI findings are probably independently associated with deterioration of LV function in patients with IVNC.

**Study limitations**

The prevalence of IVNC among patients referred to our echocardiography laboratory is very low, and this restricted the number of cases for our study. Although we did not perform cardiac catheterization in all 25 of the subjects with IVNC, we interpreted the pulsed-TDI recordings based on findings in previous, well-designed investigations.4,16,27 Due to the small size of the study population, it was not possible to assess pulsed TDI variables in relation to embolic or arrhythmic complications. Further, because of the limited numbers we were only able to analyze 2 small subsets within the disease spectrum (LV systolic function preserved [asymptomatic patients] or depressed [symptomatic patients]). Issues such as bundle branch block, pre-excitation syndromes, and neuromuscular abnormalities were excluded from this study. However, research has identified unique associations between IVNC and these various clinical conditions,3-5 and all may affect pulsed TDI variables. Another important limitation we recognize is that we did not quantitatively investigate coronary microcirculatory dysfunction in our subjects. This would have been valuable for comparison with the pulsed TDI findings. Also, owing to our small study population, it was not possible to test correlations between pulsed TDI findings and long term prognosis. Finally, all of our subjects were adults, and pulsed TDI results for this population cannot be extrapolated to IVNC in childhood.

In conclusion, our pulsed TDI data clearly show that, in patients with IVNC, systolic myocardial contraction properties along the long and short axes of the LV are distinct from those in healthy subjects. Further, these myocardial findings in patients with symptomatic IVNC are distinct from those in patients with asymptomatic IVNC. These findings suggest that pulsed TDI can identify LV systolic asynchrony and uncoordinated contraction at early stages of disease in patients with IVNC who have normal LVEF and no evidence of HF.

**References**


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O. Tufekcioglu et al.