Myocardial Acceleration During Isovolumic Contraction Relationship to Contractility

Erik Lyseggen, MD; Stein Inge Rabben, PhD; Helge Skulstad, MD; Stig Urheim, MD; Cecilie Risoe, MD, PhD; Otto A. Smiseth, MD, PhD

Background—Acceleration of the mitral ring during isovolumic contraction has been proposed as a load-independent index of global left ventricular (LV) contractility. This study investigates whether myocardial isovolumic acceleration (IVA) reflects regional contractility.

Methods and Results—In acutely instrumented, anesthetized dogs, we measured LV pressure, myocardial long-axis velocities, and IVA by tissue Doppler imaging (TDI) and sonomicrometry at different levels of global LV contractility and preload and during regional myocardial ischemia (reduced flow in the left anterior descending coronary artery). Dobutamine caused dose-dependent increments in IVA from 3.6±0.6 (mean±SEM) to a maximum of 7.1±1.4 m/s^2 (P<0.01) by TDI, and there were parallel increments in LV dP/dt_max (P<0.01). However, volume loading decreased IVA from 3.6±0.6 to 2.5±0.4 m/s^2 (P<0.05), whereas LV dP/dt_max was unchanged, and LV pressure–segment length loop analysis confirmed unchanged regional contractility. During myocardial ischemia, sonomicrometry indicated severely depressed regional function, whereas IVA remained unchanged. These findings were confirmed when IVA was measured by sonomicrometry. In contrast to peak ejection velocity that increased from apex toward the LV base, peak IVC velocity was maximum midway between apex and base. The onset of IVA coincided with onset of the first heart sound by phonocardiography. Peak IVA occurred at a LV pressure of 14±1 mm Hg, ie, close to end-diastole.

Conclusions—There was no consistent relationship between peak IVA and regional myocardial contractility. Peak IVA was markedly load dependent and did not reflect impaired myocardial function during ischemia. Peak IVA may reflect late-diastolic events and possibly wall oscillations that are related to global LV function. Peak IVA seems to have limited potential in the assessment of regional myocardial function. (Circulation. 2005;111:1362-1369.)

Key Words: echocardiography ■ ischemia ■ contractility ■ myocardial contraction ■ acceleration

Left ventricular (LV) function is commonly assessed in terms of ejection phase indices, and for global function the most widely used measure is ejection fraction. In general, when LV ejection fraction is depressed there is a decrease in contractility. However, ejection fraction also depends on loading conditions and therefore is not a reliable measure of contractility when there are marked changes in preload or afterload. Regional myocardial function is also assessed by ejection phase indices, most often with the use of 2-dimensional echocardiography and visual assessment of regional wall motion and more recently with the use of tissue Doppler imaging (TDI) to measure peak myocardial ejection velocity. Another new TDI modality is strain Doppler echocardiography, which measures regional myocardial shortening fraction and thickening fraction in the LV long and short axis, respectively. However, similar to global ejection fraction, the regional ejection phase indices are markedly load dependent and do not directly reflect contractility. Vogel et al hypothesized that myocardial velocity indices measured during the isovolumic contraction (IVC) phase would be less load dependent than ejection phase indices. In a pig model, they calculated myocardial isovolumic acceleration (IVA) from TDI velocities and demonstrated a correlation between IVA and measures of global LV contractility. Furthermore, they found that IVA was unaffected by relatively large preload reductions and afterload increases. In their study, IVA was calculated from longitudinal velocities recorded near the LV base, which means that they in essence measured global LV function.

The present study was designed to investigate whether myocardial acceleration during IVC reflects regional myocardial function. Early in the study, however, we observed a very close temporal relationship between peak IVA and end-diastole and dissociation between IVA and regional contractility during myocardial ischemia. Therefore, we also investigated whether IVA may be a reflection of global cardiac events, potentially LV wall motion subse-
The study was done in a dog model, and myocardial velocity and acceleration were measured during changes in contractility and loading conditions and after induction of ischemia. Acceleration was assessed by TDI, which measures the net motion in a region of interest, and by sonomicrometry, which measures the local segmental contribution to motion. As reference method for regional contractility, we used systolic shortening by sonomicrometry and the systolic pressure–segment length loop relationship.

Methods

Twelve mongrel dogs of either sex and with body weight 24.1 ± 0.9 kg were anesthetized, ventilated, and surgically prepared as previously described.4 The initial part of the study was performed on 6 dogs, and the data in the tables and figures are from these experiments. To address the questions about contribution of atrial contraction and the first heart sound on IVA, we performed 6 supplementary experiments. The National Animal Experimentation Board approved the study. The laboratory animals were supplied by Center for Comparative Medicine, Rikshospitalet University Hospital, Oslo, Norway.

Hemodynamic Measurements and Phonocardiography

Aortic, left atrial, and LV pressures were measured by micromanometers (MPC-500, Millar Instruments Inc), and flow in the left anterior descending coronary artery (LAD) was measured by ultrasound transit time as previously described.7 Data were digitized at 200 Hz. The timing of cardiac phases was defined by pressure crossovers (Figure 1). Intracardiac phonocardiogram was recorded in 6 dogs via the LV micromanometer with the use of a high-pass filter (Millar Audio Adapter, Millar Instruments Inc).

Sonomicrometry

In the anterior LV wall (LAD region), 1 pair of ultrasonic crystals was implanted in the inner third of the myocardium, aligned parallel to the LV long axis. In 4 dogs, 1 additional pair of ultrasonic crystals was implanted in the region supplied by the left circumflex coronary artery for reference. The crystals were connected to a sonomicrometer (Sonometrics Corp), and data were digitized at 200 Hz.

Echocardiography

We used a System FiVe digital ultrasound machine (GE Vingmed Ultrasound AS) with a phased-array ultrasound transducer. The frame rate of the recordings was 160 ± 1 per second. We recorded 2-dimensional color-coded TDI images of 3 consecutive heart cycles. Images were recorded with an apical 2-chamber view with imaging plane through the region in which the LAD crystals were positioned. The sample volume was placed between the LAD crystals to obtain velocity and acceleration by TDI in the region corresponding to the sonomicrometry recordings (apical segment) and in the middle and basal segments. In an apical 4-chamber view, we recorded velocity and acceleration from the 3 segments in the lateral wall and septum. In all recordings, we used a fixed sample volume, making sure the pericardium and endocardium were not included during the heart cycle. The sample volume was 5 range samples by 3 beams. Typically, this corresponds to a height and width of approximately 4 mm. The effect of moving the sample volume (tracking the wall) was considered insignificant to the recordings during IVC because the actual movement of the myocardium is very small during the isovolumic phases. An experimental application (System 5 TVI, version 6.0, Vingmed Sound) was used for analysis.

Calculations

Peak systolic shortening by sonomicrometry was calculated as percentage of end-diastolic dimension. Regional myocardial velocity was calculated by differentiation and inversion of the LAD segment length tracing and regional acceleration by differentiation of the velocity tracing (Figure 2).
Peak IVA by TDI was calculated as the difference between the 2 sequential IVC velocities, with the largest velocity increment divided by the frame-by-frame time interval.

The spectrograms of the acceleration tracings and the phonocardiograms were calculated by use of Matlab version 6.5 (MathWorks Inc). A spectrogram shows the frequency spectrum of a signal as a function of time with the use of a moving window on the original signal. In our case, we calculated the spectrograms (window of 250 ms) for signals from baseline, dobutamine infusion, and volume loading in 4 dogs.

**Experimental Protocol**

After a 30-minute stabilization period, baseline recordings were performed. To avoid interference between sonomicrometry and Doppler, we first recorded pressures and ECG and echocardiographic data during 10 seconds and then pressures, flow, and ECG and segment lengths during the subsequent 10 seconds. Data were recorded with the ventilator off.

**Changes in Loading Condition and Inotropy**

To determine how regional IVA in nonischemic myocardium responds to changes in loading and to increased inotropy, we performed the following interventions. Preload was reduced by transient caval constrictions and was elevated by rapid intravenous infusion of isotonic saline. Inotropy was enhanced by intravenous dobutamine administration at 2 levels (<5.0 μg/kg per minute and >5.0 μg/kg per minute), grouped by the responses in terms of heart rate and dP/dt max. Hemodynamic variables were allowed to return to baseline values before the start of each intervention.

**Asynchronous Atrioventricular Pacing**

To determine the role of atrial contraction in 4 dogs, we used asynchronous atrioventricular pacing and compared IVA with and without atrial contribution to filling.

**Myocardial Ischemia**

Recordings were performed during baseline and during 3 levels of ischemia, induced by inflating a pneumatic LAD constrictor. Ischemia levels were set according to the degree of myocardial dysfunction as measured by sonomicrometry. Flow in the LAD was reduced until there was mild hypokinesia (ischemia level 1). The LAD flow was then reduced further until there was marked hypokinesia (ischemia level 2). Finally, the LAD was occluded, and recordings were done after 1 minute, while there was slight dyskinesia (ischemia level 3). When the LAD occlusion lasted >1 to 2 minutes, the segment developed marked systolic lengthening, which resulted in a negative velocity spike during IVC. The positive velocity component that was used to calculate IVA merged with this much larger oppositely directed velocity, and the positive IVA was no longer present.

**Statistical Analysis**

Values are expressed as mean±SEM. For multiple comparisons we used 1-way ANOVA with Bonferroni posttests (GraphPad Prism version 4.02 for Windows, GraphPad Software). Baseline was compared with recordings during loading, 2 levels of dobutamine infusion, and 2 levels of ischemia. For comparisons, P<0.05 was considered significant.

**Results**

Representative recordings of myocardial velocities by TDI and sonomicrometry are displayed in Figures 1 and 2, respectively. Although velocities by TDI (V TDI) were consistently larger than those measured by sonomicrometry (V sono), velocities by the 2 methods correlated well for peak IVC velocity (\(V_{T DI} = 0.9 \times V_{sono} + 2.9; r = 0.59; P<0.001\)) and for peak systolic ejection velocity (\(V_{T DI} = 3.5 \times V_{sono} + 2.0; r = 0.78; P<0.001\)). In addition, IVA by the 2 methods (TDI [IVA TDI] and sonomicrometry [IVA sono]) correlated well (\(IVA_{TDI} = 1.2 \times IVA_{sono} + 2.1; r = 0.68; P<0.001\)).

As demonstrated in Figure 2 peak IVA occurred very early in systole, during baseline conditions at a LV pressure of only 14±1 mm Hg, compared with an end-diastolic pressure of 6±1 mm Hg. This implies that IVA occurred very close to end-diastole, only 26±2 ms after the peak R wave in the ECG.

In contrast to peak ejection velocity, which in the nonischemic ventricle decreased progressively \((P<0.01)\) from the LV base toward the apex, peak IVC velocity was maximum near the equator (6.8±0.4 cm/s) and was lower \((P<0.01)\) near the mitral ring (3.5±0.3 cm/s) and near the apex (3.9±0.3 cm/s).

**IVA During Changes in Loading Conditions and Contractility**

Dobutamine caused progressive dose-related increments in LV contractility, measured in terms of LV dP/dt max, and there were parallel increments in peak IVC velocity and IVA as measured by sonomicrometry and TDI (Figure 3 and Table 1).

![Figure 3. Responses to dobutamine. A, Responses to dobutamine in a representative experiment. Dobutamine caused dose-related increments in dP/dt max, accompanied by increments in peak IVC velocity and IVA. B, Mean data showing effects of dobutamine on IVA and dP/dt max (n=6).](http://circ.ahajournals.org/content/110/6/1364/F3)
TABLE 1. Hemodynamic, Sonomicrometric, and Doppler Variables (n=6)

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Volume Loading</th>
<th>Dobutamine &lt;5 µg/kg per min</th>
<th>Dobutamine &gt;5 µg/kg per min</th>
<th>LAD Stenosis, Ischemia Level 2</th>
<th>LAD Occlusion, Ischemia Level 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate, bpm</td>
<td>125±6</td>
<td>121±5</td>
<td>106±7</td>
<td>135±13</td>
<td>112±6</td>
<td>111±5</td>
</tr>
<tr>
<td>LV peak systolic pressure, mm Hg</td>
<td>106±3</td>
<td>126±5†</td>
<td>119±7</td>
<td>120±5†</td>
<td>103±4</td>
<td>102±4</td>
</tr>
<tr>
<td>LV end-diastolic pressure, mm Hg</td>
<td>5.9±1.3</td>
<td>19.0±2.2†</td>
<td>6.6±1.1</td>
<td>5.0±1.2</td>
<td>6.7±1.1</td>
<td>7.4±0.9</td>
</tr>
<tr>
<td>LV dp/dt&lt;sub&gt;max&lt;/sub&gt;, mm Hg/s</td>
<td>1962±183</td>
<td>2290±162</td>
<td>3211±269†</td>
<td>4077±295†</td>
<td>1785±98</td>
<td>1831±113</td>
</tr>
<tr>
<td>Peak IVA by sonomicrometry, m/s&lt;sup&gt;2&lt;/sup&gt;</td>
<td>2.1±0.4</td>
<td>0.7±0.2†</td>
<td>2.6±0.6</td>
<td>3.4±0.6†</td>
<td>1.8±0.4</td>
<td>1.7±0.5</td>
</tr>
<tr>
<td>Peak IVA by TDI&lt;sup&gt;*&lt;/sup&gt;, m/s&lt;sup&gt;2&lt;/sup&gt;</td>
<td>3.6±0.6</td>
<td>2.5±0.4†</td>
<td>5.7±0.8†</td>
<td>7.1±1.4†</td>
<td>3.2±0.4</td>
<td>3.3±0.3</td>
</tr>
<tr>
<td>IVC velocity by sonomicrometry, cm/s</td>
<td>2.3±0.5</td>
<td>0.6±0.3†</td>
<td>2.2±0.7</td>
<td>2.9±0.6†</td>
<td>1.1±0.5†</td>
<td>0.8±0.3†</td>
</tr>
<tr>
<td>IVC velocity by TDI&lt;sup&gt;*&lt;/sup&gt;, cm/s</td>
<td>4.3±0.7</td>
<td>2.3±0.1†</td>
<td>5.9±0.7</td>
<td>7.2±1.1†</td>
<td>3.5±0.6</td>
<td>3.7±0.4</td>
</tr>
<tr>
<td>Ejection velocity by TDI&lt;sup&gt;*&lt;/sup&gt;, cm/s</td>
<td>4.1±0.6</td>
<td>5.8±0.8</td>
<td>6.4±0.6</td>
<td>8.3±1.0†</td>
<td>3.7±0.3</td>
<td>3.4±0.2</td>
</tr>
<tr>
<td>Systolic shortening by sonomicrometry, %</td>
<td>12±2</td>
<td>16±3†</td>
<td>12±3</td>
<td>12±3</td>
<td>3±1†</td>
<td>-5±2†</td>
</tr>
</tbody>
</table>

Values are mean±SEM.  
<sup>*</sup>Regional function, apical segment, anterior wall.  
†P<0.05 vs baseline.

Volume loading by intravenous saline caused a small increase in LV dp/dt<sub>max</sub> (P=NS), whereas peak IVA by sonomicrometry and Doppler decreased markedly (P<0.05) (Figure 4 and Table 1). Segmental systolic shortening increased markedly during volume loading (Table 1). To investigate whether the dissociation between IVA and LV dp/dt<sub>max</sub> could be due to inability of dp/dt<sub>max</sub> to reflect regional contractility, we analyzed the systolic pressure–segment length relationship. Figure 5 shows a representative experiment and demonstrates that the end-systolic pressure–segment length relationship was essentially unchanged after volume loading, which confirms that regional contractility was preserved.

The responses of IVA to changes in preload were biphasic, as shown by a decrease in IVA when preload was markedly reduced by caval constriction and a decrease after intravenous volume loading (Figure 6).

Table 2 compares peak IVA by TDI in different regions of the left ventricle and shows that responses to dobutamine and volume loading were essentially similar in all segments that were examined. In 4 dogs with ultrasonic crystals in the posterior LV wall, the responses to changes in loading and dobutamine were similar to those for the anterior wall, i.e., in each experiment IVA increased from 1.7±0.1 to 2.5±0.4 m/s<sup>2</sup> with dobutamine and decreased from 1.7±0.1 to 1.0±0.2 m/s<sup>2</sup> with elevated preload. Similar to the observations in the LAD territory, there was a good correlation between IVA and LV dp/dt<sub>max</sub> during dobutamine infusions (r=0.92; P<0.001). Furthermore, the timing of peak IVA was similar for measurements taken in the LAD and circumflex coronary artery regions (mean difference with reference to peak R in the ECG was 0.5±2.5 ms).

IVA During Acute Ischemia
At ischemia levels 1, 2, and 3, there were progressive reductions in systolic shortening (P<0.05), but IVA was preserved. At ischemia level 3, the segment became dyskinetic, and pressure–segment length loop analysis indicated that the myocardium was behaving as a net passive structure during IVC, with lengthening and dominantly negative velocities. There was, however, still a positive velocity deflection on top of the dominantly negative velocity, and this accounted for preserved IVA (Figure 7). The IVA in nonischemic regions remained unchanged during LAD occlusion (Table 2).

Relationship between IVA and Atrial Contraction
Successful elimination of atrial contribution to filling was achieved in 3 of the 4 experiments with asynchronous atrioventricular pacing. The IVC velocity spike was still present in all the beats without preceding atrial systole. In each case peak IVA measured by TDI and sonomicrometry was reduced, from a mean of 5.1 to 3.0 m/s<sup>2</sup> and from 1.6 to 1.3 m/s<sup>2</sup>, respectively. In each case this was accompanied by a small drop in LV dp/dt, from a mean of 2652 to 2091 mm Hg/s, consistent with the relationship between IVA and LV dp/dt that was observed in the rest of the study.

Relationship Between IVA and First Heart Sound
As indicated in Figure 8, the timing of the positive deflection in the phonocardiogram coincided with the IVC velocity that accounted for IVA. On average, the initial phonocardiographic deflection preceded the initial myocardial IVC velocity by 6±2 ms (P<0.05). The peak amplitude of the first heart sound, however, was delayed 10±4 ms (P<0.05) relative to the peak IVC velocity. The peak amplitude of the first heart sound correlated well with peak IVA during enhancement of contractility by dobutamine (r=0.96; P<0.001). During ischemia, there was no significant change in either peak IVA or peak amplitude of the first heart sound. During volume loading, however, there was an increase in peak amplitude of the first heart sound, whereas peak IVA decreased.

For the acceleration tracings at baseline, dobutamine infusion, and volume loading, the peak amplitude of the frequency spectrum during IVC was 13±2, 23±1, and 20±4 Hz, respectively. This differed from the phonocardiograms, which had a peak frequency amplitude during IVC of 7±0.4 (P<0.02), 13±0.9 (P<0.01), and 9±1 Hz (P<0.06). However, the peak frequency amplitude of the 2 signals correlated...
The differences in time to peak amplitude of the frequency spectrum during IVC between acceleration tracings and phonocardiograms were 41/4, 27/6, and 41/12 ms (P<0.05), respectively.

Discussion
The present study investigates whether myocardial IVA as measured by TDI may be used as an index of regional myocardial contractility. On the basis of measurements taken during a number of different contractile states and different loading conditions, it was evident that myocardial acceleration does not reflect regional contractility. This was seen most clearly during myocardial ischemia, which caused severe impairment of regional function, whereas there was no change in myocardial acceleration. Measurements of acceleration by TDI were confirmed by measurement of acceleration by sonomicrometry. Therefore, the present observations do not support the assumption that IVA can be used as an index of regional systolic function.

Responses to Changes in Loading Conditions
In the present study peak IVA decreased markedly when preload was elevated, whereas global contractility, as measured by LV dP/dt max, was unchanged. This apparent dissociation between IVA and contractility could be due to the limited ability of LV dP/dt max to reflect regional function. Therefore, in addition we measured regional contractility in terms of myocardial pressure–segment length loops. The results from this analysis, however, were entirely consistent with those using LV dP/dt max to reflect contractility and confirm that peak IVA is preload dependent.

The responses to reductions in preload were dependent on the prevailing LV filling pressure and therefore end-diastolic volume. As illustrated in Figure 6, the response was biphasic and might reflect a relationship between IVA and myocardial elastic properties, which will change in a nonlinear fashion with changes in end-diastolic volume.

Responses to Changes in Global and Regional Contractility
When global LV contractility was increased by intravenous dobutamine infusion, there were parallel and dose-related increments in LV dP/dt max and IVA. When regional contractility was decreased by induction of ischemia, however, IVA remained unchanged. Even during the early stages of the most severe ischemia, which resulted in passive systolic lengthening of the segment, IVA was similar to that in nonischemic segments. Therefore, clearly IVA did not reflect contractility of ischemic myocardium.

When LAD occlusion was maintained for an extended period, there was more marked systolic lengthening, which resulted in a large negative IVC velocity that merged with the
smaller positive IVC velocity spike, and therefore IVA became negative. However, when systolic pressure was reduced by caval constriction, the systolic lengthening was reduced, and the positive IVC velocity spike reappeared.

Comparison With Previous Studies and Methodologies
Relatively few studies have investigated the physiological significance of myocardial IVC velocities. Vogel et al5,6,9 measured tricuspid and mitral ring velocities by TDI and observed a relationship between global right ventricular and LV contractility and IVA. The present study confirmed the findings of Vogel et al of a good correlation between IVA and LV dP/dtmax during incremental dobutamine infusion and during mild reductions in preload, and the responses were similar for measurements taken near the mitral ring (basal segments) and more distally in the LV wall. The main finding of the present study, however, is that regional IVA does not reflect regional contractility. We consider this of major importance because TDI is primarily a modality for assessing regional myocardial function.

In the present study regional function by sonomicrometry was measured primarily in the anterior LV wall. Supplemen-
Mechanisms of IVC Velocities and IVA

In the normal ventricle, there is segmental shortening before onset of LV ejection, which contributes to changes in global LV geometry\(^\text{10}\) and may mobilize blood that allows the mitral leaflets to bulge into the left atrium during systole. This pre-ejection shortening represents active contraction and most likely contributes to IVC velocities as measured by TDI. However, as shown in the present study, the velocity spike that accounts for peak IVA was unchanged during severe ischemia, which makes it unlikely that local, active fiber shortening causes this velocity component. More likely, IVA represents velocities that originate from interactions with other segments or cardiac structures.

Peak IVA occurred very early in systole, when LV pressure was only \(\approx 14\) mm Hg, and we hypothesized that the IVC velocities represent wall oscillations due to atrial-induced filling. In experiments in which we induced asynchronous atrioventricular contraction, the positive IVC velocity spike was still present. Therefore, we could not attribute peak IVA entirely to wall oscillations induced by atrial contraction.

Alternatively, one may speculate that IVA represents wall oscillations caused by mitral valve closure or the rapid change in LV pressure that occurs during IVC. Bongiorni et al\(^\text{11}\) used a microaccelerometer located on the tip of a transvenous pacing lead to measure peak endocardial acceleration. Similar to IVA in the present study, peak endocardial acceleration occurred during IVC, and amplitude and timing of the peak endocardial acceleration were independent of the recording site and atrial rhythm and appeared to be related to global ventricular contractility. Rickards et al\(^\text{12}\) proposed that peak endocardial acceleration is a reflection of vibrations generated and transmitted throughout the heart by the isometric contraction of the myocardium during IVC. Furthermore, they state that these vibrations are, in their audible component, responsible for the first heart sound.

In the present study, the onset of the first heart sound coincided with onset of IVA, and the amplitude of the heart sound correlated well with IVA during changes in inotropy. During elevated preload, however, the amplitude of the heart sound increased, whereas IVA decreased. One may speculate that this dissociation is due to increased stiffness in the LV wall at high preload.

When we analyzed the frequency spectrograms of the acceleration tracings and the phonocardiograms, the peak amplitude of the 2 signals correlated well, but there were substantial differences in both frequency and timing of the peak amplitude.

Therefore, with the present methodology, we were not able to find definite support for the assumption that peak IVA and the first heart sound are reflections of the same cardiac mechanical event.

Furthermore, one also needs to consider whether deformations associated with changes in LV geometry and torsion may account for velocity components during IVC.

Conclusions

TDI is a potentially powerful method to investigate regional myocardial function, and the most widely used measure is peak systolic ejection velocity. As demonstrated in the present study, the measurement of IVA does not provide further information about regional contractility. The mechanisms behind myocardial IVA are complex, and global cardiac events appear to be important determinants. Therefore, IVA seems to have very limited potential as an index of regional myocardial function.

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**TABLE 2. Peak IVA by TDI in Multiple Regions of the Left Ventricle (n=6)**

<table>
<thead>
<tr>
<th>Segment</th>
<th>Baseline Peak IVA, m/s(^2)</th>
<th>Dobutamine Loading &amp;&lt; 5 (\mu)g/kg per min Peak IVA, m/s(^2)</th>
<th>Dobutamine Loading &amp;&gt; 5 (\mu)g/kg per min Peak IVA, m/s(^2)</th>
<th>LAD Stenosis Ischemia 2 Peak IVA, m/s(^2)</th>
<th>LAD Occlusion Ischemia 3 Peak IVA, m/s(^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Septum Apical</td>
<td>3.4±0.3</td>
<td>2.4±0.4</td>
<td>6.6±1.4*</td>
<td>3.0±0.3</td>
<td>2.9±0.5</td>
</tr>
<tr>
<td>Mid segment</td>
<td>5.9±1.1</td>
<td>3.3±0.4*</td>
<td>10.1±1.7*</td>
<td>12.7±3.2*</td>
<td>5.3±1.0</td>
</tr>
<tr>
<td>Basal segment</td>
<td>4.9±1.8</td>
<td>2.9±0.4*</td>
<td>8.3±2.7</td>
<td>11.2±3.9*</td>
<td>5.2±0.8</td>
</tr>
<tr>
<td>Anterior wall Apical</td>
<td>3.6±0.6</td>
<td>2.5±0.4*</td>
<td>5.7±0.8*</td>
<td>7.1±1.4*</td>
<td>3.2±0.4</td>
</tr>
<tr>
<td>Mid segment</td>
<td>4.3±0.3</td>
<td>4.2±0.7*</td>
<td>5.9±0.7*</td>
<td>9.4±1.8*</td>
<td>4.1±0.8</td>
</tr>
<tr>
<td>Basal segment</td>
<td>4.4±0.8</td>
<td>3.4±0.5</td>
<td>5.8±0.9*</td>
<td>9.8±2.7*</td>
<td>4.5±1.2</td>
</tr>
<tr>
<td>Lateral wall Apical</td>
<td>3.4±0.4</td>
<td>1.4±0.3*</td>
<td>4.6±0.5*</td>
<td>6.9±0.5*</td>
<td>3.6±0.5</td>
</tr>
<tr>
<td>Mid segment</td>
<td>4.1±0.4</td>
<td>2.7±0.5*</td>
<td>6.5±0.7*</td>
<td>8.5±0.4*</td>
<td>4.1±0.3</td>
</tr>
<tr>
<td>Basal segment</td>
<td>5.2±1.6</td>
<td>2.4±0.5*</td>
<td>3.3±0.4</td>
<td>8.0±2.9*</td>
<td>4.1±1.2</td>
</tr>
</tbody>
</table>

Values are mean±SEM.

\(* P<0.05\) vs baseline.
Drs Lyseggen, Skulstad, and Urheim were recipients of clinical research fellowships from the Norwegian Council of Cardiovascular Diseases. Dr Rabben was the recipient of a postdoctoral research fellowship from the Research Council of Norway. Dr Risoe was financed by Ullevaal University Hospital. We thank Professor Jens M. Hovem for useful advice about signal interpretation, engineer Roger Odegaard for important technical assistance, and Drs Trond Vartdal, Thomas Helle-Valle, and Eirik Pettersen for beneficial collaboration in the laboratory.

Disclosure

After having finished the work on this study, Dr Rabben changed employers and currently works for GE Vingmed Ultrasound, which supplied some of the equipment used in this study.

References


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Erik Lyseggen, Stein Inge Rabben, Helge Skulstad, Stig Urheim, Cecilie Risoe and Otto A. Smiseth

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