Abnormalities in Myocardial Segmental Wall Motion during Lumbar Epidural Anesthesia

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The effect of lumbar epidural anesthesia on myocardial wall motion was compared in two groups of patients using precordial two-dimensional echocardiography (2DE). All patients were scheduled to undergo lower abdominal or peripheral surgery. Group 1 included five healthy ASA PS 1 subjects and group 2 included 10 patients with coronary artery disease (CAD). In all patients 12.5 ml of 2% lidocaine HCl was injected into the lumbar epidural space, and systolic and diastolic blood pressures, and heart rate were continuously monitored. 2DE evaluation was performed before and at 10, 20, 30, and 60 min (T10, T20, T30) after epidural lidocaine injection. The left ventricular wall was divided into 16 segments for parasternal long-axis, short-axis and apical four-chamber views. The wall motion of each segment was graded on a scale from 1 (dyskinesia) to 6 (hyperkinesia), with 5 representing normal motion. A decrease in segmental wall motion ≥ 2 grades was considered indicative of ischemia. Plasma lidocaine and catecholamine levels were measured before and 10, 20, and 60 min after epidural lidocaine injection. Peak plasma lidocaine levels in groups 1 and 2 were 2.79 ± 1.06 µg/ml (mean ± SD) and 2.58 ± 1.48 µg/ml at 10 min, respectively (NS). Plasma epinephrine and norepinephrine levels were unchanged from baseline. Systolic pressures decreased significantly in group 2 from T10 to T20. Diastolic pressure decreased significantly in the same group from T30 to T20, and in group 1 only at T10. Mean arterial pressure decreased significantly in both groups at T30, without change in heart rate. Segmental wall motion (SWM) in healthy patients increased by 1 grade in 11.4 ± 3.2 segments (mean ± SD), peaking at T20. Preanesthetic segmental wall motion abnormalities (SWMA) were detected in eight of ten CAD patients. During anesthesia SWM decreased in all ten CAD patients in 4.6 ± 2.0 segments (mean ± SD) at T20 (including segments with previous abnormalities, and those adjacent to current abnormalities) and increased in 2.4 ± 1.8 of segments having normal motion before epidural placement. SWMA indicative of ischemia (SWM decrease ≥ 2 grades) were detected in four CAD patients, three with and one without preanesthetic SWMA. SWM activity returned to near-baseline at T30 in both groups. Lumbar epidural anesthesia appears to affect SWM transiently in patients having either healthy or impaired myocardium. Hyperkinesia in the normal segments in both groups may be attributable to changes in loading conditions of the left ventricle. Decreases in SWM ≥ 2 grades in CAD patients may be due to decreased coronary perfusion pressure (suggested by the significant decline in arterial blood pressures) and may indicate transient ischemia in patients undergoing lumbar epidural anesthesia. (Key words: Anesthetic technique: epidural. Anesthetics, local: lidocaine. Monitoring: two-dimensional precordial echocardiography; segmental wall motion; hemodynamics. Heart, complications: coronary artery disease; wall motion abnormalities.)

Patients with coronary artery disease (CAD) may develop myocardial ischemia during anesthesia and surgery due to either an increase in myocardial oxygen demand or decreased myocardial oxygen supply. Lumbar epidural anesthesia (LEA) slightly modifies left ventricular afterload according to the extent of sympathetic blockade but markedly decreases preload (secondary to decreased venous return) without impairing left ventricular contractility. Recently, LEA was reported to improve ejection fraction and regional left ventricular function without changing heart rate in patients with CAD. However, regional anesthesia inducing hypotension could decrease coronary blood flow.

Because decreased coronary artery blood flow induces changes in myocardial segmental wall motion (SWM), we studied these conflicting findings by comparing the effect of lumbar epidural anesthesia on wall motion in healthy and CAD patients. We used two-dimensional precordial echocardiography (2DE) to analyze wall motion because it is a technique that is specific, sensitive, and reliable in detecting transient myocardial segmental wall motion abnormalities (SWMA). Sixteen segments of the left ventricle were viewed in each patient. Each segment was analyzed for wall motion and thickening at specified intervals during lumbar epidural anesthesia.

Materials and Methods

After approval from the Ethical Committee of our institution, we studied 15 patients scheduled to undergo lower abdominal or peripheral vascular surgery during LEA. Patients were divided into two groups. Group 1 included five ASA PS 1 patients (control group) and group 2 included ten patients with CAD documented by a history of angina pectoris, previous myocardial infarction, dipyridamole thallium scintigraphic evidence of hypofixation with or without redistribution of the isotope, and/or coronary arteriographic evidence of critical stenosis ≥ 75%
in at least one main coronary vessel. Patients having congestive heart failure and a cardiothoracic ratio ≥ 0.55 and those receiving inotropic or vasoconstrictive agents were excluded from study.

All patients were studied prior to surgery. We inserted an 18-G catheter into a peripheral vein for iv infusion of colloid. A 22-G catheter was inserted into the radial artery for sampling of plasma anesthetic and catecholamine levels. A catheter was inserted into the epidural space through an 18-G Tuohy needle via the L3–4 or L4–5 interspace and 12.5 ml of 2% lidocaine HCl solution was injected. Sensory blockade was assessed by pin prick every 5 min after lidocaine injection.

Samples were drawn for determination of plasma lidocaine and catecholamine levels (epinephrine and norepinephrine) before and at 10, 20, and 60 min (T₁₀–T₆₀) after lidocaine administration. We measured plasma lidocaine concentrations using an immunofluorescence technique (Abbott TDX). Plasma catecholamine samples were collected into chilled, heparinized tubes and immediately centrifuged at 4°C for 10 min. The separated plasma was then stored at −80°C until assayed. Plasma epinephrine and norepinephrine concentrations were calculated twice for each sample, using a double-isotope radioenzymatic assay having intracoefficients and intercoefficients of variation of 8% and 15%, respectively.

We measured systemic arterial pressure and heart rate every 5 min using an automatic blood pressure cuff (Dinamap-Critikon). An infusion of colloid (Plasmion) was administered when mean arterial pressure decreased ≥20% below preanesthetic values. Patients requiring >750 ml of colloid were excluded from study. Electrocardiographic monitoring (lead CM5) was used during the study for routine cardiac management. All hemodynamic and plasma anesthetic and catecholamine data were analyzed using ANOVA and a modified Student’s t test; P < 0.05 was considered significant.

2DE were obtained using an ATL Mark 600 echocardiograph. All echocardiograms were recorded in standard parasternal long-axis and short-axis, and apical fourchamber views. The short-axis view was recorded at the level of the midpapillary muscles. We divided the myocardial wall into 16 segments using the method of Edwards et al. (fig. 1) and graded segmental wall motion as follows: 1 = dyskinesia, no shortening of the segment and wall thinning; 2 = akinesia, no segment shortening and no wall thickening; 3 = severe hypokinesia, <10% segment shortening and minimal thickening; 4 = mild hypokinesia, 10–30% shortening of the segment and slightly reduced thickening; 5 = normal activity, >50% segment shortening and normal thickening; 6 = hyperkinesia, >30% segment shortening and thickening above baseline. A decrease in SWM ≥ 2 grades was considered indicative of ischemia.

**Results**

Group 1 was comprised of three men and two women whose mean age was 38.8 ± 23 yr (mean ± SD), and group 2 included ten men with a mean age of 66.2 ± 10 yr. Sensory blockade in all patients extended from S₅ to T₆–₁₂. Mean systemic arterial pressure decreased significantly at 30 min (T₃₀) in both groups, from 97.0 ± 14.7 to 88.0 ± 14.3 mmHg in group 1 and from 100.4 ± 14.9 to 82.9 ± 15.5 mmHg in group 2 (table 1). Diastolic arterial pressure decreased significantly in group 1 at T₁₀ after induction, and in group 2 at T₂₀–T₆₀. Systolic arterial pressure decreased significantly only in group 2 at T₁₀–T₆₀. Heart rate did not change in either group. Group 1 received a mean colloid infusion of 484 ± 120 ml (mean ± SD) and group 2 received a mean infusion of 507 ± 117 ml.

Plasma lidocaine concentrations were similar in both groups, peaking at 10 min after injection (T₁₀) in each. Plasma catecholamines concentrations did not change significantly (table 2).

Group 1 had no preexisting cardiac problems. Four CAD patients had a previous myocardial infarction and
eight had angina pectoris. One patient in group 2 had scintigraphic evidence of redistribution in regional blood flow, suggesting ischemia. Two CAD patients were receiving beta-adrenergic blocking drugs, four were taking calcium channel-blocking drugs, and five were receiving long-acting nitrates.

We analyzed 375 of 400 echocardiograms from group 1 and 695 of 800 from group 2 for wall motion and thickening. Segments that could not be analyzed were primarily parasternal long-axis views of the apex.

Group 1 had normal pre-epidural echocardiograms, and no evidence of SWMA during or after epidural injection. The pre-epidural echocardiograms of CAD patients revealed eight with SWMA in some segments. Five patients had SWMA in more than one region, two had posterior SWMA, and one had septal SWMA. Of those with SWMA in more than one region, four had apical and septal SWMA and one had anterior and septal SWMA. All preanesthetic SWMA correlated with a history of one or more of the following: ECG distribution of ST-T depression, Q-waves, angiocardioangiographic defects, and/or coronary arteriographic evidence of stenosis (table 3).

After epidural lidocaine injection, SWM activity increased within the first 20 min (T20) in all patients in the control group (n = 5). This hyperkinesia was apparent in 11.4 ± 3.2 (mean ± SD) of the 16 segments analyzed per patient. At 60 min (T60) SWM activity returned to pre-epidural levels (fig. 2). SWM activity in CAD patients decreased ≥2 grades in four patients and ≤1 grade in the other six patients, for a mean of 4.6 ± 2 segments per patient. SWMA were most severe in all CAD patients at T20, remained severe at T30, and returned close to baseline at T60 (fig. 3). SWMA occurred extensively at T10 and T20 and in a less extent at T30 (fig. 4), at the same time a significant decrease in systolic arterial pressure occurred. Four CAD patients had decreases in SWM ≥ 2 grades, suggesting ischemia; three of these had pre-epidural SWMA and one did not. Among the four CAD patients with SWMA ≥ 2 grades, two patients had a decrease in systolic arterial pressure ≥ 20% below the preanesthetic value. Hyperkinesia was apparent in 2.4 ± 1.8 segments per CAD patient; none of these segments were initially hypokinetic. Wall motion abnormalities obtained in one view were also detected in other views of the same segment.

One CAD patient experienced angina pectoris 20 min after lidocaine injection that was undetected by ECG lead CM5 but documented by echocardiography as severe hypokinesia in the septal region. At this threshold colloid infusion rate was increased and the patient's chest pain and echocardiographic abnormalities resolved at T60 with a rise in blood pressure induced by fluid infusion. Angioscintigraphy performed later documented a perfusion defect in the septal region. SWM in this patient decreased by ≥2 grades in this region.

The two echocardiographers agreed in grading SWM in 96% of segments analyzed for group 1 and 92% for

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### Table 1. Hemodynamic Changes during Lumbar Epidural Anesthesia

<table>
<thead>
<tr>
<th>Variables</th>
<th>Patient Group</th>
<th>T0</th>
<th>T10</th>
<th>T20</th>
<th>T30</th>
<th>T60</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats/min)</td>
<td>CAD</td>
<td>76.8 ± 8.2</td>
<td>74.3 ± 8.2</td>
<td>68.8 ± 8.0</td>
<td>70.3 ± 9.5</td>
<td>69.4 ± 10.9</td>
</tr>
<tr>
<td></td>
<td>H</td>
<td>80.0 ± 16.5</td>
<td>81.2 ± 20.5</td>
<td>86.0 ± 21.3</td>
<td>86.0 ± 21.2</td>
<td>77.6 ± 17.3</td>
</tr>
<tr>
<td>Systolic arterial pressure (mmHg)</td>
<td>CAD</td>
<td>137.8 ± 20.0</td>
<td>119.0 ± 16.0*</td>
<td>114.2 ± 21.6</td>
<td>121.4 ± 23.9†</td>
<td>128.8 ± 22.9†</td>
</tr>
<tr>
<td></td>
<td>H</td>
<td>130.0 ± 20.0</td>
<td>130.0 ± 23.8</td>
<td>127.0 ± 18.0</td>
<td>122.0 ± 21.3</td>
<td>126.0 ± 15.2</td>
</tr>
<tr>
<td>Diastolic arterial pressure (mmHg)</td>
<td>CAD</td>
<td>79.6 ± 12.5</td>
<td>75.2 ± 11.1</td>
<td>67.1 ± 13.4†</td>
<td>66.4 ± 13.9†</td>
<td>72.0 ± 12.8†</td>
</tr>
<tr>
<td></td>
<td>H</td>
<td>80.8 ± 14.3</td>
<td>72.0 ± 12.5†</td>
<td>73.0 ± 12.5</td>
<td>77.0 ± 13.0</td>
<td>79.0 ± 15.6</td>
</tr>
<tr>
<td>Mean arterial pressure (mmHg)</td>
<td>CAD</td>
<td>100.4 ± 14.9</td>
<td>89.8 ± 14.0*</td>
<td>83.3 ± 14.7*</td>
<td>82.9 ± 15.5*</td>
<td>90.3 ± 12.9†</td>
</tr>
<tr>
<td></td>
<td>H</td>
<td>97.0 ± 14.7</td>
<td>94.1 ± 18.3</td>
<td>90.3 ± 13.9</td>
<td>88.0 ± 14.3†</td>
<td>92.6 ± 13.0</td>
</tr>
</tbody>
</table>

H = healthy.

* P < 0.02.

† P < 0.05.

‡ P < 0.001.

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### Table 2. Plasma Lidocaine and Catecholamine Concentrations

<table>
<thead>
<tr>
<th>Patient Group</th>
<th>T0</th>
<th>T10</th>
<th>T20</th>
<th>T30</th>
<th>T60</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lidocaine (µg/ml)</td>
<td>CAD</td>
<td>0</td>
<td>2.79 ± 1.06</td>
<td>2.47 ± 0.96</td>
<td>1.62 ± 0.65</td>
</tr>
<tr>
<td></td>
<td>H</td>
<td>0</td>
<td>2.58 ± 1.48</td>
<td>2.12 ± 0.93</td>
<td>1.67 ± 0.77</td>
</tr>
<tr>
<td>Norepinephrine (µg/ml)</td>
<td>CAD</td>
<td>1.086 ± 387</td>
<td>968 ± 378</td>
<td>755 ± 279</td>
<td>798 ± 350</td>
</tr>
<tr>
<td></td>
<td>H</td>
<td>1.049 ± 325</td>
<td>840 ± 407</td>
<td>759 ± 333</td>
<td>1062 ± 426</td>
</tr>
<tr>
<td>Epinephrine (µg/ml)</td>
<td>CAD</td>
<td>119 ± 84</td>
<td>84 ± 55</td>
<td>66 ± 65</td>
<td>83 ± 72</td>
</tr>
<tr>
<td></td>
<td>H</td>
<td>116 ± 78</td>
<td>76 ± 102</td>
<td>56 ± 41</td>
<td>83 ± 72</td>
</tr>
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</table>

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### Table 3. Clinical Features of CAD Patients

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (y)</th>
<th>Cardiac History</th>
<th>ECG</th>
<th>2DE, Site, CA</th>
<th>SWMA Control</th>
<th>SWMA during LEA</th>
</tr>
</thead>
<tbody>
<tr>
<td>1*</td>
<td>57</td>
<td>Angina pectoris</td>
<td>Thallium redistribution septal region</td>
<td>No SWMA</td>
<td>↓↓ SWM septal, extension apical segment</td>
<td></td>
</tr>
<tr>
<td>2*</td>
<td>51</td>
<td>MI</td>
<td>Repolarization abnormalities Q5 V1-V3</td>
<td>—</td>
<td>Anteroseptal hypokinesia</td>
<td>↓↓ SWM same region, extension apical segment</td>
</tr>
<tr>
<td>3</td>
<td>59</td>
<td>Angina pectoris</td>
<td>Repolarization abnormalities ST depression D1VLV1-V5</td>
<td>CA with stenosis &gt;75% RCA, LAD, CFX</td>
<td>Septal hypokinesia</td>
<td>↓↓ SWM same region, extension lateral segment</td>
</tr>
<tr>
<td>4</td>
<td>80</td>
<td>Angina pectoris</td>
<td>Repolarization abnormalities T waves abnormalities V5 V6</td>
<td>—</td>
<td>Posterior hypokinesia</td>
<td>↓↓ SWM same and adjacent segments</td>
</tr>
<tr>
<td>5</td>
<td>66</td>
<td>Angina pectoris</td>
<td>Repolarization abnormalities ST depression D1 VL V5 V6</td>
<td>—</td>
<td>No SWMA</td>
<td>↓ lateropical WM</td>
</tr>
<tr>
<td>6</td>
<td>67</td>
<td>Angina pectoris</td>
<td>Repolarization abnormalities D1 VL V1-V6</td>
<td>—</td>
<td>Septoapical hypokinesia</td>
<td>↓ septal WM, extension same wall</td>
</tr>
<tr>
<td>7*</td>
<td>75</td>
<td>MI</td>
<td>Repolarization abnormalities Q5 D1 VL V5-V6</td>
<td>—</td>
<td>Posterior hypokinesia</td>
<td>↓↓ posterior WM, extension lateral segment</td>
</tr>
<tr>
<td>8*</td>
<td>69</td>
<td>MI</td>
<td>Repolarization abnormalities Q5 V1-V3</td>
<td>2DE apical akinesia</td>
<td>Septoapical hypokinesia</td>
<td>↓↓ in septal WM, extension ant + apical wall</td>
</tr>
<tr>
<td>9</td>
<td>78</td>
<td>MI</td>
<td>Repolarization abnormalities Q wave D2 D3 VF</td>
<td>CA with stenosis &gt;75% RCA and LAD</td>
<td>Septoapical hypokinesia</td>
<td>↓ SWM same region</td>
</tr>
<tr>
<td>10</td>
<td>60</td>
<td>Angina pectoris</td>
<td>Repolarization abnormalities ST depression D1VLV4-V6</td>
<td>CA with stenosis &gt;75% RCA and LAD</td>
<td>Septoapical hypokinesia</td>
<td>↓↓ SWM same region, extension lateral segment</td>
</tr>
</tbody>
</table>

* Decreased wall motion ≥2 grades.

Group 2 for the two evaluations at 6-month intervals. The 8% disagreements in group 2 concerned in all the cases disagreements by one grade concerning either normal, hypokinetic, or adjacent segments. Concerning the four patients judged as having ischemia, there was total agreement, even at 6-month intervals, between the two readers about the segments with SWMA ≥ 2 grades and disagreements for two adjacent segments that were graded as normal versus mild hypokinetic. Disagreements between the two observers were resolved by the senior of the two echocardiographers still unaware of the patient’s clinical courses.

**Fig. 2.** The number of segments (±SD) with increased wall motion during lumbar epidural anesthesia in healthy patients. The maximum changes occurred 20 min after the induction of anesthesia. At 60 min, SWM activity returned to baseline.

**Fig. 3.** The number of segments (±SD) with decreased wall motion during lumbar epidural anesthesia in CAD patients. SWMA were most severe at T30 and returned close to baseline at T60. Segments demonstrating increased wall motion at T15 were nonischemic.
FIG. 4. The dynamic percentage wall motion changes in CAD patients group over time. The maximum changes occurred at T10 and T30 and came back to near-normal at T60.

Discussion

Induction of LEA in patients with CAD appears to worsen SWMA in regions with previous abnormalities and induce SWMA or hypokinesia in previously normal segments. Healthy patients demonstrate hyperkinetic activity in response to this type of anesthesia.

SWM during LEA is affected by changes in left ventricular loading conditions and in coronary perfusion pressure.\textsuperscript{14,18} We observed a decrease in mean arterial pressure with no changes in heart rate in both groups, suggesting a decrease in preload. The decrease in pressure was more significant in CAD patients, suggesting a limited adaptation to the decrease of venous return. Such decreases in pressure also reduce coronary artery perfusion pressure, which may jeopardize myocardial oxygen supply in CAD patients, thereby inducing changes in SWM.\textsuperscript{16–20} In four of the CAD patients, SWM worsened by $\geq 2$ grades, indicating ischemia. In animals even a mild decrease in perfusion pressure can cause ischemia by significantly reducing coronary blood flow in the presence of coronary stenosis.\textsuperscript{21}

Another explanation for worsening of SWMA in our CAD patients may be transient coronary artery constriction caused by a sympathetic response to lumbar epidural blockade in the nonanesthetized region. This hypothesis seems unlikely because tachycardia indicative of sympathetic activation did not occur in our patients and because vasodilation induced by LEA is reported to extend beyond the sensory level.\textsuperscript{22,23} Sensory blockade in our patients extended to T6–12 and heart rate remained stable. SWM worsening of $\leq 1$ grade in the other six CAD patients may reflect the limit of the method for absolute detection of minor changes in wall motion.

Normal segments in healthy patients became hyperkinetic, probably due to the change in afterload secondary to lumbar epidural anesthesia.\textsuperscript{24} Normal segments in CAD patients became moderately hypokinetic ($\leq 1$ grade) when adjacent to abnormal tissue and hyperkinetic when isolated. The hyperkinetic segments in CAD patients may be those having adapted coronary perfusion,\textsuperscript{15,24} similar to those in healthy patients. Alternatively, lidocaine may stimulate the myocardium via a centrally mediated mechanism,\textsuperscript{25,26} which could increase wall motion. However, plasma catecholamine levels did not increase enough to indicate a lidocaine-stimulating effect. The evidence of moderate hypokinesia in normal segments adjacent to hypokinetic tissues in CAD patients may reflect mild ischemia or the limits of echocardiographic detection.

One patient in the CAD group had chest pain accompanied by SWMA 20 min after lidocaine injection, without changes in ECG lead CM5. The echocardiogram suggested ischemia (decrease in SWM $\geq 2$ grades). Although the use of multiple ECG leads might have demonstrated ST-T-segment changes to corroborate echocardiographic evidence of ischemia, we elected to use the ECG for only routine cardiac monitoring throughout the study. Additionally, echocardiography is reported to be more sensitive than multiple-lead ECG in the diagnosis of ischemia.\textsuperscript{27}

In a previous study, Baron et al.\textsuperscript{3} demonstrated that SWM, evaluated by radionuclide angiography, was improved in CAD patients after LEA extending to T10. One explanation for the difference between their results and ours may be the difference in study population. We included patients having hypertension and unstable angina, whereas Baron et al.\textsuperscript{3} studied only patients with stable angina. Consequently, their patients may have differed from ours in the severity of coronary artery lesions; patients who have severe stenosis are more likely to develop myocardial ischemia secondary to hypotension induced by LEA. This hypotension can compromise coronary blood flow and so myocardial oxygen supply, despite a decrease in myocardial oxygen consumption. The resulting inadequacy between oxygen supply and demand may cause ischemia. A second factor contributing to the difference in findings may be sampling times. We recorded SWM sooner after epidural lidocaine injection than did Baron et al.,\textsuperscript{3} i.e., at 10 versus 30 min. By 30 min the initial SWMA we observed began to disappear. Thus, Baron et al.\textsuperscript{3} may have missed SWM modifications accompanied by the significant hemodynamic changes that generally occur during the first 30 min following epidural lidocaine injection. A third factor is the difference in techniques. Changes in segmental ejection fraction measured by radionuclide angiography may reflect changes in loading conditions but not changes indicative of myocardial ischemia.\textsuperscript{28,29}

Limitations of the Method

Although 2DE is a reliable technique for detecting SWMA,\textsuperscript{9} it is limited in measuring wall thickening and
endocardial wall motion. To determine wall thickening requires good epicardial image resolution, which is difficult to obtain using precordial echocardiography. Our estimates of wall thickening were therefore qualitative. Our observers, however, had at least 6 yr experience in analyzing precordial echocardiograms. Additionally, each observer was blinded to the analysis of the other to limit subjective analysis.

Measuring endocardial wall motion requires a reference axis. During systole the center of mass of the left ventricle changes by translation and rotation. Use of either a floating or fixed axis permits analysis but can result in underestimation or overestimation of the extent of abnormal wall motion. We have used a fixed axis in this study because it is reported to provide more accurate analysis of wall motion indicative of ischemia. However, there is no consensus on the optimal centroid for evaluation of wall motion.

Overestimation may occur because of “tethering.” Several studies have shown that segments of myocardium adjacent to ischemic or infarcted segments may display major contraction abnormalities attributable to proximity to the affected segment. This effect may be related in part to mechanical tethering and seems to concern only 5% of the myocardial circumference.

In conclusion, LEA significantly decreased blood pressure in patients with CAD in whom moderate to severe worsening of wall motion activity simultaneously occurred. Healthy patients also had transiently decreased blood pressure and a global increase in segmental wall motion. Thus, hemodynamic deficits due to LEA, particularly changes in diastolic arterial pressure, may impair myocardial perfusion. This effect could aggravate the potential for ischemia in CAD patients who may already have inadequate coronary perfusion and oxygen supply.

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