Physiological Early Diastolic Intraventricular Pressure Gradient Is Lost During Acute Myocardial Ischemia

Michael Courtois, MA, Sándor J. Kovács, PhD, MD, and Philip A. Ludbrook, MB, BS, FRACP

A consistent pattern of intraventricular regional pressure gradients exists under physiological conditions during the rapid filling phase of diastole in the normal dog left ventricle. We hypothesized that this pressure gradient pattern is caused, in part, by early diastolic recoil of the left ventricular walls in conjunction with release of elastic potential energy stored during systole, generating suction and thus contributing to diastolic filling. If so, any condition that interferes with normal regional systolic function might be expected to modify the pattern of the normal early diastolic intraventricular pressure gradients. Accordingly, the present study was designed to determine whether acutely induced regional systolic left ventricular mechanical dysfunction is accompanied by changes in the pattern of the early diastolic intraventricular pressure gradients.

Acute myocardial ischemia was induced by balloon occlusion of the left anterior descending coronary artery (LAD) in nine anesthetized closed-chest dogs. The maximum early diastolic intraventricular pressure gradient (MIVP) was measured between the mid-left ventricle and apex with a dual-sensor micromanometer (3-cm spacing between the sensors) before and 20 minutes after LAD occlusion. Ejection fraction (EF) and number of dyskinetic chords (DChords) were measured from left ventricular contrast ventriculograms. Twenty minutes after LAD occlusion, the nine dogs evidenced significant changes in EF (56±10% to 37±8%), DChords (0±0 to 17±16 chords), left ventricular minimum pressure (−1.7±0.5 to 0±1.5 mm Hg), left ventricular end-diastolic pressure (4.2±1.2 to 5.9±2.2 mm Hg), and heart rate (90±17 to 103±18 beats/min). These changes were accompanied by a marked decline in MIVP (1.2±0.5 to 0.6±0.6 mm Hg). All changes are significant at the 0.05 level. Through both baseline and occlusion conditions, a significant linear relation between MIVP and EF was found for the nine dogs (r=0.76, p<0.001). For baseline data alone, the relation between MIVP and EF was also strong (r=0.81, p<0.008). After LAD occlusion, MIVP and EF did not correlate significantly (r=0.53, p=NS), whereas MIVP and the DChords did (r=0.80, p<0.010). These results strongly suggest that the left ventricular early diastolic apical intraventricular pressure gradient is causally related to left ventricular systolic function, and that in the setting of a severe regional wall motion abnormality, factors operate that are not explained by single-plane angiography and single-site pressure measurement.

We conclude that systolic dysfunction, engendered by extensive anterior myocardial ischemia, is associated with the attenuation, loss, or even reversal of the MIVP during the rapid filling phase of diastole. We speculate that these changes are probably related to the loss of myocardium available to store and release energy in the form of elastic recoil, and to changes known to occur in the pattern of blood flow during systole, especially in ventricles exhibiting extensive regions of dyskinesis. (Circulation 1990;81:1688–1696)
generates suction and contributes to normal filling.\textsuperscript{2} If so, then any condition that interferes with the normal sequence of regional contraction might be expected to alter the physiological early diastolic intraventricular pressure gradient pattern. Such an alteration might contribute to altered intraventricular flow and thrombus formation described in cardiac disorders affecting systolic function, such as acute myocardial infarction and dilated cardiomyopathy.\textsuperscript{3–7} The present study was performed to determine whether acutely induced regional left ventricular systolic mechanical dysfunction is accompanied by changes in the pattern of the early diastolic intraventricular pressure gradients.

**Methods**

Nine mongrel dogs of either gender weighing 27–34 kg (30±2 kg) were sedated with morphine (1 mg/kg s.c.) 30 minutes before induction of general anesthesia with sodium pentothal (12.5 mg/kg i.v.) and α-chloralose (100 mg/kg i.v.). Each dog was intubated and ventilated with room air using a Harvard respirator (Harvard Apparatus, South Natick, Massachusetts). The right jugular vein, right and left common carotid arteries, and right and left femoral arteries were isolated and a valved sheath (Hemaquet 8F, USCI) was placed in each. A bolus injection of heparin sodium (4,000–5,000 USP units) was then administered intravenously. A Swan-Ganz thermodilution catheter (model 93A-131-7F, American Edwards) was directed under fluoroscopy from the jugular vein to the superior vena cava, a micromanometric catheter (model 484A-8F, Millar Instruments, Houston, Texas) was directed from the right femoral artery to the aortic arch, and a dual-sensor micromanometric catheter with 3-cm spacing between the sensors (model PC 771-7F, Millar Instruments) was directed from the right carotid artery into the left ventricle (LV) so that the distal transducer was located close to the cardiac apex, in a position free of ectopy. After recording baseline intraventricular pressures, the catheter was left in place for the remainder of the experiment. To optimize the stability of the micromanometric signals, catheters were immersed in saline for a minimum of 12 hours before use. To record absolute left ventricular pressures, referenced solely to atmospheric pressure rather than to an external fluid-filled transducer signal that is highly dependent on the height of the external transducer relative to the height of the heart, the dual-sensor catheter was placed in a dry graduated cylinder that was immersed in a water bath warmed to 36.5–38°C, the precise temperature dependent on the temperature of the dog. Because micromanometric sensors have been shown to be sensitive to light, the room lights were extinguished before zeroing. For the dual-sensor catheter used in this experiment, darkness caused both pressure signals to shift downward 0.3 mm Hg. When the dual sensor catheter was placed in the LV, small hydrostatic pressure differences consistently existed between the two signals (Figure 1, upper panel). Arbitrarily, the proximal pressure signal was selected as the standard; the apical pressure signal was aligned with the proximal signal during the late diastatic filling period (Figure 1, lower panel). In the presence of rapid heart rates, the alignment of pressures was accomplished during the long diastatic period occurring after premature ventricular contractions stimulated mechanically by the Swan-Ganz catheter positioned in the right ventricle.\textsuperscript{8,9} At the conclusion of the experiment, the dual sensor catheter was replaced in the graduated cylinder, the room lights were extinguished, and the two zero baselines were checked. In no experiment did the signal from either sensor drift more than 1.0 mm Hg from the original zero baseline.

A low-gain pressure signal from the aortic micromanometric catheter (100 mm Hg=10.0 cm), and two high gain pressure signals from the dual sensor catheter (20 mm Hg=15 cm) were transmitted to a photographic recorder (model 1508B, Honeywell Visi- corder). Pressure recordings were made at a chart speed of 100 mm/sec. Myocardial ischemia was induced by inflation of a 3.6-mm angioplasty balloon in the proximal left anterior descending coronary artery (LAD) as previously described.\textsuperscript{10} Left ventriculograms were recorded in the left lateral projection; 22 ml of contrast medium (Omnipaque 350, Winthrop) was injected through a pigtail catheter (Cordis 7F) positioned in the LV through the left femoral artery.

All hemodynamic and ventriculographic measurements were recorded during apnea with the animal in the supine position at baseline, and then again after 20 minutes of occlusion of the LAD. Before coronary occlusion, a bolus injection of lidocaine (50 mg) was given intravenously. Core body temperature was maintained constant with use of a circulating water (38°C) heating pad and was monitored continuously with the Swan-Ganz catheter thermistor. In no experiment did the temperature drop below 36.2°C. Blood gases were measured at repeated intervals, and ventilator respiratory rate and volume were adjusted accordingly.

**Analysis of Data**

The following pressure measurements were obtained from the micromanometric pressure transducer recordings: mean aortic pressure, minimum left ventricular pressure recorded from the apical transducer, the maximum early diastolic intraventricular pressure gradient (MIVP) measured between the apical and 3-cm sensors, and left ventricular end-diastolic pressure recorded from the apical transducer.

Left ventriculograms were assessed quantitatively by analysis of left ventricular silhouettes recorded on cine film at a rate of 30 frames/sec. End-systolic and end-diastolic images were traced together with a magnification correction grid. This information was then digitized by a hand-held digitizer (MAC- TABLET, Summagraphics Corp., Seymour, Connecticut) interfaced to a personal computer (Macintosh SE, Apple Computers, Inc.) and transferred by telephone modem to a computer (VAX 750) located at the University of Washington in Seattle.
The method of analysis has been described elsewhere in detail. Ventriculographic variables reported in the present study were ejection fraction (EF) and number of dyskinetic chords. Briefly, 100 chords are constructed by the computer, perpendicular to a centerline drawn between the end-systolic and end-diastolic ventricular contours. A dyskinetic chord is defined as a chord with an end-systolic endocardial border point that is located spatially beyond its corresponding end-diastolic endocardial border point.

Differences between baseline and occlusion conditions were tested for significant changes with the paired t test. Significant changes in data and correlation coefficients were set at p values less than .05.

**Results**

*Differences in Left Ventricular Systolic Function, Hemodynamics, and Heart Rate Before and 20 Minutes After LAD Occlusion*

After LAD occlusion, significant statistical differences existed for the nine dogs in terms of EF, the number of dyskinetic chords, heart rate, left ventricular end-diastolic pressure, left ventricular minimum pressure, and the MIVP recorded in the left ventricular apex (Table 1). Only changes in mean aortic pressure failed to reach significance.

**Effect of 20-Minute LAD Occlusion on the Maximum Intraventricular Pressure Gradient in Individual Dogs**

In five of nine dogs subjected to balloon occlusion of the LAD, the early intraventricular pressure gradient was maintained at baseline or near baseline.

**TABLE 1. Group Data for Nine Dogs at Baseline and After a 20-Minute Occlusion of the Left Anterior Descending Coronary Artery**

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Occlusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>EF (%)</td>
<td>56±10</td>
<td>37±8*</td>
</tr>
<tr>
<td>Dys chords (n)</td>
<td>0±0</td>
<td>17±16*</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>90±17</td>
<td>103±18*</td>
</tr>
<tr>
<td>AOM (mm Hg)</td>
<td>90±17</td>
<td>98±16</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>4.2±1.2</td>
<td>5.9±2.2*</td>
</tr>
<tr>
<td>LVPmin (mm Hg)</td>
<td>−1.7±0.5</td>
<td>0.0±1.5*</td>
</tr>
<tr>
<td>MIVP (mm Hg)</td>
<td>1.2±0.5</td>
<td>0.6±0.6*</td>
</tr>
</tbody>
</table>

Values are mean±SD. EF, ejection fraction; Dys chords, number of dyskinetic left ventricular wall segments; HR, heart rate; AOM, mean aortic pressure; LVEDP, left ventricular end-diastolic pressure; LVPmin, minimum left ventricular pressure of apical transducer; MIVP, maximum early diastolic intraventricular pressure gradient. *p<0.05 as compared with baseline.
levels (Figure 2). In the remaining four dogs, a loss of more than 50% of the baseline early intraventricular pressure gradient was observed. In one of these four dogs, the pressure gradient reversed slightly, with the higher pressure being recorded at the apical transducer (Figure 3). Individual data points for the nine dogs for EF, MIVP, and number of dyskinetic chords are presented in Table 2.

Relation of Systolic Function to the Maximum Intraventricular Pressure Gradient

Linear correlation between EF and MIVP through both baseline and LAD occlusion conditions for the nine dogs indicates a significant relation between systolic function and the magnitude of MIVP ($r=0.76, p<0.001$) (Figure 4). Because visual inspection of Figure 4 suggests that scatter is greater after coronary occlusion, separate correlation coefficients were computed for the nine data points obtained before (Figure 5) and after LAD occlusion (Figure 6). Before coronary occlusion, a strong correlation is noted between EF and MIVP ($r=0.81, p<0.008$). After LAD occlusion, this relation is no longer significant ($r=0.53, p=NS$). Linear correlation after coronary occlusion between MIVP and the number of dyskinetic chords was significant ($r=0.80, p<0.010$) (Figure 7).

Discussion

The present results confirm previous observations by Ling et al$^1$ and ourselves$^2$ of a consistent pattern of regional pressure differences during early diastole in the normal canine LV. More importantly, they extend our understanding of this phenomenon by demonstrating that the physiological early diastolic intraventricular pressure gradient pattern can be attenuated, lost entirely, or even reversed as a consequence of extensive regional systolic dysfunction.

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**Figure 2.** Recordings showing simultaneous intraventricular pressures (mm Hg) recorded in a dog by using dual sensor micromanometric catheter (3-cm spacing). At baseline (upper panel), ejection fraction was 55%, and maximum intraventricular pressure gradient was 1.0 mm Hg. After left anterior descending coronary artery (LAD) occlusion (lower panel), ejection fraction declined moderately to 40% with 15 dyskinetic chords, left ventricular minimum and end-diastolic pressures increased, but normal early diastolic intraventricular pressure gradient was maintained (1.0 mm Hg). LVP, left ventricular pressure; ECG, electrocardiogram.
induced acutely by abrupt coronary artery occlusion. Although all pressure measurements were made during brief apnea at end expiration to avoid the effects of respiratory variation on intracardiac hemodynamics, both the normal pattern of the early intraventricular gradient (Figure 8, upper panel) and changes in the gradient pattern in some dogs during coronary occlusion (Figure 8, lower panel) were consistent throughout the entire respiratory cycle.

Based on measurement in our laboratory of left ventricular intraventricular pressure gradients in the apical region in 20 normal dogs (11 dogs in our previously published work\(^2\) and nine dogs in the present study), a consistent intraventricular gradient pattern (represented by Figures 1 [lower panel], 2, 3 [upper panel], and 8 [upper panel] in the present study and by Figure 10 in our previously published study\(^2\)) is evident throughout a wide range of heart rates (heart rate range, 59–138 beats/min), contractility (peak positive dP/dt range, 1,200–2,700 mm Hg/sec), preload (first crossover point of atrial and ventricular pressures range, 2.5–8.0 mm Hg), and afterload (aortic diastolic pressure range, 55–120 mm Hg; peak left ventricular pressure range, 85–165 mm Hg). We have recorded patterns of intraventricular pressure similar to those depicted in Figures 3 (lower panel) and 8 (lower panel) only in dogs with extensive anterior-apical wall dysfunction resulting from occlusion of the LAD. This evidence, in conjunction with the significant linear relation between EF and MIVP obtained in the present study, indicates that the normal MIVP in the apical region of the heart is related to left ventricular systolic wall function.

It is now well recognized that the driving force for early diastolic transmitral flow is the atrioventricular
TABLE 2. Individual Data Points for the Nine Dogs at Baseline and After 20-Minute Occlusion of the LAD Coronary Artery

<table>
<thead>
<tr>
<th>Dog</th>
<th>Condition</th>
<th>MIVP (mm Hg)</th>
<th>EF (%)</th>
<th>Dys chords (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Baseline</td>
<td>1.2</td>
<td>42</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Occlusion</td>
<td>1.0</td>
<td>40</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>Baseline</td>
<td>2.0</td>
<td>69</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Occlusion</td>
<td>1.6</td>
<td>37</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>Baseline</td>
<td>1.2</td>
<td>57</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Occlusion</td>
<td>1.2</td>
<td>47</td>
<td>11</td>
</tr>
<tr>
<td>4</td>
<td>Baseline</td>
<td>1.0</td>
<td>55</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Occlusion</td>
<td>1.0</td>
<td>40</td>
<td>15</td>
</tr>
<tr>
<td>5</td>
<td>Baseline</td>
<td>0.5</td>
<td>45</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Occlusion</td>
<td>0.5</td>
<td>45</td>
<td>0</td>
</tr>
<tr>
<td>6</td>
<td>Baseline</td>
<td>0.9</td>
<td>48</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Occlusion</td>
<td>-0.1</td>
<td>29</td>
<td>45</td>
</tr>
<tr>
<td>7</td>
<td>Baseline</td>
<td>1.8</td>
<td>63</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Occlusion</td>
<td>0.0</td>
<td>41</td>
<td>33</td>
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<tr>
<td>8</td>
<td>Baseline</td>
<td>1.6</td>
<td>70</td>
<td>0</td>
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<tr>
<td></td>
<td>Occlusion</td>
<td>0.2</td>
<td>31</td>
<td>19</td>
</tr>
<tr>
<td>9</td>
<td>Baseline</td>
<td>0.8</td>
<td>52</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Occlusion</td>
<td>0.1</td>
<td>24</td>
<td>32</td>
</tr>
</tbody>
</table>

LAD, left anterior descending coronary artery; MIVP, maximum apical intraventricular pressure gradient; EF, percentage of change in ejection fraction from baseline; Dys chords, number of dyskinetic wall segments.

pressure gradient created, in part, by elastic properties of the ventricle that allow for the storage of potential energy in the muscle fibers and surrounding collagen matrix during systole, and the release of this stored energy during diastole.\[13^-17\] We have previously hypothesized that the pattern of the early diastolic intraventricular pressure gradients is also causally related to elastic recoil of the left ventricular walls.\[2\] Thus, acute regional myocardial ischemia, by diminishing the amount of ventricular myocardium available for contraction and subsequent elastic recoil in one region of the ventricle, should result in loss or diminution of the diastolic intraventricular pressure gradient in that region. This, in turn, should lead to the creation of an area or "pocket" in the ventricular cavity where the regional intraventricular pressure gradient that normally aids filling is reduced or absent. In such a region, blood flow would tend to be diminished. This is consistent with recent reports indicating that certain cardiac disorders are associated with abnormal regional intraventricular flow patterns (i.e., regional slow flow or stasis) and the formation of mural thrombus in specific locations within the ventricle.\[3^-7\] Fluid dynamic theory predicts that flow is generated by the presence of regions of differential pressure; it might, thus, be anticipated that such abnormal intraventricular flow patterns should be inducible by alterations in the characteristic pressure gradient pattern between adjacent regions within the left ventricular chamber. Beppu et al,\[6\] using contrast echocardiography in dogs, reported that contrast echoes from the left atrium reached the apex in one diastole in the normally beating heart. After ligation of the LAD and circumflex arteries in the lower one third of the ventricle with consequent apical akiniesis or dyskinesia, blood no longer reached the apical area within one diastole. If, as demonstrated in the present study, the normal apical diastolic pressure differentials are attenuated or absent after coronary occlusion, regional diastolic alterations of blood flow similar to those described by Beppu et al\[6\] would be likely to result.
A characteristic pattern of intraventricular systolic pressure gradients has also been demonstrated within the left ventricular chamber by others.\textsuperscript{18,19} With extensive anterior-apical systolic dysfunction, the physiological intraventricular systolic ejection gradient is also likely to be attenuated or lost. Thus, the absence or diminution of pressure gradients during both the filling and ejection phases can contribute to the relative stasis of blood throughout the entire cardiac cycle, predisposing to formation of thrombus.

Our results indicate a stronger relation after coronary occlusion, between MIVP and the degree of dyskinesis than between MIVP and EF. It is probably true that, as assessed by single-plane angiography, the presence of dyskinetic wall segments can be a stronger predictor of severe global apical dysfunction.
than EF. This finding also seems to be consistent with the observations of Beppu et al, who reported that apical dyskinesis was associated with more extensive flow disruption than was akinesis. Thus, although extensive akinesis can be sufficient to alter the intraventricular pressure gradient and flow pattern, as suggested by the results obtained in dog 2 (Table 2), with an intraventricular gradient that decreased by 20% despite a lack of dyskinetic myocardium, it is reasonable to expect that extensive regions of dyskinesia can lead to an even greater disruption of normal diastolic intraventricular pressure and flow. Frame-by-frame examination of left ventriculograms recorded during induced acute anterior ischemia indicates that paradoxical movement of the ventricular wall occurs not only during systole (outward movement during contraction) but also during early diastole (inward movement during the late isovolumic relaxation and early rapid filling phases) as well. Thus, during systole, some elastic potential energy can be stored in the dyskinetic segment and then released when left ventricular intracavitary pressure declines to low levels during the late isovolumic relaxation and early diastolic filling phases. Such inward diastolic movement of the dyskinetic segment can impart some motion to blood pooled in the apical and anterior regions of the ventricle and thus contribute to the alteration of the normal early diastolic intraventricular pressure gradient pattern. This interpretation would be consistent with the observation by Beppu et al that contrast echoes were prevented from entering the apex by a counterflow of blood proceeding from the dyskinetic apex toward the cardiac base just before mitral valve opening. This movement of blood from apex toward base implies that a pressure gradient exists such that pressure is higher in the apical region than in the basal region. Although such measurements are difficult because of the small magnitude of the pressure gradients involved, in at least one of the nine dogs in the present study, the pressure recorded by the apical transducer seems to have exceeded that of the proximal transducer.

Similarly, after coronary occlusion, EF declined and small regions of dyskinesia were present in dogs 3 and 4 (Table 2), yet no change in the MIVP was measured in the apical region of the LV. Such departure from linearity can be attributed to several potential causes. First, it is probable that dysfunction of some critical amount of myocardium is necessary to produce changes in the normal intraventricular pressure gradient pattern. Thus, after coronary occlusion, the relation might not be linear throughout the entire data range. This is consistent with the observation by Beppu et al that small-sized areas of asynergy did not produce abnormal intraventricular flow patterns. Second, it has been shown that during coronary occlusion, global left ventricular contraction can be maintained by augmentation of contraction in nonischemic regions of myocardium. Such regional hyperkinesia, presumably resulting from sympathetic adrenergic receptor stimulation of remaining functional areas of myocardium, is frequently sufficient to maintain normal global systolic function and normal cardiac output. It might also be sufficient to maintain diastolic function by augmenting global elastic recoil, thus helping to preserve relatively normal patterns of intraventricular diastolic pressure gradients and flow patterns. Third, it is possible that the position of the transducers within the LV might not have been optimal for the detection of changes in the early intraventricular pressure gradient pattern. The catheter position used was selected primarily because it could be most easily standardized for each dog. Because Beppu et al noted that, during diastole in dogs demonstrating apical akinesis, a pathway of blood was seen to move slowly around the apex, although in dogs exhibiting dyskinesis such flow was absent, placement of the transducers in closer proximity to the anterior or septal walls might have produced different recorded patterns of regional changes in the early intraventricular diastolic pressure gradient. In our study, however, selective positioning of the catheter was difficult to achieve and frequently resulted in pressure signal instability. We might expect a much improved linear relation between function and MIVP after coronary occlusion if we were able to tightly control the locus of myocardium around a defined region of pressure measurement, and then methodically vary the functional status of that myocardium from hyperkinetic to hypokinetic, to akinetic, to dyskinetic. Because, however, the present model has limited control over the extent of the severity of wall motion abnormality because of the nature of the method used to induce ischemia, and because we have limited knowledge of the locus of ischemic myocardium in relation to the region of pressure measurement because of the use of single-plane angiography, the lack of a high linear correlation after coronary occlusion should not be surprising. Although assessing ventricular function at baseline by viewing wall motion in a single slice might give reasonable information about function in nearby zones of myocardium, after coronary occlusion a single slice tells little about the positional relation of the zone of dysfunctional myocardium to the region of pressure measurement, and provides little or no information about the extent of dysfunction or hyperfunction in nearby zones of myocardium that might impact on the production of intraventricular pressure gradients. Thus, in relation to alteration of the intraventricular pressure gradients, the location of dysfunctional myocardium might be as important as the amount. Beyond these factors, a more comprehensive model relating left ventricular function to regional maximum intraventricular pressure gradient patterns will likely require consideration of complex interactions between regional wall motion and elastic recoil, geometric distortions, and the momentum of intraventricular flow during both systole and diastole.

The present observations confirm the existence of diastolic intraventricular pressure gradients in the LV, detectable with a dual sensor micromanometric
catheter, and demonstrate for the first time that this normal gradient pattern is related to left ventricular systolic function and that it can be attenuated, lost entirely, or even reversed in the presence of extensive acute myocardial ischemia involving the anterior and apical walls. We speculate that alterations in the early intraventricular diastolic pressure gradient pattern are related, in part, to a reduction in the amount of myocardium available for contraction and subsequent elastic recoil, and to changes that can occur in the pattern of flow during systole, especially in ventricles exhibiting extensive regions of dyskinesia. Regional alterations in the early diastolic intraventricular pressure gradient might be an important factor in the characteristic changes that occur in intraventricular flow in specific cardiac disorders.

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References


KEY WORDS • intraventricular diastolic pressure gradients • diastole • acute myocardial ischemia • ventricular suction
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