Localized areas of outward motion have been observed by our group to be a common finding in the left ventricles of patients undergoing cineangiography for the evaluation of chest pain. This phenomenon seemed most obvious in left ventricles which contracted normally. Initially we thought the timing of this motion was end-systolic and that it was a form of dyskinesia as described by others1-4 in damaged left ventricles, frequently associated with aneurysm formation. More careful slow motion frame-by-frame analysis of left ventricular cineangiograms, however, has shown this outward motion occurs after the cessation of systolic inward motion and before opening of the mitral valve, hence in the isovolumic relaxation period.

Recently, outward left ventricular wall motion during the isovolumic relaxation period has been described by others and has been thought to be an abnormal type of movement.5,6

The purposes of this report are to describe the type and locations of left ventricular wall motion during the isovolumic relaxation period in patients with and without obstructive coronary artery disease, to emphasize its frequent occurrence in normally contracting ventricles and to suggest that it represents a normal variation of left ventricular relaxation.

Methods

One hundred technically satisfactory left ventricular cineangiograms were analyzed from 100 patients (71 males and 29 females) who were being evaluated for chest pain. The patients were selected from a consecutively studied group and the only reason for excluding patients was finding a technically inadequate left ventricular cineangiogram. Two subgroups of patients were identified: one consisting of 59 patients with obstructive coronary artery disease and the other 41 patients with angiographically normal coronary
arteries. Obstructive coronary artery disease was considered to be present if at least one area of 50% stenosis was present in a coronary artery branch as visualized by selective coronary cineangiography. Patients with hypertensive, rheumatic, or primary myocardial disease were excluded. The angiograms included in this study were ones in which the left ventricular walls were clearly outlined by contrast material, the cycle analyzed was a sinus beat and did not follow a premature ventricular contraction (PVC), and opening of the mitral valve was clearly visible.

Left ventricular cineangiography was performed during retrograde left heart catheterization with the patient in the 30° right anterior oblique (RAO) position using 36 to 40 ml of 90% hypaque, injected during a 3-4 sec interval. Films were exposed at a rate of 60 frames per second with a 35 mm Arriflex cine camera mounted on a Siemens 10-inch image intensifier. The left ventricular injection was done just prior to selective coronary cineangiography. The patient's electrocardiogram and a cinemarker indicating each frame and the duration of the injection were recorded on high-speed photographic paper in an Electronics for Medicine DR-12 recorder.

Each ventriculogram was observed for the pattern of contraction when projected both rapidly and in a slow frame-by-frame sequence. The outlines of the ventricle at the onset of ejection, at end-systole, and at the end of the isovolumic relaxation period were traced from the projected images of the cinefilm, superimposed on the same paper. The onset of ejection was taken from the frame just prior to the onset of inward motion of the ventricular walls. End-systole was taken from the last frame which showed uniform inward motion of the left ventricular walls. The end of the isovolumic relaxation period was taken from the frame just before nonopacified blood was seen crossing the mitral valve. The isovolumic relaxation period was measured by counting the frames from maximal inward left ventricular wall movement to the opening of the mitral valve and multiplying by 16.67 msec.

The selection of frames was made following a review of the cineangiograms by all three authors. In order to test the agreement between different observers in selecting the appropriate frames, two of the authors subsequently reviewed in a blinded fashion 25 cardiac cycles in 13 consecutively studied patients from the group of 81 patients with SERP. The average differences in their selections was less than one frame for each category, specifically for onset of ejection, 0.96 frame, for end-systole, 0.80 frame, and for end of the isovolumic relaxation period, 0.4 frame. No attempt was made to superimpose aortic valve outlines. Since the angiographic table could be moved during filming, the spine or a rib margin was drawn on each frame that was traced to assure that the patient had not moved. Such movement rarely occurred. This method of superimposing ventricular outlines does not take into account rotation or displacement of the left ventricle which may occur during the cardiac cycle, but in our group of normally contracting ventricles the method confirmed uniform inward motion of the walls during systole.

Ventricular volumes were calculated by the area-length method.\(^7\)\(^-\)\(^9\) At end-diastole and at end-systole the length of the long axis of the left ventricle was measured directly from the middle of the aortic valve to the apex and the area of the ventricular silhouette was planimetered. This data, along with a correction factor for magnification and distortion and the patient's body surface area, was placed into a Hewlett-Packard model 9100 A computer for automated computations of the left ventricular volumes. The ejection fraction was calculated by dividing stroke volume by the end-diastolic volume. A correction factor was obtained by counting and planimetering one centimeter squares in a grid which was filmed at the level of the patient's left ventricle using the same fluoroscopic height that was used during the angiogram. The correction factor is a ratio of the counted area to the planimetered area.

Left ventricular pressures were measured using fluid-filled catheters and Statham strain gauge transducers (P23-Db series). End-diastolic pressure was read from photographic recordings of high-amplification pressure tracings. The first derivative of left ventricular pressure (dp/dt)\(^\text{max}\) was obtained from a precalibrated R-C differentiating circuit and maximum dp/dt was measured.

Systolic time intervals were measured in each patient on the day prior to the angiographic procedure and the projection period was used to left ventricular ejection time (PEP/LVET) ratio was calculated by the method described by Weissler.\(^10\)

**Results**

The isovolumic relaxation period measured by the angiographic method averaged 100 ± 23 msec and correlated well with values obtained by other investigators\(^11\)\(^,\)\(^12\) who have used the graphic method of measuring the time from the aortic component of the second heart sound to the 0 point of the apexcardiogram (table 1). In five patients the isovolumic relaxation period was measured on separate occasions by both angiographic and graphic methods and the average duration was within 11 msec.

In 83 of the 100 patients outward left ventricular wall motion during the isovolumic relaxation period was observed. This motion was apparent on rapid speed projection of the cineangiograms but was best seen and timed with slow motion frame-by-frame analysis. In 81 of the 83 patients the outward

<table>
<thead>
<tr>
<th>Table 1</th>
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<tr>
<td><strong>Comparison of Angiographic and Graphic Methods of Measuring Isovolumic Relaxation Period</strong></td>
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<tr>
<td>----------------------------------------</td>
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<tr>
<td><strong>Duration of Isovolumic Relaxation Period</strong></td>
</tr>
<tr>
<td>Angiographic method</td>
</tr>
<tr>
<td>Graphic method</td>
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<td>Benchimol</td>
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[Circulation, Volume XLVIII, September 1973]
wall motion was localized, hence termed segmental early relaxation phenomenon or SERP for convenience of description. The outward motion was diffuse in two patients, involving all of the ventricular silhouette that could be seen in the RAO projection. A simultaneous lesser inward left ventricular wall motion during the isovolumic relaxation period was seen in the inferobasal wall or apex in 29 patients. In 25 consecutively studied patients with SERP, mitral valve motion during the isovolumic relaxation period was closely observed. In 14 patients the mitral valve complex could be seen descending into the ventricle prior to opening of the valve. Ventricular volumes were calculated at the end of the isovolumic relaxation period in 39 patients. In this group there was an average increase from the end-systolic volume of 6.1 ml ($P < 0.001$).

Of the 100 patients studied 48 had normal left ventricular contractile motion while 52 showed varying degrees of left ventricular contraction abnormalities. SERP was observed in 44 of the 48 patients with normal left ventricular contractile motion. Table 2 shows the sites of outward motion in these patients. The most frequent sites were in the anterolateral wall (fig. 1), and in the anteroapical area (fig. 2). Less common sites were in the

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**Table 2**

<table>
<thead>
<tr>
<th>Areas of SERP</th>
<th></th>
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</thead>
<tbody>
<tr>
<td>Anterolateral</td>
<td>18</td>
</tr>
<tr>
<td>Antero-apical</td>
<td>11</td>
</tr>
<tr>
<td>Apical</td>
<td>6</td>
</tr>
<tr>
<td>Anterolateral and inferior</td>
<td>5</td>
</tr>
<tr>
<td>Infero-apical</td>
<td>2</td>
</tr>
<tr>
<td>Inferior</td>
<td>1</td>
</tr>
<tr>
<td>Extensive</td>
<td>1</td>
</tr>
<tr>
<td>No SERP</td>
<td>4</td>
</tr>
</tbody>
</table>

$N = 48$

---

**Figure 1**

Anterolateral SERP (segmental early relaxation phenomenon). The dashed line indicates the ventricular silhouette at end-systole. Prior to mitral valve opening the midportion of the anterolateral wall has moved out while a more apical segment has moved in and the mitral valve has descended. A basal segment of the inferior wall is hypokinetic.
apex (fig. 3) and anterolateral with inferior wall.

Of the 52 patients with left ventricular contraction abnormalities SERP occurred in 39. Table 3 shows the locations of SERP in these patients. When SERP was observed in ventricles with contractile abnormalities (hypokinetic or akinetic areas), it usually occurred in normally contracting areas (25 of 39 patients, table 3). SERP occurred infrequently (4 of 16 patients) in the presence of extensive contraction abnormalities. An example of an extensive contraction abnormality without SERP is depicted in figure 4.

Of the 59 patients with obstructive coronary artery disease 42 had left ventricular contractile abnormalities. SERP was seen in 30 of the 52 patients and was absent in 12. To illustrate the rarity of SERP in patients with marked left ventricular dysfunction, figure 5 shows the hemo-

**Table 3**

*Sites of Outward Wall Motion in Ventrices with Local Contraction Abnormalities (LCA)*

<table>
<thead>
<tr>
<th>Areas of SERP</th>
<th>Anterolateral</th>
<th>Apex</th>
<th>Areas of LCA</th>
<th>Inferior</th>
<th>Extensive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterolateral</td>
<td>5</td>
<td>3*</td>
<td>6*</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Inferior</td>
<td>1*</td>
<td></td>
<td></td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>Antero-apical</td>
<td>1</td>
<td>3</td>
<td>8*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterolateral and inferior</td>
<td>1</td>
<td>1*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infero-apical</td>
<td>1*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apical</td>
<td>3*</td>
<td></td>
<td>1*</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Extensive</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>12</td>
</tr>
<tr>
<td>No SERP</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

N = 52

The asterisks indicate the occurrence of SERP in normally contracting areas (25 of 39 patients with SERP).

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Figure 3

Apical SERP. The dashed line indicates end-systole. As in fig. 1, the mitral valve has descended.

dynamic data of the 42 patients with obstructive coronary artery disease and contraction abnormalities. The 12 patients without SERP are the ones described as no change. Those with SERP had a significantly higher ejection fraction (EF), 0.55 ($P < 0.001$), and maximum dp/dt, 2006 mm Hg/sec ($P < 0.025$), with lower values for EDP, 9 mm Hg ($P < 0.001$), EDV, 97 ml/m² ($P < 0.001$), and PEP/LVET, 0.39 ($P < 0.001$) than those without SERP (EF = 0.34, dp/dt = 1478 mm Hg/sec, EDP = 16 mm Hg, EDV = 147 cc/m² and PEP/LVET = 0.50).

Discussion

Outward left ventricular wall motion during the isovolumic relaxation period is a very frequent angiographic finding in patients who are being evaluated for chest pain, whether or not they have obstructive coronary artery disease. Despite this frequency it has been described in humans only quite recently. Gooch et al. described outward motion in the anterior wall during this period in patients with prolapse of the mitral valve. They interpreted this motion as an early relaxation but thought it was present only in patients with mitral valve prolapse. We also have observed this motion.
in such patients as well as in patients with chronic rheumatic valvular disease. Thus it appears to be a more universal phenomenon than was originally thought. Our findings in regard to location of SERP are similar to those of Ruttley et al., in that it is usually though not always found in the apical half of ventricles which have normal contractile motion, while in ventricles with contraction abnormalities it is usually seen in normally contracting areas.

It is not clear why left ventricular wall motion during the isovolumic relaxation period has not been reported in the past from the many laboratories engaged in cineangiographic studies. A possible reason for its being overlooked is that it may have been mistimed. This could readily happen since the basal part of the inferior wall may continue to move in right up to the time of mitral valve opening, giving the impression that systole has not ended. If one inspects the anterolateral wall and apex, however, inward motion has ceased well before mitral valve opening, in our patients by an average of 100 msec.

If SERP were thought to be an end-systolic event, it might be confused with dyskinesis or paradoxical systolic expansion of a portion of the left ventricular wall. In ventricles with normal or slightly abnormal contractile motion, these two entities really should not be mistaken for one another since dyskinesis has been described mainly in ventricles with markedly impaired contraction.1-3 It is in such ventricles that it is most difficult to determine the timing of end-systole since there is so little apparent contractile motion. In extensively damaged ventricles the fact that dyskinetic areas are confined mainly to areas of marked hypokinesis or of actual aneurysm formation whereas SERP is seen in normally contracting segments should aid in differentiating the two entities.

If it is granted that there is segmental outward left ventricular wall motion during the isovolumic relaxation period, what is its significance? A catheter across the aortic valve may induce slight regurgitation, increasing ventricular volume as postulated by Ruttley et al., but this is an unlikely explanation since it does not account for the segmental patterns of outward motion.

The concept of motion without volume change due to early relaxation was advanced by Bove13 to explain outward apical motion during the isovolumic relaxation period he and Hawthorne14 observed in the canine left ventricle.

If the isovolumic relaxation period is truly isovolumic, outward left ventricular wall motion must be countered by an inward motion which we have frequently observed in the inferobasal wall or in the apex. It is of interest that a small but significant increase in calculated ventricular volume occurred in our patients during this interval. Whether an actual volume change occurred during this supposedly isovolumic period is open to question since we have been limited to inspection of the left ventricle in just one plane. It may well be that the inward inferobasal motion during isovolumic relaxation is related to descent of the mitral valve which was clearly seen in some of our patients, but the relationship of mitral valve motion to outward motion in other parts of the ventricle remains unclear.

Left ventricular wall motion has recently been demonstrated in man during the isovolumic contraction period and SERP could be an analogous phenomenon occurring in the isovolumic relaxation period. While Karliner, Bouchard, and Gault15 postulated that posterior displacement of the mitral valve could account for an apparent volume loss during isovolumic contraction, they thought fiber shortening was taking place during this interval. Since all segments of the left ventricle apparently do not contract simultaneously, it seems reasonable to assume that relaxation is also not always symmetrical, but may occur segmentally, thus justifying the use of the term, segmental early relaxation phenomenon.
While other groups that have recently reported outward motion during the isovolumic relaxation period have considered this finding an abnormality, we propose that such motion is most likely a normal variation of left ventricular relaxation. This postulate is supported by its frequent presence in normally contracting ventricles, by its infrequent occurrence in abnormally contracting areas, and by the rarity of its occurrence in extensively damaged ventricles.

Acknowledgments

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