Abnormal Left Ventricular Wall Motion at Mid-ejection in Patients with Coronary Heart Disease

By Richard F. Leighton, M.D., Mary Ellen M. Pollack, M.D., and Thomas G. Welch, M.D.

SUMMARY
The degree of inward motion at mid-ejection was examined for seven segments on the silhouettes of left ventriculograms taken in the 30° RAO projection in patients with normal coronary arteries. The pattern of wall motion described in these patients was used to distinguish abnormalities in mid-systolic wall motion. One or more abnormally contracting segments were found at mid-ejection in 27 of 42 patients with obstructive coronary artery disease and normal end-systolic wall motion. Of the 57 segments found in these patients, 41 or 72% corresponded to sites of significant coronary artery obstruction. Seven patients had electrocardiographic evidence of prior infarction. Following coronary graft surgery in eight patients improved motion was found in association with graft patency in seven previously delayed segments and two new areas of delayed wall motion associated with nonpatent grafts and electrocardiographic changes of infarction appeared. We postulate that some of the myocardial fibers in late contracting segments have been injured or infarcted and are able to contract effectively only during the latter half of ejection when ventricular wall tension is reduced.

Within the past decade the more widespread use of left ventricular cineangiography in the study of patients with coronary heart disease has led to descriptions of regional abnormalities of contractile motion. Such abnormalities have been found commonly1-4 and it has been shown that their locations frequently correspond to sites of coronary artery obstruction.1,9 In our experience and in the experience of others,2 however, a normal contraction pattern may be found in the presence of significant obstructive coronary artery disease when only the onset and the end of the ejection phase of systole are analyzed in the left ventricular cineangiogram. We have observed that many patients with these angiographic findings, that is, obstructive coronary artery disease and apparently normal patterns of left ventricular contraction, have localized abnormalities of contraction which occur only during the first half of ejection. Thus, when the motion at mid-ejection is examined, delayed, paradoxical, or no motion may be found. The investigation of these contraction abnormalities is the substance of our report.

Method
A retrospective and prospective analysis was made of selective coronary and left ventricular cineangiograms performed in our laboratory and two groups of patients were selected. One group was composed of 20 patients who had chest pain with angiographically normal coronary arteries. Normal values for parameters of left ventricular function were defined as being within the group range for these patients: ejection fraction > 0.59, end-diastolic volume < 95 ml/m², and end-diastolic pressure < 15 mm Hg. Normal values for percent shortening of seven endocardial segments on the 30° RAO left ventriculogram from the onset of ejection to end-systole were defined from the findings in these 20 patients, as being no greater than two standard deviations from the group mean.6

The second group was composed of 42 patients with coronary artery disease with 70% or greater luminal obstruction in at least one major coronary artery branch in the presence of normal left ventricular end-diastolic volume and pressure, a normal ejection fraction, and a normal end-systolic contraction pattern as defined above. All of the patients with coronary artery disease had chronic stable angina pectoris and no evidence of congestive heart failure. Eight patients who underwent aortocoronary saphenous vein graft surgery were selected from the group on the basis of normal postoperative values for left ventricular ejection fraction, end-diastolic volume, and end-systolic wall motion. Their postoperative left ventriculograms were obtained an average of 61 days following graft surgery (range 12 to 142 days).

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Single plane left ventricular angiography was performed in the 30° RAO position using 36 to 42 ml of 90% hypaque injected during a 3–4 sec interval while the patient maintained full inspiration. Films were exposed at a speed of 60 frames per second in a 35 mm Arriflex camera mounted on a Siemens 10-inch image intensifier. Left ventricular volumes and ejection fraction were determined by the area-length method as previously described. 7, 10 Technically poor angiograms, as well as those with obvious contraction abnormalities, were excluded. Premature beats and post-premature beats were not analyzed. The recognition of premature beats was aided by recording the patient’s electrocardiogram and a cinemarker indicating each frame on high-speed photographic paper in an Electronics for Medicine DR-12 recorder. Following the ventriculogram, selective coronary angiography was performed.

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 ventricles at the onset of ejection, at mid-ejection, and at end-systole were traced from projected frames superimposed on the same paper. A longitudinal axis and eight hemiaxes were drawn. The methods for superimposing silhouettes, for selecting frames corresponding to the onset of ejection and to end-systole, and for drawing longitudinal axes and hemiaxes have been respectively described in detail. 9 Mid-ejection was taken from the frame equidistant between the onset of ejection and end-systole. The ventricular silhouette at mid-ejection was realigned and retraced in the same manner as the end-systolic silhouette, to correct for upward rotation of the apex during ejection.

The superimposed silhouettes at onset of ejection, mid-ejection, and end-systole, and the axes are shown in figure 1. The points of intersection of hemiaxes with endocardial silhouette were numbered 1–4 on the anterolateral wall and 5–8 on the inferior wall. Since it was previously found that the degree of shortening to end-systole for segment 8 and for the apex showed too much variation to permit definition of normal values, 6 these segments were excluded from the statistical analysis at mid-ejection. The amount of shortening along each hemiaxis for segments 1–7 was measured and the distance from onset of ejection to mid-ejection was expressed as a percentage of the total distance from onset of ejection to end-systole. In the 20 normal patients the tracings and measurements were made independently by two observers.

Measurements of the percent shortening from onset to mid-ejection of each of the seven endocardial segments were then made in the patients with coronary heart disease and the values were compared to the corresponding values from the normal patients. A local area of delayed contraction was defined as an area where the percent shortening at mid-ejection was less than two standard deviations from the mean percent shortening obtained in the normal group. In addition lack of motion and paradoxical motion in segments 1–7 as well as in the apex were considered abnormal since they were not found in the normal patients. In separating areas of abnormal wall motion at mid-ejection to correspond to locations of coronary artery lesions, the apex was included with segments 1–4 in the distribution of left anterior branches, while segments 5–7 were considered to be in the distribution of either left posterior or right coronary branches. In three patients, one from the normal group and two with coronary disease, wall motion measurements were made from every two or three cineangiographic frames throughout ejection.

Systolic time intervals were determined in the patients with coronary disease from rapid speed simultaneous recordings of a carotid pulse tracing, phonocardiogram, and electrocardiogram, and the PEP/LVET ratio was calculated.

Results

Values obtained by both observers for the normal mean percent shortening of the seven segments at mid-ejection with two standard deviations from the means are shown in figure 2. No significant differences were found between the two observers’ measurements either when the average percent shortening for each

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**Figure 1**

Superimposed tracings of left ventricular silhouettes from a patient with normal coronary arteries, showing placement of axes and locations of segments used to define extent of motion both at end-systole and at mid-ejection.

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**Figure 2**

Percent shortening at mid-ejection for normal patients. Values from the 20 patients with normal coronary arteries for mean extent of left ventricular wall motion at mid-ejection with two standard deviations ±SD from the mean. The measurements were made by two observers; the data of one indicated by the solid lines, the other by the dashed lines. For patients with coronary artery disease, values less than two ±SD defined abnormal motion in each segment.
segment or the 140 individual measurements were compared as paired samples. Since the two observers' values for the lower limits of normal wall motion (average percent minus two standard deviations) differed, the lower of the two values was used to define abnormal motion in each segment.

Using these criteria, one or more abnormally contracting segments at mid-section were found in 27 of 42, or 64%, of the patients with coronary heart disease (table 1). In the other 15 patients wall motion in all seven segments at mid-ejection was within two standard deviations of the normal mean and some inward motion was observed at the apex. Of a total of 57 abnormally contracting segments, 41, or 72%, corresponded to sites of significant coronary artery obstruction.

Sixteen of the abnormal segments exhibited paradoxical motion (fig. 3) and six had not moved at all at mid-ejection. All of the segments which moved paradoxically at mid-ejection were associated with 70% or greater coronary stenoses. In each instance when the apex was the site of paradoxical motion the appropriate coronary lesion was located proximally — either in the left anterior trunk or at the origin of the anterior descending artery.

Abnormally contracting segments at mid-ejection were more commonly found in the inferior wall (34 of 57 segments, see fig. 4) than in the anterolateral wall or apex (23 segments), although an almost equal number of patients had such segments in the anterior wall or apex alone (11 segments) compared to the inferior wall alone (12 segments). Four patients had abnormal segments in both walls.

Of the 16 abnormal segments which did not correspond to sites of 70% or greater coronary artery obstruction, seven were associated with a coronary artery obstruction of less than 70%. There was no angiographic evidence of coronary artery disease in

Table 1

<table>
<thead>
<tr>
<th>Wall segments</th>
<th>Anterolateral-Apex</th>
<th>Inferior</th>
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<tbody>
<tr>
<td></td>
<td>1 2 3 4</td>
<td>5 6 7</td>
</tr>
<tr>
<td>LA, R</td>
<td>25 22 15 9</td>
<td>19 24 19</td>
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*Values given are percents of shortening at mid-ejection.

Abbreviations: SME = shortening at mid-ejection; LA = left anterior; LP = left posterior; R = right; P = paradoxical motion; sd = standard deviation.

Figure 3

Left ventricular silhouettes from a patient with paradoxical motion at the apex and at segments 5 and 6 with no motion at segment 7 by mid-ejection. By end-systole normal wall motion is evident. This patient had complete obstruction of the left anterior trunk and 95% stenosis of the proximal right coronary artery with ECG evidence of an old anteroseptal infarct.

Figure 4

Superimposed left ventricular tracings from a patient with delayed inferior wall motion involving segments 5, 6 and 7. This patient had a 70% stenosis of the proximal left posterolateral coronary branch and complete obstruction of the LAD branch with a normal ECG.
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the appropriate branches for the other nine segments.

Historical, enzymatic, and electrocardiographic evidence for myocardial infarction was reviewed in these patients. A history of one or more episodes of prolonged substernal chest pain was a common finding both in patients with abnormal wall motion at mid-ejection and in those with normal ventricular contractile patterns (7 of 15). While only one of the patients with normally contracting ventricles had electrocardiographic Q waves consistent with a previous infarction, seven patients with abnormal wall motion at mid-ejection had such Q waves and two of these seven had documented rises in serum enzymes at the times of their apparent infarctions. A completely normal electrocardiogram was unusual in either group.

Among patients who had complete coronary branch occlusions, appropriate segments of abnormal wall motion at mid-ejection were present no more frequently than were normally contracting areas (9 vs 13 times). Abnormally contracting areas at mid-ejection were no more frequently sites of collateral formation than were normally contracting areas (10 vs 12 times).

The absence of an apical S2 sound was examined and found not to be useful in separating patients with abnormal wall motion at mid-ejection (16 of 27, or 59%) from those with normally contracting ventricles (9 of 15, or 60%). Likewise measurements of the PEP/LVET ratio, available in all 27 of the patients with abnormal wall motion at mid-ejection and in 14 of the 15 patients with normally contracting ventricles, were not significantly different between the two groups (average 0.37 vs 0.36).

Inward wall motion throughout ejection appeared to progress at a fairly uniform rate in the one normal patient (fig. 5). By contrast in one patient with a single late-contracting inferior wall segment and in another with paradoxical motion of anterior wall segments, inward motion of these segments during the second half of ejection appeared to be accelerated.

Of the eight patients who had analysis of wall motion at mid-ejection both before and after saphenous vein graft surgery, five had abnormal wall motion at mid-ejection and three had normal contractile motion in the preoperative studies. Their electrocardiographic and left ventricular wall motion findings are summarized in Table 2. In the first three patients' grafts the effects of graft patency on seven previously abnormal segments could be observed. Following surgery all seven segments showed normal or improved motion (fig. 6). Concomitantly three previously normal segments located in the distribution of patent grafts showed delayed contraction following surgery.

In three patients with nonpatent grafts the effects of nonpatency on previously normal segments were observed. In two segments abnormal wall motion at mid-ejection was evident postoperatively, associated in each patient with electrocardiographic evidence of

Figure 5

Left ventricular wall motion throughout ejection in three patients. The graphs are placed appropriately to represent segments around a left ventricular silhouette. On the vertical axes are percents of wall motion and on the horizontal axes percents of time throughout ejection. The lower limits of normal motion at mid-ejection are shown by the short horizontal lines which appear above the 50% time marks. The closed circles indicate values for one normal patient, the open circles for a patient with coronary disease and delayed motion in segment 6, the closed triangles for a patient with coronary disease and paradoxical motion during the first half of ejection in segments 1, 2, 3, and 4. Accelerated motion during the latter half of ejection is evident in the appropriate segments.

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new infarction. Four additional segments underwent changes in contractile motion at mid-ejection, unrelated to graft patency or nonpatency.

Discussion

Abnormalities in contraction of portions of the left ventricle at mid-ejection may well prove to be a common finding in patients with significant coronary artery disease. This phenomenon was observed in approximately 70% of our patients, all of whom had normal wall motion at end-systole, as well as normal ejection fractions, end-diastolic volumes, and pressures. Contraction abnormalities occurring during the early phases of ejection have been noted by other investigators. Sniderman and his colleagues have described akinesis and dyskinesis in the first half of systole with an exaggerated shortening in late systole in patients with coronary artery disease. Furthermore, Johnson and her colleagues have recently found diminished left ventricular volume change in the first

Table 2

<table>
<thead>
<tr>
<th>Patients</th>
<th>Graft sites</th>
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<th>LV Segments</th>
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<td></td>
<td>A NSSTTA</td>
<td>7</td>
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<tr>
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<td>B Anteroseptal infarct</td>
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<td>A Unchanged</td>
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<td>LA – NP</td>
<td>B NSSTTA</td>
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<td>A Anterolateral infarct</td>
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<td>LA – NP</td>
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<td>A NSSTTA</td>
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Abbreviations: Pat = patent; NP = nonpatent; B = before surgery; A = after surgery; SEI = subendocardial injury; NSSTTA = non-specific ST-T wave changes; N = normal; LV = left ventricular; P = paradoxical motion.

![Figure 6](image)

*Left ventricular tracings before (on the left) and after (on the right) insertion of a patent saphenous vein graft into the LAD coronary artery in a patient with delayed motion in the anterolateral wall. Following surgery wall motion at mid-ejection has become normal in segments 2 and 3 and has improved in segment 4. Postoperatively there is also a more extensive contraction of the apex in the latter half of systole and a smaller end-diastolic volume.*
third of ejection with an accelerated volume change in the middle third of ejection in patients with coronary artery disease. It also seems likely that one or more areas of delayed contraction may have been present in patients previously described as having asynchronous ventricular contraction.1, 8

It may seem surprising that this common disorder of left ventricular contraction has apparently not achieved wider recognition in cineangiographic laboratories. Once the phenomenon has been recognized, it has been our experience that abnormal wall motion during the early phase of ejection may be obvious when rapidly projected cineangiographic frames are viewed. Frequently, however, the presence of late contracting segments may not be obvious at the time of projection of cineangiograms and in such cases a large part of the failure to recognize this contraction abnormality may be related to the method used to analyze ventricular wall motion. Obviously delayed contraction will not be appreciated if left ventricular silhouettes are traced only at the beginning and end of ejection. Furthermore, it is important to exclude the presence of hypokinetic segments. While hypokinetic segments may also contract late, we have not attempted to define delayed contraction in such segments since its demonstration would be technically hindered by the small amplitude of total inward motion. A method of wall motion analysis, to be useful in the detection and exclusion of hypokinetic segments, should include correction for potential sources of artifactual motion including thoracic cage and diaphragmatic motion, descent of the aortic valve and rotation of the apex, all of which may occur throughout the ejection phase of systole. Thus realignment of traced silhouettes is required both at end-systole and at mid-ejection. The presence and location of late contracting segments may be obvious only after realignment of the traced left ventricular silhouettes.

In the detection of delayed contraction it is important to begin counting frames from the onset of uniform inward ventricular wall motion rather than from end-diastole, in order to exclude the isovolumic contraction period. If this period of relative lack of wall motion is included, a normal contraction sequence might be interpreted as late contraction.

Using this quantitative method,4 we were able to define normal end-systolic wall motion in a given segment as being within two standard deviations of the mean for a group of patients with angiographically normal coronary arteries and left ventricles. In the same patients we similarly defined normal values at mid-ejection. The extent of inward motion by mid-ejection in each area was not equal. For this reason motion in each segment on the ventricular silhouette was analyzed separately.

Paradoxical motion or lack of motion at mid-ejection seemed to us to constitute obvious abnormalities which did not require statistical definition since they were not observed in the normal patients. The analysis of wall motion in multiple cineangiographic frames throughout ejection would provide a more precise and graphic display of abnormalities in the sequence of contraction. Such an analysis, however, is extremely tedious and probably can be practically accomplished only by use of a computerized technique.

The phenomenon of paradoxical motion at mid-ejection would appear to be a more profound contraction abnormality than delayed contraction. In our patients it was always associated with a significant coronary lesion. Nevertheless, paradoxical motion was noted to revert to normal motion following successful graft surgery. The significance of the high incidence in our patients of such paradoxical motion at the apex is uncertain. It may be that the apex is vulnerable to more serious injury because of its distance from severe proximal lesions in the anterior coronary circulation.

The precise mechanisms for late contraction remain uncertain. Some of our findings suggest that these segments may be the sites of previous myocardial infarction. These findings include the high degree of correlation (72% of cases) with sites of significant coronary artery obstruction, the presence of electrocardiographic findings of infarction in seven of the 27 patients, and the appearance of new areas of abnormal motion at mid-ejection in two patients with nonpatent grafts who developed electrocardiographic evidence of fresh infarction in the postoperative period. The finding that some segments exhibited abnormal wall motion unrelated to significant coronary obstruction need not negate infarction as an etiology since the syndrome of myocardial infarction with angiographically normal coronary arteries is now well known.13 Furthermore, inappropriate responses of segmental wall motion to graft surgery might be explained by intraoperative injury.

The reversibility of abnormalities in the sequence of wall motion associated with successful graft surgery suggests that not all of the fibers in late contracting segments need be infarcted. Since peak wall tension during ejection has been shown to be greatest during the early phases,14 it may be that partially infarcted or injured fibers are able to contract effectively only during the late phases of ejection when ventricular wall tension is reduced. The restoration of effective blood flow to these segments might permit recovery of the normal contractile sequence by enough fibers to present an over-all appearance of normal contraction.

Another factor to be considered in improved wall motion noted in the first few months after graft sur-

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gery is a greater endogenous catecholamine effect which, however, should be expected to exert a uniform effect on the motion of ventricular segments.

Segmental delays in left ventricular contraction may well be found in other forms of heart disease. We have looked for their presence only in patients with significant coronary artery disease, and otherwise apparently normal left ventricular function. In these patients these abnormalities of wall motion at mid-ejection, which we have collectively termed tardokinesis, appear to constitute distinct abnormalities of ventricular contraction and their recognition provides an added means of assessing left ventricular function.

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