Ventricular Apical Vents and Postoperative Focal Contraction Abnormalities in Patients Undergoing Coronary Artery Bypass Surgery

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SUMMARY Ventriculograms made 9–15 months after surgery in 48 patients with normal preoperative apical contraction were reviewed to determine the influence of apical venting on apical wall motion in patients undergoing coronary bypass surgery. After interpretation of postoperative apical wall motion, the patients were subdivided into two groups. One group consisted of 34 patients who were vented by inserting a catheter through the apex of the left ventricle and the second group included 14 patients in whom no transventricular vent was made. The two groups were similar clinically and hemodynamically before surgery, and the surgical procedures were similar with the exception of vent site. Following surgery, incidences of graft patency and antegrade flow to the apex were also similar. Nineteen (56%) patients in the apically vented group had apical dyskinesia or akinesia observed on the postoperative ventriculogram while none of the patients who were not apically vented had these findings. None of the patients with apical dyskinesia or akinesia had congestive heart failure following surgery. The postoperative ventriculograms of 12 patients with mitral stenosis who underwent valvulotomy by inserting a Tubbs dilator through the apex were also analyzed. Only one patient (8.5%) had apical dyskinesia or akinesia. Since the patients with mitral stenosis probably did not have significant coronary artery disease, it is possible that the combination of the apical vent and ischemic heart disease was responsible for the focal contraction abnormalities observed.

FOCAL AREAS OF ASYNERGY occur in patients with coronary artery disease and are thought to represent areas of myocardial ischemia or infarction. A recent study of postoperative angiograms in patients who had undergone coronary artery bypass surgery identified a high incidence of apical asynergy both in patients who had ECG or enzyme evidence of perioperative infarction and in patients with no evidence of infarction. The etiology of the focal asynergy was uncertain since in nearly all of the patients a ventricular vent was inserted at the apex to decompress the left ventricle during cardiopulmonary bypass. Previous reports have suggested that apical asynergy might result from apical venting; however, this has not been documented by postoperative ventriculograms.

The purpose of the present study was to determine the influence of apical venting on apical wall motion in patients who underwent coronary bypass surgery. This was accomplished by comparison of pre- and postoperative ventriculograms in patients who were vented through the apex and in patients who did not have transventricular vents. An additional group of patients with mitral stenosis who had transventricular mitral valve dilation using a Tubbs dilator are included to compare the incidence of wall motion abnormalities in patients thought not to have coronary artery disease.

Methods

Patients with Coronary Artery Disease

The following criteria were used to select patients for the present study: 1) coronary arterial bypass grafting during the period from January 1, 1972 through June 30, 1974 without concurrent ventricular or valvular surgery; 2) normal apical wall motion before surgery documented by preoperative catheterization within two months of surgery 3) elective postoperative catheterization 9–15 months following surgery regardless of presence or absence of symptoms (informed consent was obtained from all patients); 4) both pre- and postoperative left ventricular cineangiograms adequate for interpretation of apical wall movement. Among 329 patients who underwent coronary arterial bypass surgery during the designated period, 48 (15%) met all of the above criteria. Thirty-four of the 48 patients were apically vented, and the remaining 14 patients did not receive apical vents.

All 48 patients were evaluated before and after surgery with complete left and right heart catheterizations in the Cardiovascular Laboratory of the Duke University Medical Center. Each evaluation included hemodynamic measurements, left ventricular cineangiography, and selective visualization of both coronary arteries and bypass grafts. Although the current study is a retrospective analysis, all the clinical, hemodynamic, and angiographic data were collected prospectively and stored in the data bank as described previously.

The patterns of ventricular contraction were interpreted by two observers without knowledge of the type of venting procedure employed. Apical contraction patterns were analyzed by comparing the drawings of the end-systolic and end-diastolic silhouettes from the pre- and postoperative left ventricular cineangiograms in the right anterior oblique position. The findings were also confirmed by viewing the cine in motion. Abnormal contraction patterns were classified as hypokinesia, akinesia, or dyskinesia as defined by Herman et al. The two observers had no disagreement in stating whether there was a distinct change from normal to dyskinesia or akinesia between the pre- and postoperative ventriculograms. Nor was there any disagreement regarding absence of distinct change, i.e., normal to normal or nor-
mal to hypokinesia. The determination of pattern of blood flow in the apex (antegrade, retrograde, or absent) was obtained by consensus of four senior coronary angiographers in conference.

Twelve-lead ECGs of all patients during preoperative, perioperative, and follow-up periods (until time of restudy) were reviewed. The evidence for myocardial infarction was determined by QRS changes. The findings of patients with and without apical vents were then compared statistically using the Student's t-test for continuous variables and chi-square method for discrete parameters.

The operative procedure involved aortocoronary saphenous vein bypass grafts in 46 patients and internal mammary grafts in two patients. In 34 patients the left ventricle was vented by inserting a 7 mm plastic drainage catheter through a small apical stab wound (fig. 1). The vent was closed by a purse string suture and buttressed with a teflon pledget. Eight of the 14 "nonapically vented" patients had no vent at all. They had right coronary and/or LAD bypass, and the surgeon determined that the procedure would not require ventricular decompression. All patients in whom the left circumflex was bypassed were vented. Five of the 14 nonapically vented patients were vented through the right superior pulmonary vein by a method previously described, and one was vented through the left atrium. The operative procedure was otherwise identical in patients with and without apical vents. The retrospective nature of the study does not permit identification of the basis for the choice of vent site. Extracorporeal circulation was utilized in all patients who were apically vented, and in 13 of the 14 nonapically vented patients. A bubble oxygenator, mild hypothermia (30°–32°C), moderate hemodilution, and induced fibrillation were used during cardiopulmonary bypass.

Patients with Mitral Stenosis

The postoperative left ventricular cineangiograms of 12 patients who met the following criteria were also reviewed:

1) patients had predominant mitral stenosis with no other significant congenital or valvular defect; 2) closed mitral valvulotomy with a 9 mm Tubbs dilator; and 3) postoperative cardiac catheterization with left ventriculograms up to 15 months following surgery because of progression of symptoms related to mitral stenosis.

The method of evaluating apical wall motion in the postoperative ventriculograms in these patients was similar to that described above. Only two of these 12 patients had selective coronary angiograms, and these were reviewed.

The technique employed in mitral commissurotomy involved placement of purse string sutures around the left atrial appendage and in the apex of the left ventricle. After introducing a finger into the left atrium and palpating the mitral leaflets, a small nick was made at the apex of the left ventricle and the Tubbs dilator was introduced. The valve was dilated, the dilator was removed, and the purse string suture at the left ventricular apex was tied. Cardiopulmonary bypass was not used.

Results

The coronary artery disease patients with and without apical vents were quite similar in pertinent characteristics during the preoperative, operative, and postoperative periods (table 1). There was no significant difference in age, NYHA classification for severity of angina, incidence of myocardial infarction by electrocardiogram, cardiac index, or systemic arterio-venous oxygen difference prior to surgery. None of the apically vented patients and only one of the control patients had a history of congestive heart failure before surgery.

At surgery, patients in the two groups received similar numbers of bypass grafts with a similar proportion of each group having grafts to the left anterior descending coronary artery. There was no significant difference in incidence of documented (ECG) myocardial infarction during operative and perioperative periods.

One patient in the apically vented group and one in the group who were not vented at the apex developed signs of congestive heart failure by the time of follow-up evaluation. Both of these patients had normally contracting apical segments at the time of postoperative evaluation. There was no significant difference between these two groups in ECG evidence of myocardial infarction during the 9–15 months following surgery. The incidences of both total and left anterior descending graft patency were also similar.

None of the 14 patients who were not vented at the apex had dyskinetic or akinetic left ventricular apices at the time of postoperative evaluation. In contrast 19 of 34 (56%) patients who received apical vents developed focal areas of dyskinesia or akinesia at the apex (P < 0.005) (table 2). Figure 2 illustrates the contraction abnormalities which were observed in three of these 19 patients. Three of the 14 (21%) nonapically vented patients had hypokinetic apical contraction following surgery while four of 34 (12%) apically vented patients had hypokinetic apical wall motion at the time of postoperative evaluation. Eleven of 14 (79%) nonapically vented patients, and 11 of 34 (32%) apically vented patients had normal apical contraction on the postoperative ventriculogram.

Incidences of new nonapical contraction abnormalities

Figure 1. Tubbs dilator (closed position) (9 mm) and venting catheter (7 mm).
TABLE 1. Comparison of Patients Who Were Vented at the Apex and Patients Who Were Not Vented at the Apex

<table>
<thead>
<tr>
<th>Variables</th>
<th>Preoperatively vented patients</th>
<th>Patients not preoperatively vented</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>34</td>
<td>14</td>
</tr>
<tr>
<td>Age (mean and sd)</td>
<td>49 (5.3)</td>
<td>50 (8.4)</td>
</tr>
<tr>
<td>Severity of angina NYHA class</td>
<td></td>
<td></td>
</tr>
<tr>
<td>II (%)</td>
<td>26</td>
<td>14</td>
</tr>
<tr>
<td>III (%)</td>
<td>21</td>
<td>22</td>
</tr>
<tr>
<td>IV (%)</td>
<td>53</td>
<td>64</td>
</tr>
<tr>
<td>History of congestive heart failure (%)</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>ECG evidence of myocardial infarction (%)</td>
<td>23</td>
<td>28</td>
</tr>
<tr>
<td>Cardiac Index (L/min/m²) (mean and sd)</td>
<td>3.1 (.6)</td>
<td>3.2 (.6)</td>
</tr>
<tr>
<td>A-VO₂ difference (vol%) (mean and sd)</td>
<td>4.5 (.6)</td>
<td>4.5 (.6)</td>
</tr>
<tr>
<td>Surgical and Perioperative Variables</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average number of coronary bypass grafts per patient</td>
<td>1.7</td>
<td>1.5</td>
</tr>
<tr>
<td>% of patients with grafts to LAD</td>
<td>85</td>
<td>79</td>
</tr>
<tr>
<td>ECG evidence of perioperative infarction (%)</td>
<td>20.5</td>
<td>14.3</td>
</tr>
<tr>
<td>% of patients on cardio-pulmonary bypass during surgery</td>
<td>100</td>
<td>93</td>
</tr>
<tr>
<td>Postoperative Follow-up (9-15 months)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ECG changes indicative of perioperative infarction (%)</td>
<td>14.7</td>
<td>14.3</td>
</tr>
<tr>
<td>Graft Patency:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of all grafts patent at postoperative catheterization</td>
<td>81%</td>
<td>50/62</td>
</tr>
<tr>
<td>% of grafts to LAD patent at postoperative catheterization</td>
<td>76%</td>
<td>22/29</td>
</tr>
<tr>
<td>Congestive heart failure (%) at postoperative catheterization</td>
<td>64%</td>
<td>7/11</td>
</tr>
<tr>
<td>Antegrade flow to apex through the distal LAD</td>
<td>58%</td>
<td>71%</td>
</tr>
</tbody>
</table>

None of the differences were statistically significant by Student's t-test.

were also determined in the two groups. Ten of the 34 apically vented patients (30%) and six of the 14 nonapically vented patients (43%) (NS) had new asynergy of either the anterior or posterior walls. Thus the groups were different only in regard to changes in contraction patterns at the apex.

Coronary angiograms at the time of restudy were reviewed to determine if there was any difference in pattern of blood flow to the apex which might account for the apical asynergy that was observed in the apically vented patients. In all patients the left anterior descending artery (LAD) extended to the apex. The distal portion of the LAD located at the apex filled in antegrade manner in 19 of 34 (56%) apically vented patients and 10 of 14 (71%) nonapically vented patients. This was not a statistically significant difference. However, within the subgroup of the 19 apically vented patients with apical dyskinesia or akinesia, 11 (58%) with no antegrade flow to the apex via the LAD could be identified. Only four of the 15 (27%) apically vented patients who did not demonstrate new dyskinesia or akinesia had absence of antegrade flow (P < 0.05). Occlusion of the graft to the LAD and/or progression of disease in the distal LAD were identified in all 15, and were presumed to explain the absence of antegrade filling (fig. 3).

None of the 12 patients with mitral stenosis had angina pectoris or QRS changes indicative of infarction. Ten of them were females and the mean age was 45.5 years. The coronary arteries were interpreted as normal in the two patients who had coronary arteriograms. Only one of the 12 patients (8%) with mitral stenosis had apical akinesia or dyskinesia that was evident on the postoperative ventriculogram — a marked contrast (P < 0.05) to the apically vented patients who underwent coronary artery bypass surgery.

TABLE 2. Postoperative Contraction Patterns at the Apex

<table>
<thead>
<tr>
<th></th>
<th>Normal (%)</th>
<th>Hypokinesia (%)</th>
<th>Akinesia (%)</th>
<th>Dyskinesia (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apical vent, coronary bypass graft (34 pts)</td>
<td>32% (11)</td>
<td>12% (4)</td>
<td>12% (4)</td>
<td>44% (15)</td>
</tr>
<tr>
<td>No apical vent, coronary bypass graft (14 pts)</td>
<td>79% (11)</td>
<td>21% (3)</td>
<td>0% (0)</td>
<td>0% (0)</td>
</tr>
<tr>
<td>Mitral valvulotomy, Tubbs dilator (12 pts)</td>
<td>75% (9)</td>
<td>17% (2)</td>
<td>8% (1)</td>
<td>0% (0)</td>
</tr>
</tbody>
</table>
Discussion

Coronary artery bypass surgery has variable effects upon myocardial contraction. Two important determinants are the potential reversibility of the myocardial damage and the blood flow provided by the grafted vessels. 

It is evident from the present study that location of vent site should also be a consideration in assessing the effect of surgery on myocardial contraction.

In the patients with coronary artery disease included in this study, all of whom had normal apical contraction preoperatively, 56% of the apically vented group and none in the group without apical vents had apical dyskinesia or akinesia following surgery. Because the two groups were comparable in other respects summarized in table 1, this suggests that apical venting causes focal areas of abnormal contraction in a significant number of patients with coronary artery disease.

Ischemia or infarction, which also result in asynergy, would be expected to occur with similar frequency in the two groups and should not cause the difference in incidence of focal apical contraction abnormalities that were observed. Because of the lack of sensitivity of the electrocardiogram in diagnosing apical myocardial infarction and of coronary angiograms in detecting focal areas of ischemia, it is impossible to state the exact number of patients in whom apical ischemia or infarction was present. The absence of a significant difference in patency of grafts to the LAD or presence of antegrade flow to the apex between apically vented and nonapically vented patients substantiates but does not prove the hypothesis that apical ischemia was not the sole cause of the abnormalities in apical contraction observed in the coronary bypass patients.

The comparison of apical contraction in the mitral stenosis patients, in whom coronary artery disease was unlikely, with the apically vented coronary bypass patients was employed to test the hypothesis that apical manipulation alone was responsible for producing apical asynergy. The technique of insertion of a Tubbs dilator, which was approximately the same diameter as an apical venting catheter (fig. 1), into the left ventricular apex in patients with mitral stenosis was similar to that of apical venting. Closure of the apical wound in both operations was accomplished by insertion of a purse string suture.

Focal apical dyskinesia or akinesia following closed mitral commissurotomy with a Tubbs dilator was infrequent compared with incidence in apically vented coronary bypass patients. Only one of 12 (8%) mitral stenosis patients had apical dyskinesia or akinesia on the postoperative ventriculogram. The different incidence of apical contraction abnormalities between these two groups of patients (P < 0.05) might have been due to different procedures, to the fact that cardiopulmonary bypass was not utilized during the mitral valvulotomy procedure, or to differences in the ability of the apical myocardia to heal. The first explanation is unlikely because of the similarity of procedures involving the left ventricular apex. The role of cardiopulmonary bypass cannot be ascertained from this study. The third explanation is quite possible since it has been established that healing is at least partially dependent on adequate blood flow.19 The poorer blood supply due to coronary arterial insufficiency may render the myocardium susceptible to a similar impairment in healing in patients with coronary artery disease. This conclusion is supported by the higher incidence of absence of antegrade filling of the distal LAD in the subgroup of apically vented patients who developed apical asynergy in this study. Thus it is possible that the combination of abnormal blood supply and trauma, rather than either alone, was responsible for the focal asynergy that was observed.

The long-term clinical significance of apical contraction abnormalities in patients with coronary artery disease remains unknown. During the 9-15 month follow-up period, no patients with apical dyskinesia or akinesia had symptoms of congestive heart failure or peripheral embolism. In chronic disease, however, the interrelationships of many factors determine the eventual outcome of patients. The location of vent site during coronary bypass surgery is one variable being entered into the computerized data base.20 The long term clinical significance of venting via the left ventricular apex will only be established by continued follow-up of these patients.

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The Relationship between Enzymatic and Histologic Estimates of the Extent of Myocardial Infarction in Conscious Dogs with Permanent Coronary Occlusion

CHARLES R. ROE, M.D., FREDERICK R. COBB, M.D., AND C. FRANK STARMER, PH.D.

SUMMARY Relationships between enzyme estimates (EE) and histologic measurements of infarct size (HIS) were analyzed in 14 conscious dogs with permanent occlusion of the circumflex coronary artery. EE were derived from serial CPK, CPK-MM, CPK-MB, and CPK-BB. Estimates were obtained using methods of Shell et al., Norris et al., and Roberts et al. HIS was determined from multiple histologic sections 5–6 days after infarction.

In 14 animals, HIS ranged from 0.1–26.6 grams. Regression analysis demonstrated poor correlation ($r^2 < 0.06$) between EE by each method and HIS. Using the Shell method and restricting the analysis to HIS of < 13 grams, improved the correlation ($r^2 = 0.42$).

In 1971 SHELL ET AL. proposed a mathematical model for estimation of the extent of acute myocardial infarction in dogs. Recently an assessment of the sensitivity of the original method to reported variation in values for each parameter was presented. These variations produced significant distortion of the estimates. Newer modifications include the introduction of the concept of the individualized disappearance rate parameter ($K_a$) by Norris and associates. In a recent report, Roberts modified values for the original parameters and included the concept of the individualized $K_a$. All three techniques are currently being used to estimate the extent of infarction. In this paper we evaluate each of them, using the same experimental data, relative to histological analyses. We tested, in awake dogs, the correlation between enzyme estimates and the histologic extent of myocardial infarction.

Methods

Animal Preparation

Fourteen mongrel dogs weighing between 13 and 28 kg were subjected to a left thoracotomy. In order to produce a
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