Significance of New Q Waves after Aortocoronary Bypass Surgery
Correlation with Changes in Ventricular Wall Motion

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SUMMARY
From June 1969 to December 1974, 142 patients underwent aortocoronary bypass surgery at the Veterans Administration Hospital, San Francisco. Seventeen patients developed new Q waves; two of four deaths in the hospital occurred among these 17 patients. Preoperative and postoperative catheterization data were available for analysis in 12 of the 15 surviving patients. Longitudinal, transverse, and hemiagonal segmental analysis of preoperative and postoperative single plane, 30° right anterior oblique left ventriculograms revealed deterioration in wall motion in all 12 patients. The deterioration corresponded to the area on the electrocardiogram where the new Q waves appeared. In ten of 12 patients, ejection fraction also deteriorated. Twenty-six of 29 grafts were patent. New Q waves after aortocoronary bypass surgery correlate well with the appearance of localized abnormalities in wall motion. We found no evidence that perioperative appearance of Q waves was not due to new perioperative myocardial infarction.

AORTOCORONARY SAPHENOUS VEIN BYPASS surgery has been widely applied in the treatment of coronary artery disease. This has stimulated large scale efforts to evaluate its effectiveness. The occurrence of perioperative Q waves is an important consideration in evaluating the results of bypass surgery. New myocardial infarctions occur in between 7% and 40% of patients, based on inspection of postoperative electrocardiograms or vectorcardiograms. However, Bassan et al. suggested that the appearance of Q waves postoperatively does not necessarily signify new myocardial infarction. Therefore, we investigated the significance of new Q waves appearing after aortocoronary saphenous vein bypass surgery with respect to changes in left ventricular wall motion.

Materials and Methods
One hundred forty-two patients from the Veterans Administration Hospital, San Francisco, underwent aortocoronary saphenous vein bypass surgery from June 1969 through December 1974. Patients who had valve replacement, ventricular aneurysmectomy, or both in combination with insertion of a bypass are not included in the present report. Preoperative and postoperative electrocardiograms were reviewed by independent observers, and the appearance of new Q waves was noted. Preoperative electrocardiograms were taken no longer than 48 hours before surgery, and the postoperative electrocardiograms were obtained immediately postoperatively and daily during the first postoperative week. Preoperative and postoperative vectorcardiograms were also obtained for many of these patients.

Seventeen patients developed new Q waves during the immediate postoperative period; two of them died before leaving the hospital. One patient was lost to our follow-up. Of the remaining 14 patients, two had postoperative studies that were technically inadequate for analysis, leaving 12 patients with adequate preoperative and postoperative left ventriculograms available for analysis. Also included in this report is a 13th patient, who had postoperative reappearance of Q waves that had been present on an electrocardiogram seven months before surgery, but not on an immediate postoperative electrocardiogram. Sixty additional patients from this series, all of whom had preoperative and postoperative studies adequate for analysis, and none of whom developed perioperative Q waves, were studied as controls.

All patients undergoing aortocoronary saphenous vein bypass surgery were asked to return for postoperative studies, including graft injection and left ventriculography, done according to a protocol approved by the University of California Committee on Human Experimentation. In the perioperative Q wave group, catheterizations were performed at a mean of eight months postoperatively, with a range of three to 32 months. In the control group, the postoperative studies were performed at a mean of five months postoperatively (range two to 13 months).

Left ventriculography was performed in the single plane, 30° right anterior oblique projection. The left
ventriculograms were quantitatively characterized by longitudinal, transverse, and hemiaxial segmental analysis according to the method of Herman et al. In this method, the long axis of the ventricle from the midaortic root to the apex and a bisecting perpendicular are constructed on both end-systolic and end-diastolic films in the 30⁰ right anterior oblique projection. The end-systolic and end-diastolic profiles are then superimposed on the same axes and its perpendicular as reference points. Preoperative and postoperative left ventricular function could thus be assessed for symmetry and degree of abnormalities of wall motion (fig. 2).

Ejection fractions were determined using volumes obtained by the area-length method.

**Results**

Of the 142 patients reviewed there were four hospital deaths, an operative mortality rate of 2.8%. Seventeen patients developed new Q wave changes, an incidence of 11.9%. Of the four operative deaths, two occurred among these 17 patients, 11.8%.

The 12 patients studied were men, ranging in age from 41 to 65 years (mean 55 years). Twenty-nine grafts were placed, and three were judged to be occluded (patency rate, 89.6%).

Seven of the 12 patients (58%) had electrocardiographic evidence of old infarction before catheterization and surgery. The distribution of new Q waves in these patients was: two anteroseptal, six inferior, two anterolateral, and two anterior and inferior (table 1 and fig. 2). The results of the preoperative and postoperative vectorcardiograms, the extent of preoperative coronary artery disease, graft patency, ejection fraction, and left ventricular end-diastolic pressure are listed in table 1.

Segmental analysis of preoperative and postoperative ventriculograms showed deterioration of wall motion postoperatively in all 12 patients, corresponding to the area of new Q waves (fig. 2). Postoperatively, ten of the 12 patients showed deterioration in ejection fraction.

Sixty additional patients, who had adequate preoperative and postoperative left ventriculograms, served as controls. None of these patients had any electrocardiographic evidence of new Q waves after operation. The control patients were all men ranging in age from 38 to 69 years (mean 52 years). One hundred fifty-three grafts were placed, and 22 were judged to be occluded (patency rate, 88.6%) (table 2). Of 60 control patients, 35 (58%) had electrocardiographic evidence of old myocardial infarction before surgery.

Of 420 wall segments analyzed (seven segments for each of the 60 patients), 115 deteriorated by 20% or greater. In the 12 patients who had new perioperative Q waves, 46 of 84 segments deteriorated by 20% or more. The difference between these two groups was significant at a level of P < 0.005 by Chi² analysis.
### Table 1

**Summary of Preoperative and Postoperative Clinical Data on 13 Male Patients Who Developed Perioperative Q Waves after Bypass Surgery**

<table>
<thead>
<tr>
<th>Patient, age</th>
<th>Interval between surgery/ follow-up (months)</th>
<th>Preoperative status of coronary arteries (% occluded)</th>
<th>Graft patent</th>
<th>Electrocardiogram</th>
<th>Vectorcardiogram</th>
<th>Ejection fraction (% volume)</th>
<th>Left ventricular end-diastolic pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RCA</td>
<td>LAD</td>
<td>LCF</td>
<td>RCA</td>
<td>LAD</td>
<td>LCF</td>
<td>Before surgery</td>
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<tr>
<td>1. 55</td>
<td>22</td>
<td>99</td>
<td>90</td>
<td>95</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>2. 47</td>
<td>3</td>
<td>60</td>
<td>70</td>
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<tr>
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<td>80</td>
<td>100</td>
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<tr>
<td>4. 63</td>
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<td>60</td>
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<td>*</td>
<td>*</td>
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<tr>
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<td>80</td>
<td>80</td>
<td>Yes</td>
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<tr>
<td>6. 62</td>
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<td>99</td>
<td>90</td>
<td>30</td>
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<td>7. 59</td>
<td>12</td>
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<td>80</td>
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<tr>
<td>8. 62</td>
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<td>80</td>
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<td>Yes</td>
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</tr>
<tr>
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<td>70</td>
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<tr>
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<td>3</td>
<td>99</td>
<td>100</td>
<td>90</td>
<td>Yes</td>
<td>Yes</td>
<td>ND</td>
</tr>
</tbody>
</table>

*Patient 4 had received a diagonal graft which was patent at follow-up.
†Patients 7 and 10 each received two grafts to the LCF system.
‡Patient 13, who had postoperative reappearance of Q waves, is discussed in the text.

Abbreviations: AMI = anterior myocardial infarction; ASMI = anteroseptal myocardial infarction; ALSIM = anterior lateral myocardial infarction; IMI = inferior myocardial infarction; LAD = left anterior descending artery; LCF = left circumflex coronary artery; LMCI = lateral myocardial infarction; LVH = left ventricular hypertrophy; PMI = true posterior myocardial infarction; RBBB = right bundle branch block; RCA = right coronary artery; ND = not done.
Discussion

Aortocoronary bypass surgery is widely applied to treat symptoms of ischemic heart disease, i.e., angina pectoris. In evaluating this procedure, one must try to appreciate the risks as well as the benefits derived. It is well recognized that about 85% of patients undergoing coronary artery bypass surgery have either total or significant relief of their symptoms of chest pain.\(^\text{15}\)

However, to achieve this one must accept an operative mortality of 3 to 10%.\(^\text{16, 17}\) The perioperative appearance of Q waves, a major cause of significant morbidity with this procedure, is reported to be between 7 and 40%.\(^\text{19}\) The present study deals with the left ventriculographic significance of these Q waves.

Good correlation has been shown between abnor-
malities of left ventricular wall motion and electrocardiographic evidence of previous myocardial infarction.\textsuperscript{11-13, 18} Stewart et al.\textsuperscript{18} recently reported on six patients in whom left ventriculography was performed before and after the occurrence of myocardial infarction; no patient had cardiac surgery. Five of their six patients developed new or more severe abnormalities in left ventricular wall motion in the areas that, by electrocardiogram, were affected by myocardial infarction. Five of the six patients also had reduced ejection fractions after myocardial infarction. However, Bassan et al.\textsuperscript{10} reported that, in four of 11 patients developing new Q waves after bypass surgery, there was no evidence of increased abnormalities in wall motion that correlated with the distribution of the Q waves. In fact, they found improved wall motion in the area opposite the location of electrocardiographic Q waves.

In the present study 17 of 142 patients undergoing bypass surgery developed perioperative Q waves. Of these 17 patients, two died in the hospital. All 12 of our patients developed new abnormalities in wall motion in the area of the perioperative Q waves. Ten of the 12 had decreased ejection fractions. Because our findings differed from those of Bassan et al.,\textsuperscript{10} attempts were made to determine if our data were in any way biased to show abnormalities of wall motion. In addition, an attempt was made to determine if patients in the perioperative Q wave group differed from patients not developing perioperative Q waves. Accordingly, 60 patients undergoing aortocoronary bypass surgery during the same interval and who had adequate preoperative and postoperative studies were randomly selected as controls.

Since all patients in the study were operated upon by the same surgeon (D.U.), it is unlikely that surgical technique played a large role, except that as more patients have undergone surgery there has been a fall in the operative mortality rate and the incidence of perioperative Q waves has decreased.

Our postoperative studies were not conducted at uniform time intervals after surgery. Although the mean times of postoperative study of the control group and the perioperative Q wave group are not
statistically different, it is possible that the perioperative Q wave patients, especially the two who were studied 22 and 32 months after surgery, developed abnormalities of wall motion as part of the natural progression of their coronary artery disease. However, all grafts in these two patients were patent. In addition, clinical follow-up revealed no further evidence of myocardial infarction, such as elevation of cardiac enzymes or additional electrocardiographic changes, in the late postoperative period.

There is no difference in graft patency for the two groups (table 2). Analysis of time on cardiopulmonary bypass shows that the perioperative Q wave group was on bypass an average of ten minutes longer than the control group, but the difference is not statistically significant. Comparison of preoperative electrocardiograms showed statistically equal percentages of normal tracings and of old myocardial infarctions (table 2).

Because the contrast agent may depress over-all myocardial function and could, therefore, enhance a local abnormality in wall motion, the order in which the left ventriculograms were performed in relation to selective angiograms was investigated (table 3). In patients 1, 2, and 3, the preoperative left ventricu-
triculogram preoperatively but not postoperatively. However, postoperatively the patient was taking a digitalis glycoside. Because nitroglycerin can increase ejection fraction, it might be argued that the preoperative ejection fraction was artificially elevated. This patient worked as a heavy equipment operator until four days before he was admitted for surgery. There was no evidence of an acute myocardial infarction preoperatively. During surgery, there was great difficulty weaning the patient from cardiopulmonary bypass, and his circulation was assisted with an intra-aortic balloon device for four days. Since discharge he has been very limited by symptoms of congestive heart failure. In fact, he has had two hospital admissions in less than 12 months because of pulmonary edema. He is one of two patients in our study who are functionally class IV because of symptoms of congestive heart failure. It is unlikely that this patient’s left ventricular function was as poor preoperatively as it is postoperatively.

No patient was taking digitalis glycoside preoperatively but patients 4 and 5 were taking glycosides postoperatively. Finally, no patients received propranolol preoperatively or postoperatively.

It is widely recognized that Q waves after myocardial infarction may regress over time.22 Bassan et al. hypothesized that Q waves might reappear in an area of old infarction if an ischemic contralateral wall became normally perfused postoperatively.10 Our patient 13 had had an inferior myocardial infarction diagnosed by electrocardiogram and vectorcardiogram seven months before surgery. The preoperative electrocardiogram and vectorcardiogram were not diagnostic of an inferior myocardial infarction, but left ventriculographic analysis showed inferior wall hypokinesis. Postoperative Q waves were seen in leads II, III, and aVF of the electrocardiogram, and the vectorcardiogram revealed an inferior myocardial infarction. Bypass grafts to the right coronary artery and left anterior descending artery were both patent. The postoperative left ventriculogram showed that the inferior wall remained hypokinetic, and there was distinct deterioration postoperatively of anterior wall motion (fig. 3). It appears unlikely that our patient’s anterior wall was electrically ischemic but mechanically intact preoperatively and mechanically ischemic but electrically intact postoperatively.

Patient 3 developed perioperative Q waves in the inferior leads of the electrocardiogram, and had improved motion in his anterior wall with regeneration of R waves in the anterior precordial leads of the electrocardiogram. These changes occurred after insertion of a graft to the left anterior descending artery. It is

![Diagram](image)

**Figure 3**

Preoperative and postoperative ventriculograms of patient 13, who had postoperative reappearance of Q waves.

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**Table 4**

Heart Rate, Deterioration of Wall Segments, and Ejection Fraction in Patients with Perioperative Q Waves and in Two Subgroups of Control Patients

<table>
<thead>
<tr>
<th>Patient group</th>
<th>No.</th>
<th>Heart rate (mean ± sd)†</th>
<th>Wall segments‡</th>
<th>Ejection fraction (mean ± sd)¶</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Preoperative Postoperative</td>
<td>No. deteriorated /no. examined</td>
<td>% deteriorated Preoperative Postoperative</td>
</tr>
<tr>
<td>Perioperative</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q wave</td>
<td>12</td>
<td>74.7 ± 10.4 81.9 ± 15.2</td>
<td>46/84</td>
<td>55</td>
</tr>
<tr>
<td>Control A</td>
<td>51</td>
<td>73.2 ± 11.2 81.2 ± 14.2</td>
<td>101/357</td>
<td>28</td>
</tr>
<tr>
<td>Control B</td>
<td>9</td>
<td>81.1 ± 12.3 82.0 ± 8.8</td>
<td>14/63</td>
<td>22</td>
</tr>
</tbody>
</table>

*In Control Group B, the preoperative left ventriculograms were performed before selective angiography and the postoperative left ventriculograms were performed after selective angiography. Control subgroup A includes the remainder of the 60 control patients.

†By Student's unpaired t-test, there were no significant differences between heart rates before and after surgery in any group, nor between groups compared before or after surgery.

‡By Chi² analysis, there was a significant difference (P < 0.005) between perioperative Q wave patients and combined control patient groups.

¶By Student’s t-test, there was a significant difference in ejection fractions before and after surgery in the perioperative Q wave group (P < 0.05); neither control subgroup had a significant difference.
unlikely that this represents unmasking of an old inferior infarction since there was deterioration of inferior wall motion postoperatively and the graft to the right coronary artery was occluded.

In our experience, new Q waves after aortocoronary bypass surgery correlated well with the appearance of localized abnormalities of wall motion in our 12 patients. Our data support the hypothesis that the appearance of a Q wave is accompanied by deterioration of left ventricular wall motion in the area of the new electrocardiographic changes. Four of the 12 patients in our study are functionally in class IV (two with congestive heart failure and two with chest pain), whereas none of the control patients are in this functional class. Therefore, it seems that the appearance of perioperative Q waves after aortocoronary bypass surgery is associated with: 1) deterioration of left ventricular wall motion, 2) an increased risk of operative death, and 3) poor functional outcome. There is no evidence to suspect that the perioperative Q wave changes seen in our patients were not due to new perioperative myocardial infarctions.

Acknowledgment

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References

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