Non–Q wave perioperative myocardial infarction: assessment of the incidence and severity of regional dysfunction with quantitative two-dimensional echocardiography

THOMAS FORCE, M.D., ANDREW J. KEMPER, M.D., PETER BLOOMFIELD, M.B., DONALD E. TOW, M.D., SHUKRI F. KHURI, M.D., MIGUEL JOSA, M.D., AND ALFRED F. PARISI, M.D.

ABSTRACT  Since the widespread use of hypothermic potassium cardioplegia began, marked reductions in perioperative mortality and the rate of Q wave–associated myocardial infarctions have been noted. No study to date has evaluated whether there has been an equally dramatic improvement in the incidence of postoperative myocardial infarctions unassociated with Q wave development. We used a previously validated quantitative two-dimensional echocardiographic analytic algorithm to determine the incidence and severity of regional wall motion abnormalities (RWMAs) and first-pass radionuclide ventriculography to assess deterioration in global left ventricular function in the four following groups of patients (total n = 65): (1) those with peak postoperative creatine kinase (CK)–MB levels equal to or less than the mean value for patients undergoing coronary artery bypass surgery at our institution (n = 10), (2) those with CK-MB levels between the mean and 1 SD above the mean (n = 10), (3) those with peak CK-MB levels higher than 1 SD above the mean (n = 25), and (4) those with new pathologic Q waves on the postoperative electrocardiogram (n = 20). All patients had electrocardiograms without pathologic Q waves and normal wall motion and ejection fraction by contrast ventriculography before surgery. The incidence of postoperative RWMA by two-dimensional echocardiography for groups 1 through 4 was 0%, 20%, 55%, and 89%, respectively. Percent of abnormal left ventricular segments, wall motion scores, and the deterioration in left ventricular ejection fraction as assessed by radionuclide ventriculography were similar for patients with new RWMAs whether or not new Q waves developed (p = NS for all). In summary, although the incidence of both Q wave and non–Q wave perioperative myocardial infarctions appears to have declined significantly with the use of cold potassium cardioplegia, the incidence of non–Q wave infarction remains high and in this study was calculated to be three times greater than the 4.5% incidence of Q wave infarction. Furthermore, the impact of these non–Q wave myocardial infarctions on left ventricular function is significant and equal to that of Q wave infarctions. Future evaluations of methods of myocardial preservation should include analyses of regional left ventricular function designed to identify these patients.

many patients as those developing pathologic Q waves.4–8

Similar studies have not been performed since the use of cold cardioplegia has become widespread. Thus, although cardioplegia has dramatically reduced the incidence of Q wave myocardial infarction, the effect it has had on the incidence of deterioration in regional function due to non-Q wave myocardial infarction is not known. Furthermore, neither the extent of these regional abnormalities nor their effects on global left ventricular function have been examined adequately. This may be due to the lack of adequate noninvasive methods of regional wall motion analysis. In the perioperative period, current methods have been unable to differentiate the normal apparent septal hypokinesis seen in most patients undergoing uncomplicated CABG from that due to infarction.9–13 In a prior study we evaluated a quantitative two-dimensional echocardiographic analytic algorithm for wall motion using a floating-axis system and found it to be a reliable method for analyzing regional left ventricular function, including that of the interventricular septum. 14 Subsequently we found it to be accurate at differentiating patients with perioperative Q wave myocardial infarction from patients undergoing uncomplicated CABG. 15 Sensitivity of the method was 88% and specificity was 80%. In the present study, we applied this system of analysis to 65 patients undergoing CABG to determine the incidence of perioperative RW MAs unassociated with new Q waves on the electrocardiogram. We also compared the effects of these RW MAs on regional and global left ventricular function with those in a group of patients with Q wave–associated perioperative myocardial infarction.

Methods

Patient selection. Only patients with no documented history of a prior myocardial infarction who were scheduled for CABG surgery were considered eligible for study. Patients undergoing concomitant valve replacement or aneurysmectomy were excluded. The electrocardiograms of all subjects were reviewed and patients with preexisting pathologic Q waves (>0.04 sec in duration) were excluded. The preoperative contrast ventriculograms (done no more than 3 months before surgery) were reviewed and only patients with normal regional wall motion, by the consensus of three observers, and global ejection fraction greater than or equal to 55% were considered eligible. After CABG, all perioperative electrocardiograms were reviewed and patients who survived for 24 hr after surgery and who did not develop new pathologic Q waves were divided into three groups based on the peak creatine kinase (CK)–MB isoenzyme level. We had previously determined the mean and SD of peak CK-MB at our hospital to be 92 ± 49 IU/liter by reviewing the data on a consecutive series of 106 patients who underwent CABG without developing new Q waves. Group 1 was defined as those patients with peak CK-MB between 0 and 92 IU/liter. The peak value for group 2 patients was between 93 and 141 IU/liter and that for group 3 patients was greater than 141 IU/liter. The final group studied, group 4, was composed of those patients who developed new pathologic Q waves in the perioperative period.

Before the commencement of the study we established goals with regard to the number of patients from each group that would be included in the study because we wished to avoid studying large numbers of patients with high probabilities of having normal postoperative wall motion. Accordingly, based on data from our prior studies, 18, 19 our goal was to include 10 patients from group 1, 10 patients from group 2, 25 patients from group 3, and 20 patients from group 4.

Surgical technique. All patients underwent cardiopulmonary bypass at a systemic temperature of 25°C. The left ventricle was vented through the right superior pulmonary vein. Distal bypass graft anastomoses were performed during one period of aortic cross-clamping. Myocardial preservation was achieved with cold (4°C) potassium crystalloid cardioplegia administered through the aortic root and through the free ends of the grafts immediately after the distal anastomoses were completed. Topical hypothermia was produced with iced saline slush. Myocardial temperature was continuously monitored in the interventricular septum anteriorly in all patients and was maintained below 15°C throughout the period of aortic cross-clamping. The proximal anastomoses were performed with an aortic side-biting clamp while the patient was being rewarmed. In all patients segments of reversed saphenous vein were used exclusively. The pericardium was left open after surgery in each case.

Determination of cardiac enzyme levels. Total serum CK activity was determined by a modification of the method of Rosalki. 20 Total CK-MB levels were determined by a modification of the method of Somer and Konttinen, 21 as described by Wlodarski et al. 22 Blood samples were drawn immediately, 8, and 24 hr after surgery. Occasionally some patients had additional samples drawn at the discretion of their physicians.

Two-dimensional echocardiographic studies. The methods of image acquisition and quantitative analysis have been previously described in detail, 18, 19 and are reiterated briefly.

Image acquisition techniques. Two-dimensional echocardiographic studies were performed by a technician, blinded to the purpose of the study and the clinical status of the patient, with a commercially available Hewlett-Packard phased-array sector scanner (Model 77020A) with 2.5 and 3.5 MHz transducers or an ATL-ADR (Advanced Technology Labs, Inc.) 90 degree mechanical sector scanner and recorded on videotape (Sony Betamax). To allow recovery of function in regions rendered ischemic during surgery the studies were performed as late as possible in the hospital course.

Endocardial tracings were made on a transparent overlay placed on a video monitor screen. All tracing was performed without knowledge of group assignment, clinical status of the patient, CK-MB level, or electrocardiographic or radionuclide ventriculographic data.

Quantitative image analysis. Quantitative analysis of regional wall motion was made from the apical four-chamber and two-chamber (with aorta) views as described by Feigenbaum. 23 The method of floating-axis analysis with an Irex Cardio 80 computer has been previously described. 18, 19 The long axis of the left ventricle is defined by internal points of reference for each diastolic and systolic image. The long axis is identified from the midpoint of the apex to the midpoint of the mitral valve for the apical four-chamber view and from the apex to the junction of the mitral and aortic valve in the apical two-chamber view. The midpoint of the long axis for each end-diastolic and end-systolic image is determined by the computer. The end-systolic image is transposed so that the long axes and midpoints of each image are exactly superimposed. From the midpoint of the long axis 24 radii are drawn to the systolic and diastolic outlines of the left
ventricle. The percentage change in area (area shrinkage) for each segment is then calculated from the formula (end-diastolic area - end-systolic area)/end-diastolic area × 100%.

For the apical four-chamber view the following segments were defined:18, 19: basal septal, apical septal, apical, apical lateral, and basal lateral. The apical two-chamber view was similarly divided into anterobasal, anteroapical, apical, inferoapical, and inferobasal segments. The mean percent area shrinkage was determined for each region. Hypokinesis was defined as mean percent area shrinkage more than 2 SDs below the mean value for a group of normal subjects studied previously.19

A wall motion score was generated for each patient, based on the number of regions involved and the severity of the abnormality, with a modification of the method of Hegel et al.24 Hypokinesis was defined as mean percent area shrinkage for a segment of between −10% (that is, 10% systolic expansion of the area) and +10%, and was given a score of 2. Dyskinesis was defined as systolic expansion of the area of more than 10% and was given a score of 3. Hypokinesis was a mean percent area shrinkage of between +10% and 2 SDs below the normal mean18 and was assigned a score of 1. Normal area shrinkage was scored as 0. Total score was derived by multiplying the number of dyskinetic segments by 3, akinetic segments by 2, and hypokinetic segments by 1 and adding the total. The score for the apical segment was derived by taking the mean of the score in apical four- and two-chamber views. Because normal apical endocardial motion can be minimal19, 25 and we wanted to minimize false-positive results, isolated apical hypokinesis was not considered abnormal.26 Isolated apical akinesis or dyskinesis and apical hypokinesis in association with other regional abnormalities were considered abnormal and were scored accordingly.

To assess the probability that our patient selection criteria might have failed to exclude some patients with abnormal preoperative two-dimensional echocardiograms, we applied this echocardiographic analysis system to 15 eligible patients before CABG. Eleven (73%) had complete studies. Of these, two (18%) had a regional abnormality by quantitative two-dimensional echocardiography (a specificity of 82%). In both of these, the abnormality was mild hypokinesis involving only one segment (wall motion score = 1).

Radionuclide ventriculography. First-pass radionuclide ventriculography was performed during the week before surgery and again 10 to 14 days after surgery. Details of the technique have been published previously.27 Briefly, a bolus of 15 mCi of 99mTc pertechnetate was injected into a large-bore intravenous catheter placed in an antecubital vein. A multicrystal scintillation camera in the 30 degree left anterior oblique position with 20 degrees of caudal tilt was interfaced with a computer that acquired data at 40 frames/sec. The left ventricular region of interest was defined manually and a time-activity curve was generated for the left ventricle with use of a computer algorithm. Ejection fraction was determined by averaging the counts of 5 to 8 consecutive beats. Ejection fraction determined by this technique is highly reproducible (y = 3.4 + 0.92x, where y is the first determination and x is the second; r = .97).27 Measurements of left ventricular ejection fraction in our hospital are significantly correlated with contrast cineangiography (r = .88; SEE = 0.07).28 However, ejection fractions determined by radionuclide ventriculography are consistently lower than those derived from contrast cineangiograms. The regression equation is y = 1.16x + 3.6, where y is the contrast angiographic value and x is the ventriculographic value.29 Thus, a radionuclide ventriculographic ejection fraction of 44% is equivalent to a contrast angiographic ejection fraction of 55%, the lower limit of normal in our laboratory.

Adequacy of revascularization. Adequacy of revascularization was assessed grossly by dividing the coronary circulation into three arterial distributions — the anterior (left anterior descending artery and diagonal branches), the lateral (circumflex artery and marginal branches), and the inferior (posterior descending artery and left ventricular extension branches). Maximal number of coronary arteries that could be diseased was thus three. For the purpose of this study, if a graft were placed to a region, that region was considered adequately revascularized even if a branch of the major vessel were diseased and did not receive a graft.

Statistical analyses. Differences between groups with respect to the frequency of an event were determined by the multiple chi-square analysis. Presence of significant differences between multiple groups was determined by analysis of variance and the modification of Tukey’s method for unequal sample sizes. When comparisons involved only two groups, significance was determined by the t test for independent means.

Variability. Variability in the process of identifying the long axis of the ventricle was examined previously.18 When two observers identified the long axis of the same diastolic and systolic endocardial outline, the interobserver difference in mean percent area shrinkage was 4% and 3% for the septum and lateral walls, respectively. Variability in our method of tracing endocardial outlines has also been examined.19, 30 Correlation coefficients for mean percent area shrinkage ranged from .88 to .97 for various regions of the ventricle and SEEs were less than 6% (intraobserver) and 9% (interobserver). These analyses were not repeated.

Results

Patient population. Preoperative characteristics of the four groups were similar. Ejection fraction by contrast angiography (69 ± 5%, 63 ± 8%, 68 ± 6%, 67 ± 8% for groups 1 through 4, respectively) and mean number of vessels diseased (2.5 ± 0.7, 2.9 ± 0.4, 2.7 ± 0.7, 2.7 ± 0.6 for groups 1 through 4) were not significantly different. The patients in groups 1 through 3 were enrolled over a 16 month period and those in group 4 were enrolled over a 34 month period. Eighty patients (58% of all eligible patients) had a peak CK-MB of 92 IU/liter or less. Ten of these were randomly chosen for entry into the study. Thirty-two patients (23% of all eligible patients) had a peak CK-MB of 93 to 141 IU/liter and 10 of these were also selected. All 20 of the patients from groups 1 and 2 survived to undergo two-dimensional echocardiographic examination. All 25 group 3 patients (18% of all eligible patients) were entered into the study, but two were transferred to another institution and one died before undergoing two-dimensional echocardiographic examination. Thus, 22 or 88% of all eligible group 3 patients were studied. All eligible group 4 patients were entered into the study; one died and the remaining 19 (95%) underwent two-dimensional echocardiographic examination. The overall incidence of Q wave perioperative myocardial infarction during the 16 month period was 4.5%. The new Q waves were located in anterior leads in eight patients, the inferior leads...
in seven, the lateral leads in one, the inferior and lateral leads in two, and the anterior and inferior leads in two.

Complete two-dimensional echocardiographic examinations technically adequate for quantitative analysis were obtained for 79% of patients with new Q waves and 83% of those without new Q waves on the electrocardiogram. Studies in patients in group 1 were performed 12.2 ± 3.7 days after surgery, those in group 2 were performed at 12 ± 5.9 days, those in group 3 at 13.6 ± 6.8 days, and those in group 4 at 13.5 ± 4.8 days (p = NS). A large pericardial effusion was present on the initial study in one group 3 patient, but since the effusion had partially resolved to moderate size by the time of the follow-up study, data from the patient were included in the analysis. Seventy-four percent of patients who developed new Q waves on the electrocardiogram and 86% of patients without new Q waves underwent preoperative and postoperative radionuclide ventriculographic examinations.

Incidence of RWMA. RWMA were detected in none of the group 1 patients, two (20%) of the group 2 patients, 12 (55%) of the group 3 patients, and 17 (89%) of the group 4 patients (p < .01 for groups 3 and 4 vs group 1; p < .01 for group 4 vs group 2; no other comparisons showed a statistically significant difference). The increasing risk of a RWMA with increasing peak CK-MB can also be seen by comparing peak CK-MB values in those with vs those without such regional abnormalities (229 ± 128 vs 143 ± 149 IU/liter, respectively, p < .01).

Severity of RWMA. Those patients in groups 2 and 3 with new RWMA were compared with those in group 4 with new RWMA to determine the relative severity of systolic dysfunction. These data are presented in table 1 and in figures 1 and 2. There were no significant differences in extent or severity of regional involvement (assessed as number of abnormal regions

| TABLE 1 |
| Summary of regional function data |

<table>
<thead>
<tr>
<th></th>
<th>% of abnormal regions per patient</th>
<th>Mean wall motion score</th>
<th>Mean score per abnormal segment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-Q wave infarction (n = 14)</td>
<td>43 ± 25</td>
<td>6.2 ± 4.1</td>
<td>1.6 ± 0.8</td>
</tr>
<tr>
<td>Q wave infarction (n = 17)</td>
<td>46 ± 22</td>
<td>5.8 ± 4.0</td>
<td>1.5 ± 0.7</td>
</tr>
</tbody>
</table>

p = NS for all comparisons between Q wave and non-Q wave groups.

FIGURE 1. Frequency distribution of the number of abnormal regions per patient in Q wave and non-Q wave myocardial infarction groups.

per patient [figure 1], wall motion score [figure 2], or mean score per abnormal segment [table 1], in patients with non-Q wave and those with Q wave infarction. Absolute mean percent area shrinkage for Q wave and non-Q wave groups was compared on a segment-by-segment basis. Again, no significant differences were found.

Global left ventricular function. Changes in global left ventricular function were assessed by changes in ejection fraction from before to after surgery by radionuclide ventriculography. Before surgery the mean ejection fractions for the four groups were not significantly different and ranged from 50 ± 6.5% for group 1 to 53.5 ± 7.5% for group 3. All values were equal to or greater than 44%. Figure 3 is a plot of the change in ejection fraction in (1) patients without new Q waves and normal regional wall motion after surgery, (2) patients without new Q waves but with abnormal regional wall motion, and (3) patients with new Q waves and abnormal regional wall motion. Mean deterioration in ejection fraction for the patients without new Q waves but with a regional abnormality (8.9 ± 10%) was not significantly different from that in the Q wave group (6.7 ± 10.7%). Both of these values are signifi-
groups with higher peak CK-MB. However, in those patients with the highest levels of CK-MB (group 3), inadequate revascularization tended to predict the development of wall motion abnormalities. Eighty-three percent of inadequately revascularized group 3 patients developed a regional abnormality vs 41% of adequately revascularized patients. The trend failed to reach statistical significance.

**Discussion**

Experimental observations in animals have shown that the use of hypothermia and potassium cardioplegia can effectively preserve regional and global ventricular function as well as myocardial ultrastructure during prolonged periods of anoxia.\(^ {31-33}\) Similar data exist for patient studies,\(^ {2, 34, 35}\) but the Coronary Artery Surgery Study has provided the strongest evidence that hypothermic cardioplegia is beneficial. Berger et al.\(^ {1}\) reported reductions in operative mortality of nearly 5% over normothermic intermittent ischemic arrest. Also, rates of Q wave infarction were reduced by half when compared with normothermia or hypothermia alone. Although this data is certainly encouraging, several workers in the field have expressed the concern that statistics on survival and Q wave myocardial infarction alone may be too insensitive to properly evaluate the adequacy of current preservation techniques.\(^ {2-4}\)

Support for this viewpoint comes from the study of Bulkley and Hutchins, which was done before cold cardioplegia was used.\(^ {5}\) In their postmortem study of patients who died within 1 month of cardiac surgery, they found operative myocardial injury of some degree in 83% of patients and regional transmural necrosis in 38%. In only 14% of the patients with transmural necrosis was the injury detected by Q waves on the electrocardiogram. Further support comes from studies in which \(^ {99m}\)Tc pyrophosphate was used for imaging of myocardial infarction.\(^ {36-41}\) Although done before the advent of cold cardioplegia and not specifically addressing the problem of non-Q wave myocardial infarction, most of these studies have reported an incidence of positive \(^ {99m}\)Tc pyrophosphate scans without associated Q waves on the electrocardiogram at least as great as the incidence of positive scans with new Q waves on the electrocardiogram. Gray et al.\(^ {40}\) and Platt et al.\(^ {37}\) found incidences of positive scans without new Q waves that were 1.5 and 2 times the incidence of positive scans with new Q waves. Given the limited sensitivity of planar imaging for detecting non-Q wave myocardial infarction,\(^ {42-44}\) the true relative incidence may be significantly higher. Indeed, the three contrast ventriculographic studies (again, done before cold car-

---

**FIGURE 3.** Change in radionuclide ventriculographic ejection fraction from preoperative to postoperative study. No RWMA = patients with normal postoperative regional wall motion; RWMA = patients with new postoperative RWMA but without new Q waves; Q wave + RWMA = patients from group 4 (Q waves on postoperative electrocardiograms) with new RWMA.

---

<table>
<thead>
<tr>
<th>NO RWMA</th>
<th>RWMA</th>
<th>Q Wave + RWMA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

---

<table>
<thead>
<tr>
<th>CHANGE IN EJECTION FRACTION (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>-20</td>
</tr>
<tr>
<td>P &lt; 0.01</td>
</tr>
</tbody>
</table>

---

**Clinical correlates of non-Q wave myocardial infarction.** Unstable angina (i.e., uncontrolled angina at rest requiring surgery within 7 days) was a significant predictor of subsequent development of a wall motion abnormality. Seventy-one percent of the patients with unstable angina developed a wall motion abnormality vs 29% of those with stable angina (p < .05). Neither extent of coronary disease nor preoperative ejection fraction were predictive of RWMA.

Operative variables examined were aortic cross-clamp time (as an index of ischemic time) and adequacy of revascularization. Longer cross-clamp time was not required in those patient groups with higher peak CK-MB levels or in those who developed wall motion abnormalities (normal wall motion, 47 ± 14 min; abnormal wall motion, 51 ± 20 min). Similarly, inadequate revascularization was not more common in the

Vol. 72, No. 4, October 1985 785
dioplegia was used) that examined this question found that the incidence of deterioration in wall motion without associated Q waves on the electrocardiogram was 2.3 to 5.5 times greater than the 6.2% to 15% incidence of Q wave myocardial infarction.

In summary, use of hypothermic cardioplegia has dramatically reduced the incidence of Q wave infarction. However, contrast ventriculographic studies with adequate numbers of patients analogous to the ones referenced above have not been done since the use of cold cardioplegia has become widespread. Thus, it is not clear whether the newer preservation technique has similarly reduced the occurrence of left ventricular regional dysfunction unassociated with Q waves on the electrocardiogram, which seemingly is a more common outcome of inadequate preservation than is Q wave infarction.

There are several reasons for this lack of data. Since suboptimal preservation presumably occurs infrequently, prohibitively large numbers of consecutive (unselected) patients would need to undergo contrast ventriculography — a procedure that investigators are hesitant to do and patients are equally hesitant to undergo in the perioperative period — to collect a series of adequate size to draw meaningful conclusions. Thus, Raabe et al. studied 50 consecutive patients but found only eight with RWMAs. Although noninvasive techniques would be ideal, regional wall motion analysis with radionuclide ventriculography has been limited by its inherent fixed external-frame-of-reference system, which cannot correct for the exaggerated anteromedial systolic translation of the postoperative heart. Consequently there is false enhancement of lateral wall motion and false hypokinesis of septal segments, as noted in comparison with systolic thickening data.

We used a quantitative two-dimensional echocardiographic analysis system that has been evaluated in two previous reports. This method used an internal frame of reference or floating-axis system that corrected for the exaggerated postoperative systolic translation of the heart. Thus, regional endocardial motion closely reflected regional systolic function as measured by systolic thickening analysis. With two-dimensional echocardiographic floating-axis analysis, we detected RWMAs in 88% of patients who underwent CABG and developed new pathologic Q waves on postoperative electrocardiograms. Specificity was 80% and was increased to 90% without a decrease in sensitivity when isolated apical hypokinesis was considered normal, as suggested by the data of Sheehan et al., and Schnittger et al.

In the present study we again detected RWMAs in a high percentage of patients (89%) with new pathologic Q waves. In the patients without Q waves, the risk of developing a new wall motion abnormality increased with higher peak CK-MB values. To extrapolate an overall incidence of new RWMAs unassociated with new Q waves, the percentage of all CABG patients falling into each group during the 16 month period of the study can be multiplied by the observed incidences of wall motion abnormalities within each group. Thus, the expected incidence of RWMAs without new Q waves would be 14.4% (with a standard error of 3.5%) of 3.2 times the incidence of Q wave myocardial infarction. This relative incidence is similar to that reported in the contrast ventriculographic studies done before the advent of cold cardioplegia, although absolute incidence is markedly lower. Thus, improved methods of preservation appear to have reduced the occurrence of non-Q wave myocardial infarction in parallel with the reduction in Q wave infarction so that absolute incidence has dropped significantly but relative incidence remains high. These data confirm the suspicions of Conti et al. and others that a measure more sensitive than ones employed previously must be used to evaluate new methods of preservation.

To ensure that the observed incidence of non-Q wave infarction was not due to minor amounts of hypokinesis, we examined the effects of the non-Q wave myocardial infarction on regional and global ventricular function. Previous studies examining this question have been limited by the use of highly nonspecific electrocardiographic criteria for patient selection, qualitative assessment of contrast ventriculograms, nonblinded reviewers, study patients with unusual distributions of location of infarction, and small study populations. In general, however, results of these studies have suggested that the effects on left ventricular function of both non-Q wave and Q wave infarctions are minimal. Our results differ significantly. We found that not only are the effects on regional and global left ventricular function similar, but also that they are important in the majority of patients. We detected no difference between Q wave and non-Q wave infarction groups with respect to any parameter of regional or global function, and the ejection fraction of 43% in patients with new wall motion abnormalities deteriorated by at least 10%. Given that in patients with no evidence of myocardial infarction ejection fractions increased by a mean of 6%, this is a significant decline.

Limitations. The first potential question is whether our results can be generalized to patients being treated...
PATHOPHYSIOLOGY AND NATURAL HISTORY—NON-Q WAVE INFARCTION

at other institutions. Several factors argue that they can. First, the graft patency rates (80% at 1 year) for series of patients at our institution are equivalent to those reported by others. Second, the perioperative rate of Q wave infarction during the time of our study of 4.5% is similar to the overall rate of 3.8%\(^1\) and the rate of 6.4% for the randomized subgroup in the Coronary Artery Surgery Study. Since results with respect to these two easily identifiable outcomes of operative therapy are comparable to those at other institutions, there is no reason to suspect that marked variances in other aspects of operative and postoperative care exist.

The second limitation of our study is the lack of preoperative two-dimensional echocardiograms on our patients. Thus, the possibility that patients with preexistent wall motion abnormalities were entered into the study is raised. Given the stringent entrance criteria of no pathologic Q waves on the electrocardiogram and normal wall motion and ejection fraction by contrast ventriculography, we believe this is unlikely. To confirm this we analyzed two-dimensional echocardiograms from 15 preoperative patients who met our entrance criteria. Although 18% had a regional abnormality as assessed by quantitative two-dimensional echocardiography, the abnormalities were minimal (mild hypokinesis of one segment). Thus, it seems unlikely that many patients with preexisting significant RWMAs would have been studied.

Finally, our results may have been affected by the timing of the echocardiographic examinations, which were performed a mean of 13 days after surgery. Regional and global left ventricular function may not necessarily be stable over time in this patient population. Quantitative data on the evolution of regional contraction abnormalities in man after acute myocardial infarction are very limited. It is clear that global left ventricular function may change significantly in individual patients during the first year after nonoperative acute infarction. For these patients as a group, however, ejection fraction changes very little during this time. Postoperative changes in ventricular function do occur over time and may be related to perioperative alterations in preload and afterload, diastolic compliance, sympathetic nervous system activity, and potentially to the resolution of intraoperative or preoperative ischemia. Global ejection fraction decreases very early after CABG, but then improves to near preoperative levels by the first postoperative week. Minimal further improvement may occur over the next year. Thus, a greater incidence and severity of RWMAs might have been observed if studies had been performed during the very early postoperative period. Extrapolation from data on nonoperative acute infarction suggests that it is unlikely that any substantial improvement in wall motion would have been observed if studies had been performed at late follow-up.

In conclusion, hypothermic potassium cardioplegia has certainly reduced the incidence of perioperative Q wave myocardial infarction, and appears to have reduced the incidence of RWMAs without associated Q waves on the electrocardiogram (non-Q wave infarction) to a similar degree. However, non-Q wave infarction remains roughly three times more common than Q wave infarction, and half of the patients with non-Q wave infarcts will have a significant deterioration in ventricular function. Our findings indicate that evaluations of newer techniques of myocardial preservation should include a method that allows prospective quantitative analysis of regional left ventricular function.

We thank Dr. Peter Kowey for his review of this manuscript, Carol Cohen, Michelle Gilfoil, and Lori Perkins for their technical assistance, and Donna Kantarges and Clare Smith for their help in the preparation of this manuscript.

References


Non-Q wave perioperative myocardial infarction: assessment of the incidence and severity of regional dysfunction with quantitative two-dimensional echocardiography.
T Force, A J Kemper, P Bloomfield, D E Tow, S F Khuri, M Josa and A F Parisi