Diagnostic Criteria for Acute Myocardial Infarction in Patients Undergoing Coronary Artery Bypass Surgery

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SUMMARY Current techniques for diagnosing perioperative myocardial infarction were studied in 58 patients who underwent coronary bypass surgery. All patients had preoperative and postoperative ECGs and technetium-99m stannous pyrophosphate myocardial scintigrams; serum CK-MB was measured immediately after surgery and daily for 3 days. Postoperative bypass graft visualization and left ventriculography were performed before hospital discharge in every patient. Nine patients (16%) had new Q waves postoperatively. Five of these nine patients had positive pyrophosphate scintigrams, positive CK-MB and new wall motion abnormalities, and the remaining four had negative CK-MB, negative pyrophosphate scintigrams and no new wall motion abnormalities. Seven patients (12%) had newly positive postoperative pyrophosphate scintigrams, positive CK-MB and new wall motion abnormalities on postoperative ventriculography, but only four had new Q waves postoperatively. Eight patients (14%) had new wall motion abnormalities; seven had positive pyrophosphate scintigrams and all had positive CK-MB, but only five had new Q waves. Sixteen patients (28%) had positive CK-MB, including all patients with either positive pyrophosphate scintigrams or new wall motion abnormalities. Eight patients had positive CK-MB without other evidence of perioperative infarction.

A newly positive postoperative pyrophosphate scintigram is more sensitive and specific than the development of new postoperative Q waves for the diagnosis of hemodynamically significant perioperative myocardial infarction. CK-MB is highly sensitive, but too nonspecific to be useful for the diagnosis of perioperative infarction.

THE GOALS of coronary artery bypass surgery, in addition to relief of angina and prolonging life, include prevention of myocardial infarction and preservation of functional left ventricular muscle. To assess the efficacy of surgery in reaching these goals, the incidence of perioperative myocardial infarction must be considered. Accurate detection of myocardial infarction in patients undergoing coronary artery bypass surgery, however, may be difficult. The postoperative development of new Q waves on the ECG, elevation of serum cardiac enzymes or the finding of myocardial uptake of technetium-99m stannous pyrophosphate on a postoperative myocardial scintigram have all been proposed as diagnostic criteria for perioperative infarction. However, false-positive and false-negative results have been noted with all of these techniques.

In the absence of pathologic examination of the heart, the most specific indicator of perioperative infarction is the development of a new area of abnormal ventricular contraction. Studies of the incidence of new wall motion abnormalities after bypass surgery have shown that most patients with new postoperative Q waves on electrocardiography have corresponding areas of new postoperative left ventricular dyskinesis. However, for a variety of reasons, some patients with new Q waves do not show new wall motion abnormalities. The relationships among postoperative development of Q waves, pyrophosphate myocardial scintigraphy and new wall motion abnormalities have not been examined.

The current study was undertaken to examine the
value of the available techniques for diagnosing perioperative myocardial infarction in patients undergoing coronary artery bypass surgery. All patients had preoperative and postoperative electrocardiography, pyrophosphate myocardial scintigraphy and postoperative measurement of serum creatine kinase (CK) and the myocardial fraction of creatine kinase (CK-MB). Bypass graft visualization and left ventriculography were performed before hospital discharge after bypass surgery in every patient.

Methods

Study Population

Since October 1978, all patients undergoing elective coronary artery bypass surgery without other cardiac surgical procedures have had preoperative and postoperative technetium-99m stannous pyrophosphate myocardial scintigrams and have been asked to undergo graft visualization and left ventriculography before discharge from the hospital. Fifty-eight patients consented to undergo these procedures; eight were female and 50 were male, mean age 51.5 years. Nine patients had one vein graft, 13 had two vein grafts, 25 had three vein grafts, and 11 had four vein grafts. The operative technique was the same for all patients. The left ventricle was vented through a pulmonary vein and core cooling to 30°C was accomplished. The myocardium was cooled to 10-15°C during potassium cardioplegia. The aorta was cross clamped and the proximal anastomoses were constructed before the institution of cardiopulmonary bypass.

Preoperative coronary arteriograms were performed by the brachial or femoral technique. Each coronary artery was visualized in multiple projections. Left ventriculography was performed at least 10 minutes after coronary angiography in both the 30° right anterior oblique projection and the 60° left anterior oblique projection. A minimum of 10 minutes elapsed between ventriculograms. Forty-five milliliters of meglumine diatrizoate were injected over a 3-second period, and filming was accomplished using Kodak Double X 35-mm film at 32 frames/sec. All medications, including propranolol and long-acting nitrate preparations, were withheld for at least 12 hours before catheterization. Although sublingual nitroglycerin was frequently given to patients during coronary angiography, a minimum of 15 minutes elapsed between the administration of sublingual nitroglycerin and the first ventriculogram.

Postoperative catheterization was performed before discharge from hospital by the femoral or brachial approach. Each patent graft was visualized in at least two projections. Opacification of the grafts was aided by the placement of markers on the aorta at the time of coronary artery bypass surgery. All patients underwent left ventriculography in the 30° right anterior oblique projection and 29 patients also underwent ventriculography in the 60° left anterior oblique projection. The amount of contrast, the rate of injection and the filtering rate were the same as used in the preoperative ventriculogram. Wall motion analysis was performed as shown in figure 1. The ventricle was divided into seven segments and each segment was scored as follows: 0 — normal wall motion; 1 and 2 — mild, moderate and severe hypokinesia, respectively; 4 — akinesis; and 5 — dyskinesia. A new wall motion abnormality was defined as an increase in score of 2 or more points in any segment. Premature and post premature beats were not analyzed. Ejection fraction was calculated using the area-length method of Dodge.22 Ventriculographic analysis was performed by two observers who had no knowledge of the electrocardiographic, enzymatic or scintigraphic data. Preoperative and postoperative studies were read separately. In cases where the two observers disagreed on a segment score, the films were reviewed jointly and a consensus was reached.

Technetium-99m stannous pyrophosphate myocardial scintigrams were obtained 1–2 days preoperatively and 2–5 days postoperatively in all patients. Fifteen millicuries of technetium-99m stannous pyrophosphate were injected intravenously, and imaging was accomplished 2 hours later with an Ohio Nuclear 420 mobile gamma camera equipped with a parallel-hole, all-purpose collimator; 500,000 counts/image were accumulated. Images were obtained in the anterior, 45° left anterior oblique and left lateral projections. All myocardial scintigrams were recorded on Polaroid and x-ray film and interpreted by two investigators who had no knowledge of the results of the postoperative ECGs, serum enzymes or angiograms. Scans were classified from 0-4+ using the scoring system of Willerson,23 in which 0 represents no uptake, 1+ represents questionable uptake, 2+ represents definite uptake that is less in intensity than that of the sternum, 3+ represents definite uptake equal in intensity to that of the sternum, and 4+ represents definite uptake with intensity exceeding that of the sternum. Images with 2+ or greater uptake were considered positive for myocardial infarction.
Standard 12-lead ECGs were obtained the day before operation, immediately after surgery, daily for 3 days and before hospital discharge. ECGs were also obtained as deemed clinically appropriate. The ECGs were analyzed by two observers without knowledge of the scintigraphic, enzymatic or arteriographic results. The criterion used to define a new transmural infarction was the presence of a new Q wave of at least 0.04 second in duration in two or more anatomically adjacent leads. Loss of R-wave voltage without Q-wave development was not considered diagnostic of myocardial infarction.3

Blood for serum CK and CK-MB was obtained immediately after operation and daily for 3 days. Total CK was measured by the Rosalki technique.24 The presence of CK-MB was determined using cellulose acetate electrophoresis and visual examination for fluorescence under a Woods lamp.

Results

Regional Wall Motion

The interval from surgery to repeat cardiac catheterization was 10.5 ± 4.0 days (range 5–24 days). Fifty-five patients had repeat catheterization performed within 14 days of bypass surgery. In patients 2, 6 and 10, catheterization was done 17, 16 and 24 days postoperatively. Patient 7 had ventricular fibrillation with injection of a patent right coronary artery graft; otherwise, there were no serious complications of repeat cardiac catheterization. Eight patients (14%) had new wall motion abnormalities on the postoperative ventriculogram (table 1). In five patients, the area of new wall motion abnormality was located in the anterior or apical region of the left ventricle, in two patients in the inferoapical portion and in one patient in the posterolateral area of the left ventricle. Seven patients with new wall motion abnormalities had positive pyrophosphate scintigrams postoperatively and all had positive CK-MB, but only five patients, three with anterior infarctions, one with inferior infarction and one with lateral infarction, had new Q waves on postoperative ECGs. Two of the three patients without new Q waves showed decreased R-wave voltage in the lateral precordial leads and the third patient without new Q waves had only T-wave flattening leads in V4 to V6 on postoperative ECGs. Vein grafts to the coronary arteries supplying the area of myocardium showing new wall motion abnormalities were occluded in six of these eight patients.

Left ventricular ejection fraction decreased by 0.10 or more in seven of these patients and increased by

<p>| Table 1. Characteristics of Patients with One Abnormal Postoperative Test |
|---|---|---|---|---|</p>
<table>
<thead>
<tr>
<th>Pt</th>
<th>Q-EKG</th>
<th>PYP</th>
<th>Peak CK*</th>
<th>CWMA†</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>Ant Lat</td>
<td>3+ Ant</td>
<td>1224</td>
<td>+ Ant (0–3)</td>
</tr>
<tr>
<td>2</td>
<td>Ant</td>
<td>3+ Ant</td>
<td>989</td>
<td>+ Ap (0–4)</td>
</tr>
<tr>
<td>3</td>
<td>Ant</td>
<td>2+ Ant Lat</td>
<td>985</td>
<td>+ Ant-Sep (0–3)</td>
</tr>
<tr>
<td>4</td>
<td>Lat</td>
<td>2+ Post</td>
<td>483</td>
<td>Inf-Ap (1–3)</td>
</tr>
<tr>
<td>5</td>
<td>Inf</td>
<td>—</td>
<td>1718</td>
<td>+ Inf (1–3)</td>
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<tr>
<td>6</td>
<td>Inf</td>
<td>—</td>
<td>943</td>
<td>—</td>
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<td>9</td>
<td>Inf</td>
<td>—</td>
<td>288</td>
<td>—</td>
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<td>10</td>
<td>—</td>
<td>4+ Lat</td>
<td>542</td>
<td>+ Ant-Ap (2–4)</td>
</tr>
<tr>
<td>11</td>
<td>—</td>
<td>3+ Lat</td>
<td>1047</td>
<td>Post-Lat (0–2)</td>
</tr>
<tr>
<td>12</td>
<td>—</td>
<td>3+ Lat</td>
<td>1047</td>
<td>+ Ant (1–3)</td>
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<tr>
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<td>229</td>
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<td>—</td>
<td>—</td>
<td>776</td>
<td>+ LAD, OM</td>
</tr>
<tr>
<td>19</td>
<td>—</td>
<td>—</td>
<td>241</td>
<td>+ LAD, OM, Post-Lat</td>
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<tr>
<td>20</td>
<td>—</td>
<td>—</td>
<td>1075</td>
<td>+ RCA</td>
</tr>
</tbody>
</table>

*Normal value 2–81 IU.
†Numbers in parentheses indicate preoperative and postoperative segment scores.

Abbreviations: Q-EKG = location of new postoperative Q waves; NWMA = new wall motion abnormalities; PYP = pyrophosphate myocardial scintigram; ΔEF = change in ejection fraction; Ant = anterior; Lat = lateral; Inf = inferior; post = posterior; Sep = septal; Ap = apical; Post-Lat = posterolateral branch of the circumflex; OM = obtuse marginal coronary artery; RCA = right coronary artery; LAD = left anterior descending coronary artery; Diag = diagonal coronary artery.

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0.07 in patient 11. In patients with new wall motion abnormalities, the mean ejection fraction fell from 0.70 preoperatively to 0.53 postoperatively (p < 0.002). The mean ejection fraction did not change in patients without new wall motion abnormalities (0.68 preoperatively vs 0.71 postoperatively, p > 0.05). These changes in ejection fraction and regional wall motion were not influenced by differences in left ventricular preload or afterload, because left ventricular systolic pressure and left ventricular end-diastolic pressure were not significantly different either preoperatively or postoperatively when patients with and without new wall motion abnormalities were compared.

Electrocardiography

Nine patients (16%) had new Q waves on the postoperative ECG (table 1). New Q waves were seen in the inferior leads in five patients, in the anterior leads in three patients and in the lateral leads (V₅ and V₆) in one patient. All three patients with anterior Q waves (patients 1–3) had positive pyrophosphate scintigrams, new wall motion abnormalities of the anterior left ventricular wall on ventriculography and positive CK-MB (fig. 2). In these patients, grafts to the left anterior descending coronary artery were occluded on the postoperative study. The patient with new lateral Q waves (patient 4) also had a positive pyrophosphate scintigram postoperatively, positive CK-MB, a new wall motion abnormality and an occluded vein graft to the posterolateral branch of the circumflex coronary artery. Compared with the preoperative study, ejection fraction fell 0.10 or more in all of these patients.

Five patients had new Q waves in the inferior leads, but only patient 5 had a new wall motion abnormality and a positive CK-MB postoperatively. In this patient, a graft to the right coronary artery was occluded; the pyrophosphate scintigram done on postoperative day 3 was negative. The other four patients with new inferior Q waves showed no other evidence of an acute myocardial infarction: grafts to the right coronary arteries were patent, no new wall motion abnormalities developed and CK-MB was negative (fig. 3). Three of these four patients had negative postoperative pyrophosphate scintigrams, and the fourth (patient 6) had pre- and postoperative scintigrams showing anterolateral uptake of the radionuclide. In patients with new inferior Q waves without other laboratory evidence of acute myocardial infarction, ejection fraction increased postoperatively by 0.11 in two patients, was unchanged in one patient, and decreased by 0.06 in one patient. In the patient with new inferior Q waves and other evidence of acute myocardial infarction, ejection fraction decreased by 0.21. Thus, of nine patients with new Q waves postoperatively, only the five with other evidence of acute myocardial infarction had a significant decrease in left ventricular function.

Pyrophosphate Scintigraphy

Fifty-seven patients had negative preoperative pyrophosphate scintigrams. In patient 6, the preoperative scintigrams showed 3+ uptake in the anterolateral region, which corresponded to an area of anterior akinesis on left ventriculography. The postoperative scintigram was unchanged in this patient and was considered a negative scintigram in the data analysis. Seven patients (12%) had positive pyrophosphate scintigrams postoperatively (table 1). Uptake was anterior in three patients, lateral in three patients and posterior in one patient. All patients with a positive postoperative scintigram had a positive CK-MB and new wall motion abnormalities. Patients 1–4, with positive postoperative pyrophosphate scintigrams, had new Q waves and have already been described. Patients 10–12, with positive pyrophosphate scintigrams, had no electrocardiographic evidence of a new transmural myocardial infarction. Patient 10 had a positive pyrophosphate scintigram in the anteroapical area without evidence of transmural infarction on electrocardiography. This

![Figure 2](http://circ.ahajournals.org/doi/abs/10.1161/01.CIR.62.4.872)

**Figure 2.** Preoperative and postoperative pyrophosphate scintigrams. ECGs and outlines of the left ventricle in systole (dotted line) and diastole (solid line) in patient 2. The preoperative pyrophosphate scintigram and left ventriculogram are normal, whereas the preoperative ECG shows T-wave inversion in leads V₁ to V₅. Postoperatively, this patient had a 3+ positive pyrophosphate scintigram anteriorly, new Q waves in V₁ to V₅, severe hypokinesis of the anterior left ventricular wall and a decrease in ejection fraction (EF) from 0.72 to 0.49. CK-MB was positive. LAO = left anterior oblique.
patient had marked deterioration in ventricular function, with a decrease in ejection fraction from 0.40 preoperatively to 0.20 postoperatively and new anteroapical akinesis even though all four vein grafts were patent (fig. 4). Patient 11, with a positive pyrophosphate scintigram in the lateral wall of the left ventricle, had new posterolateral hypokinesis on postoperative ventriculography and an occluded vein graft to the obtuse marginal branch of the circumflex coronary artery. Grafts to the left anterior descending, diagonal and right coronary arteries were patent, and the ejection fraction increased from 0.58 preoperatively to 0.64 postoperatively. Patient 12 had a positive pyrophosphate scintigram in the lateral wall of the left ventricle, a new area of hypokinesis in the anterior wall on ventriculography, a decrease in ejection fraction of 0.10 and an occluded graft to the left anterior descending coronary artery. Thus, all seven patients with positive postoperative pyrophosphate scintigrams had positive CK-MB and a new wall motion abnormality corresponding to the area of pyrophosphate uptake. In six of these patients, the graft to the area of pyrophosphate uptake was occluded. Six of these seven patients had a decrease in left ventricular ejection fraction of greater than 0.10.

Creatine Kinase

Of 16 patients (28%) with positive postoperative CK-MB, eight (patients 1–5 and 10–12) had other evidence of perioperative myocardial infarction (table 1). All eight had new wall motion abnormalities, seven
had positive pyrophosphate scintigrams and five had new Q waves. Ejection fraction decreased by more than 0.10 in seven of these eight patients. In the remaining eight (patients 13–20), a positive CK-MB was the only postoperative laboratory evidence of perioperative myocardial infarction (fig. 5). Left ventricular ejection fraction decreased by more than 0.10 after surgery in two of these patients.

The relationship between the number of abnormal postoperative tests and the presence or absence of new wall motion abnormalities is shown in figure 6. Two or more postoperative studies were abnormal in eight patients. New wall motion abnormalities were present in all of these patients and the left ventricular ejection fraction decreased by 0.13 in this group. In contrast, none of the 12 patients with only a single abnormal postoperative study had a new wall motion abnormality, and the left ventricular ejection fraction increased by 0.01 after surgery in these patients.

Thirty-eight patients had no evidence of perioperative myocardial infarction. In these patients the postoperative pyrophosphate scintigrams and serum CK-MB were negative and new Q waves did not develop on postoperative ECGs. Ejection fraction decreased by more than 0.10 in only seven of these 38 patients.

Discussion

The reported incidence of perioperative myocardial infarction in patients undergoing coronary bypass surgery varies from 4–40%.

Recent series show a lower incidence than series that reflect early surgical experience. The influence of perioperative infarction on in-hospital mortality and morbidity and functional status after discharge from the hospital is controversial. Some authors have claimed that perioperative infarction in patients undergoing coronary bypass surgery is a relatively benign event with little or no adverse effect on in-hospital or posthospital morbidity or mortality.

Phillips et al. attributed this lack of adverse effect to the fact that 73% of patients with perioperative infarction have patent grafts to the area of infarction, presumably with retention of viable myocardium in these areas. Others, however, have found that a perioperative infarction adversely influences in-hospital mortality, left ventricular function and the incidence of angina pectoris and congestive heart failure after discharge from the hospital. Thus, the accurate diagnosis of perioperative myocardial infarction is essential in caring for patients who have undergone coronary artery surgery as well as in assessing the role of coronary surgery in the treatment of patients with coronary artery disease.

In patients admitted to a coronary care unit with chest pain, the development of new Q waves accompanied by evolutionary ST-T-wave changes is considered highly specific for the diagnosis of acute myocardial infarction. The Q wave is thought to represent electrically silent infarcted tissue that provides an “electrical window” on intracavitary potentials. In support of this viewpoint, Brewer et al. found that all patients who had new Q waves after
coronary bypass surgery and died and underwent autopsy had pathologic evidence of acute myocardial infarction. Sternberg et al. found that all 12 patients with new Q waves postoperatively had corresponding areas of new wall motion abnormality on postoperative ventriculography 9–32 months after surgery. Some authors have suggested that the development of new Q waves after coronary surgery may not be indicative of perioperative myocardial infarction. Morton et al. and Phillips et al. found that 33% and 25%, respectively, of patients with new postoperative Q waves did not have corresponding new wall motion abnormalities. The validity of these studies, however, is diminished because postoperative ventriculograms were performed many months after surgery in most patients. Bassan et al. restudied 48 patients 8–17 days after surgery, nine of whom had new Q waves on postoperative electrocardiography. Four of these patients had evidence of an old inferior myocardial infarction on preoperative ventriculography and an inferior left ventricular scar at surgery without inferior Q waves on preoperative electrocardiography. Postoperatively, new Q waves appeared in the inferior leads despite the absence of worsening of inferior wall motion abnormalities in these four patients. These workers concluded that revascularization of an ischemic anterior left ventricular wall can “unmask” an old, electrically silent inferior myocardial infarction and that new inferior Q waves do not necessarily indicate a new inferior infarction.

In the current study, five patients had new postoperative Q waves indicative of sufficient myocardial damage to produce new wall motion abnormalities. All five had CK-MB detected in their serum, four had positive postoperative pyrophosphate scintigrams, and in all five, vein grafts subserving the area of wall motion abnormality were occluded. However, the presence of new Q waves without other laboratory evidence of myocardial infarction was not accompanied by the development of new wall motion abnormalities and occurred despite patent vein grafts to the area of myocardium reflected by these Q waves. We believe that these patients did not have a perioperative myocardial infarction, although the reason for the development of these Q waves is not clear. Transient metabolic abnormalities, which have been shown to cause transient Q waves in patients undergoing cardiopulmonary bypass, cannot be blamed because the Q waves in our patients persisted until hospital discharge. None of these patients had an old inferior infarction that might have been unmasked by improved anterior wall perfusion, as suggested by Bassan et al.

In patient 6, anterior wall motion improved postoperatively, but inferior wall motion was normal both before and after operation.

Myocardial scintigraphy with technetium-99m stannous pyrophosphate was introduced into clinical practice in 1974 by Willerson et al. and has been shown to be a sensitive and specific technique for diagnosis of acute myocardial infarction, although false-positive and false-negative studies do occur. Platt et al. examined the use of pyrophosphate myocardial scintigraphy in 77 patients undergoing coronary bypass surgery and found that two and one-half times as many patients had positive pyrophosphate scintigrams postoperatively as had new Q waves. Most of the patients with positive pyrophosphate scintigrams without new Q waves had a 2+ diffuse pattern and were thought to have had nontransmural infarctions. Kraus et al. performed postoperative pyrophosphate myocardial scintigrams in 51 patients undergoing coronary bypass surgery. Positive postoperative pyrophosphate scintigrams were found in 10 patients, eight of whom had new Q waves. Unfortunately, only two of these 10 patients had preoperative scintigrams, and the possibility that these positive postoperative scintigrams represented persistently positive scintigrams rather than newly positive scintigrams cannot be excluded. One patient with a newly positive postoperative scintigram was shown to have a new area of wall motion abnormality on left ventriculography performed before hospital discharge. In the studies of Platt et al. and Kraus et al., all patients with new Q waves had positive pyrophosphate scintigrams. Righetti et al. performed preoperative and postoperative pyrophosphate scintigrams in 41 patients who underwent coronary bypass surgery. Ten patients had newly positive postoperative pyrophosphate scintigrams; five had new Q waves and five had “ischemic” ST-T-wave changes. One patient with new Q waves postoperatively had a negative pyrophosphate scintigram. Howe et al. studied 70 patients who underwent coronary bypass surgery. Only 31 of these patients had preoperative scintigrams, but five had abnormal preoperative scans, a finding that emphasizes the need for both preoperative and postoperative scintigrams if this technique is to be used properly to detect perioperative myocardial damage. Fourteen patients (20%) had positive postoperative scintigrams, 10 of whom had new Q waves and four of whom did not. In addition, two other patients with new Q waves had normal postoperative scintigrams.

We found positive postoperative pyrophosphate scintigrams in seven of 58 patients undergoing elective coronary bypass surgery. All seven patients had positive CK-MB and new wall motion abnormalities on postoperative ventriculography that corresponded to the area of pyrophosphate uptake. Three patients with positive postoperative pyrophosphate scintigrams did not have new Q waves on postoperative electrocardiography. All three manifested definite new areas of wall motion abnormality on postoperative ventriculography and a positive CK-MB. These patients are examples of perioperative nontransmural infarction that can only be diagnosed by the presence of a positive postoperative pyrophosphate scintigram and positive CK-MB.

Patient 5, who had a definite postoperative inferior myocardial infarction by electrocardiography and ventriculography, had a false-negative pyrophosphate scintigram. In this patient, evidence of infarction was present on an ECG obtained immediately after sur-
surgery, and scintigraphy was performed approximately 72 hours after surgery. The reason for this false-negative scintigram is unknown. False-negative scintigrams are more likely to occur in patients with inferior than anterior infarctions, because the detector tends to see inferior infarctions on edge rather than end face. Falkoff et al. suggested that images that are negative at 72 hours in patients with acute myocardial infarction may be positive at 96 hours. Unfortunately, a later scintigram was not obtained in this patient.

No false-positive scintigrams were found in our study. This high specificity is probably related to the following points: (1) We were careful to differentiate between blood pool activity and diffuse left ventricular uptake, the pattern of pyrophosphate uptake most commonly associated with false-positive results. (2) We were careful to differentiate between rib uptake due to intraoperative trauma and localized myocardial uptake (fig. 5). (3) All patients had preoperative scintigrams available for comparison with postoperative studies, and only areas of new postoperative uptake were considered positive. Thus, areas of persistent positivity that could cause false-positive results in patients with previous infarction were correctly identified.

We conclude that pyrophosphate myocardial scintigraphy is a useful technique for detecting perioperative myocardial infarction. However, to ensure the highest possible sensitivity and specificity, this procedure must be performed properly. All patients should have preoperative scintigrams with which postoperative scintigrams can be compared. Postoperative imaging should optimally be done 48-72 hours and certainly not later than 5 days after surgery. Serial scanning may be necessary in some patients to detect perioperative infarction.

Sixteen patients (28%) in this series had positive CK-MB, including all patients with either positive pyrophosphate scintigrams or a new wall motion abnormality. This is a much lower incidence of positive CK-MB in patients undergoing coronary bypass surgery than in other series and may be related to sampling frequency and the sensitivity of the method used to detect CK-MB in our study, but probably does not represent a true improvement in the specificity of this test for the diagnosis of postoperative myocardial infarction. Eight of the 16 patients with positive CK-MB had other laboratory evidence of myocardial infarction, and eight patients had positive CK-MB as the sole laboratory manifestation of perioperative myocardial infarction (table 1). None of eight patients with only a positive CK-MB had new wall motion abnormalities on postoperative ventriculography, but in three (patients 13-15), left ventricular ejection fraction decreased by 0.09-0.14. These three patients might have suffered diffuse subendocardial damage that did not produce a new wall motion abnormality or new Q waves on postoperative electrocardiography. Because the sensitivity of pyrophosphate myocardial scintigraphy in detecting subendocardial myocardial damage has been questioned, the negative pyrophosphate scintigrams in these patients do not completely exclude this possibility.

### Clinical Implications

We have shown that, for the detection of perioperative myocardial infarction, a newly positive pyrophosphate myocardial scintigram is more sensitive and more specific than the development of new Q waves on electrocardiography and more specific than the presence of CK-MB in the serum. However, all three tests should be used to maximize sensitivity and specificity for diagnosing perioperative myocardial infarction. Table 2 shows the eight possible permutations of positive and negative results using these three diagnostic tests. Use of this schema assumes that pyrophosphate scintigrams are obtained both preoperatively and within 5 days postoperatively and, further, that CK-MB is determined immediately after operation and daily for 3 days. A diagnosis of a definite myocardial infarction should be made if all three tests are abnormal (pattern 1) or if the pyrophosphate scan is abnormal with either new Q

### Table 2. Interpretation of Multiple Testing Results

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<thead>
<tr>
<th>Pattern</th>
<th>PYP</th>
<th>CK-MB</th>
<th>Q-ECG</th>
<th>AMI</th>
<th>Comments</th>
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<tr>
<td>1</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Definite</td>
<td>Transmural infarction</td>
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<tr>
<td>2</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>Definite</td>
<td>CK-MB (negative) because of infrequent sampling</td>
</tr>
<tr>
<td>3</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>Definite</td>
<td>Nontransmural infarction</td>
</tr>
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<td>+</td>
<td>Probable</td>
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<td>False-positive PYP</td>
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<tr>
<td>6</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>No</td>
<td>False-positive CK-MB; small nontransmural AMI possible</td>
</tr>
<tr>
<td>7</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>No</td>
<td>False-positive ECG; usually inferior AMI</td>
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<tr>
<td>8</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>No</td>
<td>None</td>
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</tbody>
</table>

Abbreviations: PYP = pyrophosphate myocardial scintigram; Q-ECG = new Q waves on postoperative ECG; AMI = acute myocardial infarction.
waves on electrocardiography (pattern 2) or a positive CK-MB (pattern 3). We would make the diagnosis of probable myocardial infarction in the presence of new Q waves plus a positive CK-MB combined with a negative pyrophosphate scintigram (pattern 4). Because both CK-MB and electrocardiography have significant false-positive rates, some of these patients will not have had an infarction. If only one test is positive or if no tests are positive, we would not consider an infarction to have occurred (patterns 5–8).

Conclusions

In 58 patients undergoing coronary bypass surgery, we found a newly positive postoperative pyrophosphate scintigram to be a sensitive and specific diagnostic tool for detecting perioperative myocardial infarction of sufficient magnitude to produce new wall motion abnormalities. The development of new postoperative Q waves is both nonspecific (one-half of the patients with new Q waves had no other evidence of acute myocardial infarction) and insensitive (three patients with definite perioperative infarction did not manifest new Q waves on postoperative electrocardiography). The detection of CK-MB in the serum of postoperative coronary bypass patients may be the most sensitive test for the detection of perioperative infarction, but the poor specificity of this test renders it useless as an isolated finding. Because of its high sensitivity, however, the absence of CK-MB in the serum virtually excludes significant perioperative myocardial damage.

References

31. Wilson FN, Johnson FD, Hill IGW: The form of the electrocardiogram in experimental myocardial infarction. IV. Additional observations on the later effects produced by ligation of the anterior descending branch of the left coronary artery. Am
Body Surface Distributions of Repolarization Forces During Acute Myocardial Infarction

I. Isopotential and Isoarea Mapping

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SUMMARY Although ST-segment abnormalities during acute myocardial infarction are clinically important, the total thoracic distribution of these repolarization potentials has not been reported. To provide this information, 24 patients with acute myocardial infarction were studied. Isopotential body surface maps were constructed from potentials sensed by 150 anterior and posterior electrodes. Patterns from 12 patients with anterior lesions demonstrated the appearance of repolarization potentials 21.3 ± 4.6 msec before the end of the QRS complex. During the ST segment, potential distributions were characterized by a single anterior maximum that remained fixed in location but increased in intensity as repolarization progressed. Distributions in the remaining subjects with inferior lesions were analogously characterized by (1) the onset of repolarization 34.6 ± 12.4 msec before termination of the QRS complex and (2) a single anterior minimum located on the left anterior superior thorax, with positive potentials distributed around the lower thoracic margins. These data suggest that electrocardiographic changes after acute myocardial infarction include (1) marked overlap between activation and recovery patterns and (2) isopotential surface patterns with relatively simple topographic configurations, such as expected of a single-dipole equivalent cardiac generator.

THE ELECTROCARDIOGRAPHIC ST segment after myocardial infarction is significant for three reasons. First, it is the pattern during this period that may identify the acute phase of the disorder.1-3 Second, the distribution of abnormalities in the various ECG leads serves to approximate the cardiac locus of the ischemic damage.4 Third, the magnitude of the ST-segment deviation has been proposed as a measure of the extent of the ensuing myocardial infarction.8-9

Methods of investigating these abnormalities include standard electrocardiography,9 vectorcardiography10 and, most recently, multielectrode grids placed upon the left precordial body surface.6-8 Each approach assumes that all relevant electrocardiographic information is sampled by the recording system. In each case, however, deficiencies may be identified. Both standard and multilead precordial systems depend on topographic proximity to the myocardial lesion and to the bulk of the generated electrical field. All lesion loci could not be expected to underlie such fixed location systems. Vectorcardiography, though relatively location-independent, is primarily sensitive to forces generated by a single-current dipole; it has been documented that nondipolar forces, such as generated by greater than one

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