Anesthesiology
71:818–826, 1989

Postoperative Myocardial Infarction Documented by Technetium Pyrophosphate Scan Using Single-Photon Emission Computed Tomography: Significance of Intraoperative Myocardial Ischemia and Hemodynamic Control

Davy C. H. Cheng, M.Sc., M.D., F.R.C.P.C., Frances Chung, M.D., F.R.C.P.C.,†*
Robert J. Burns, M.D., F.R.C.P.C.,‡ Patricia L. Houston, M.D., F.R.C.P.C.,§
Christopher M. Feindel, M.D., F.R.C.S.C.¶

The aim of this prospective study was to document postoperative myocardial infarction (PMI) by technetium pyrophosphate scan using single-photon emission computed tomography (TcPPi-SPECT) in 28 patients undergoing elective coronary bypass grafting (CABG). The relationships of intraoperative electrocardiographic myocardial ischemia, hemodynamic responses, and pharmacological requirements to this incidence of PMI were correlated. Radionuclide cardioangiography and TcPPi-SPECT were performed 24 h preoperatively and 48 h postoperatively. A standard high-dose fentanyl anesthetic protocol was used. Twenty-five percent of elective CABG patients were complicated with PMI, as documented by TcPPi-SPECT with an infarcted mass of 38.0 ± 5.5 g. No significant difference in demographic, preoperative right and left ventricular function, number of coronary vessels grafted, or aortic cross-clamp time was observed between the PMI and non-PMI groups. The distribution of patients using preoperative β-adrenergic blocking drugs or calcium channel blocking drugs was found to have no correlation with the outcome of PMI. As well, no significant differences in hemodynamic changes or pharmacological requirements were observed in the PMI and non-PMI groups during prebypass or postbypass periods, indicating careful intraoperative control of hemodynamic indices did not prevent the outcome of PMI in these patients. However, the incidence of prebypass ischemia was 39.3% and significantly correlated with the outcome of positive TcPPi-SPECT, denoting a 3.9-fold increased risk of developing PMI. Prebypass ischemic changes in leads II and V_5 were shown to correlate with increased CPK-MB release ($P < 0.05$) and tends to occur more frequently with lateral myocardial infarction. No association was found between patients with or without prebypass ischemia on intraoperative hemodynamic changes, drug requirements, or postoperative ventricular function. There was no correlation between postbypass ischemia and positive TcPPi-SPECT. (Key words: Anesthesia: cardiac. Heart, ischemia: intraoperative. Measurement technique, myocardial infarction: technetium pyrophosphate-SPECT. Monitoring: electrocardiography. Surgery: cardiac.)

This article is accompanied by an editorial. Please see: Hurford WE, Lowenstein E, Strauss HW: Are all myocardial infarctions alike? ANESTHESIOLOGY 71: 815–817, 1989.

* Lecturer in Anesthesia, Department of Anesthesia.
† Associate Professor of Anesthesia, Department of Anesthesia.
‡ Assistant Professor of Anesthesia, Department of Anesthesia.
§ Assistant Professor of Nuclear Cardiology.
¶ Associate Professor of Cardiovascular Surgery.

Received from the Departments of Anesthesia, Nuclear Cardiology, and Cardiovascular Surgery, Toronto Western Division, Toronto Hospital, 399 Bathurst Street, Toronto, Ontario M5T 2S8, Canada. Accepted for publication July 29, 1989. Supported in part by a Physicians' Services Incorporated Grant and Grant AN-410 from the Heart and Stroke Foundation of Toronto. Presented in part at the Annual Meeting of the American Society of Anesthesiologists, Atlanta, Georgia, October 1987.

Address reprint requests to Dr. D. C. H. Cheng.

IN PATIENTS UNDERGOING CABG, postoperative myocardial infarction (PMI) is a serious event with mortality rates varying between 3%[1] and 34%.[2] However, the reported incidence of PMI following CABG varies with diagnostic techniques;[1–5] the occurrence of PMI as diagnosed by Q-wave change, cardiac enzymes, and/or nuclear scan ranged from 2.8%[3] to 31%.[4] Prebypass electrocardiographically determined ischemia has been shown to be associated with an increased risk of PMI.[5] This relationship is based on the outcome study of large transmural myocardial infarction (MI) using the insensitive determinants of both ECG change and high CPK-MB release (> 80 U/l); however, it may obscure the relationship in more subtle outcomes of small or subendocardial MI. Quantitation and localization of small MIs including nontransmural infarction by technetium pyrophosphate (TcPPi) uptake is now possible using single-photon emission computed tomography (SPECT).[6,7]

The primary aim of this prospective study was to document PMI in detail using first pass radionuclide angiocardiography (RNA) and TcPPi-SPECT on patients undergoing CABG. A secondary aim was to correlate the significance of intraoperative electrocardiographically determined myocardial ischemia, hemodynamic responses, and pharmacological requirements with this incidence of PMI.

Materials and Methods

The protocol was approved by the University of Toronto Human Ethical Committee. Informed consent was obtained from 30 patients with stable angina and good ventricular function undergoing elective CABG. These 30 patients in this study represent a subset of a larger group in whom TcPPi-SPECT was used to determine the incidence of acute myocardial infarction in patients undergoing CABG.[6] Exclusion criteria included unstable

angina, previous CABG, associated procedures, (*e.g.*, ventricular aneurysm resection), and an electrocardiogram that prevented the diagnosis of ischemia, (*e.g.*, LBBB, digoxin treatment). All data related to perioperative characteristics, electrocardiographic, ventriculographic, and TcPPi-SPECT interpretation were collected and analyzed by independent investigators who had no direct role in the patient's care. All cardiac medication was continued until the time of surgery.

Anesthetic, Cardiopulmonary Bypass and Surgical Techniques

All the patients received diazepam 0.15 mg/kg per os 2 h before surgery; morphine 0.15 mg/kg, and perphenazine 0.07 mg/kg intramuscularly 1 h before surgery. In the operating room, i.v., radial, and pulmonary arterial catheters were inserted under local infiltration and during oxygen supplementation. Fentanyl, 50 μg/kg was used for induction of anesthesia and tracheal intubation, and a total dose of approximately 100 μg/kg was used for surgery. Pancuronium 0.1 mg/kg was used to facilitate tracheal intubation and for maintenance of muscle relaxation. Anesthesia was supplemented with 1–5 mg diazepam. The lungs of all patients were ventilated with 100% oxygen.

Hemodynamic parameters were measured and ECG tracings of lead II and V_5 were recorded (Hewlett Packard, HP® 78534C Monitor/Terminal, with multichannel recording and dual ECG with independent controls—Diagnostic 0.05 to 100 Hz) for 2 min at each of the following: preinduction as baseline control (CONT); during the prebypass period at 1 min after induction (IND), tracheal intubation (INT), skin incision (INC), sternotomy (STERN), and aortic cannulation (CANNU); and again during the postbypass period at 1 minute after discontinuing CPB (PUMP), protamine administration (PROTA), pericardial closure (PERIC), sternal wiring (WIRE), and skin closure (CLOSE). Thermodilution cardiac output measurements were averaged in triplicate using a Gould Laboratory® cardiac output computer and injections of 10 ml of 5% dextrose in water. Heart rate (HR) and blood pressure (BP) were maintained within 20% of the average ward value. For increased BP, additional fentanyl bolus 0.5–1 mg with or without nitroglycerin infusion was used. For decreased BP, volume titrated to a pulmonary capillary wedge pressure (PCWP) of 10–14 mmHg and phenylnephrine infusion was given, if necessary. For increased HR ≥ 90 bpm, incremental iv propranolol 1 mg was used. The electrocardiogram was examined retrospectively for evidence of myocardial ischemia by two independent investigators (blinded to outcomes of study). The ST-segment was evaluated with respect to the PQ junction at a point 80 ms following the S-wave nadir. Myocardial ischemia was defined as 1 mm or more of downsloping or horizontal ST-segment depression, greater than 2 mm of upsloping ST-segment depression, or ST-segment elevation greater than 1 mm.

Following anticoagulation with heparin, cardiopulmonary bypass using a membrane oxygenator was instituted, and patients' temperature was decreased to 28° C. A single two-stage right atrial cannula and an ascending aortic arterial return cannula were employed. The heart was arrested with infusion of 1,000 ml of cold blood (9° C) hyperkalemic cardioplegic solution into the aortic root. Additional doses of 300 ml were given every 20 min. All proximal and distal anastomoses were performed during a single aortic cross clamp period.

Postoperatively, patients' lungs were ventilated for 16–20 h in the Intensive Care Unit. Cardiac filling pressures and systolic BP were maintained between 8–14 mmHg and 100–120 mmHg, respectively. Serial 12 leads ECG tracings were recorded daily in the perioperative period. Quantitative serum CPK-MB determinations were performed preoperatively and every 6 h postoperatively for the first 48 h with a radioimmunoassay technique.

Preoperative Studies

On the day before surgery, subjects underwent first-pass radionuclide ventriculography and TcPPi-SPECT. Seven hundred and forty MBq (20 mCi) of TcPPi were injected as a bolus into an antecubital vein followed by a 20-ml bolus saline flush. Right anterior oblique (30 degree) images were acquired in a list mode with ECG gating using a large field-of-view gamma camera (Elscint Apex® 415) with a general-purpose parallel-hole collimator. Images were acquired for 30 s at 25 images per s.

Four hours after injection, patients underwent SPECT imaging. Sixty images were acquired over 360 degrees in 64 × 64 matrices. Speed of rotation was varied to insure a minimum of 7,000 K total counts, and were normalized for motor speed, isotope decay, camera face sensitivity, and vertical (ventilatory) motion.

Postoperative Imaging

Forty-eight hours after surgery, first-pass radionuclide ventriculography and TcPPi-SPECT were repeated according to the preoperative protocol. Two patients whose conditions were considered unsuitable for departure from the Cardiovascular Intensive Care Unit were removed from the study.

Scintigraphic Analysis

Left and right ventricular ejection fractions were determined after visual inspection of first-pass images in cine format and manual construction of left and right ventricular regions of interest, using background-corrected end-systolic and end-diastolic ventricular counts averaged over three cardiac cycles during which left and right ventricular activity (respectively) were maximal.

Transaxial tomograms were reconstructed in one-pixel thick slices by filtered backprojection using a Hanning Filter.® Areas of abnormal myocardial uptake were identified by the consensus of two blinded observers. Myocardial infarct mass was calculated using an automated computer algorithm which identified the "hottest" voxel within the three-dimensional infarct region, and determined the number of voxels containing at least 70% of this activity. Myocardial infarct mass was calculated as this number of voxels multiplied by voxel volume (0.34 ml) and by the specific gravity of cardiac muscle (1.05 g/ml).

Statistical Analysis

Comparison of data between patients with positive and negative TcPPi-SPECT, as well as patients with perioperative ischemia and without ischemia, was done by student *t* tests for two independent samples with correction of equal variance when appropriate. Hemodynamic parameters and CPK-MB levels were analyzed using a repeated measure analysis of variance. *Post hoc* analysis was accomplished using contrast and simple *t* test. Chi-square analysis without Yate's correction for continuity was applied to analyze the incidence of perioperative ischemia to PMI, as appropriate. $P < 0.05$ was considered statistically significant. All data are given as mean ± SEM.

Results

Scintigraphy

Thirty patients were studied. Two patients were excluded from the study: one had suffered perioperative cerebral vascular accident and eventually died; the other required an intra-aortic balloon pump postoperatively and could not be moved from the ICU for follow-up scan. One patient had positive preoperative TcPPi-SPECT with extension of uptake postoperatively. Six other patients had abnormal TcPPi-SPECT postoperatively. Thus, 7/28 patients (25%) suffered PMI. Their average irreversible myocardial necrotic mass was 38.0 ± 5.5 g and ranged from 22–51 g. Example of positive TcPPi-SPECT in a patient is presented in figure 1.

Patients with positive postoperative TcPPi-SPECT did not differ from patients with negative postoperative TcPPi-SPECT in regards to age, basal surface area (BSA), ASA physical status, preoperative left ventricular end-diastolic pressure (LVEDP), preoperative right and left ventricular ejection fractions (EF), number of coronary vessels grafted, and anoxic aortic cross-clamp time (table 1). The distribution of preoperative cardiac medications taken by patients was found to have no correlation with the outcome of positive postoperative TcPPi-SPECT (table 2).

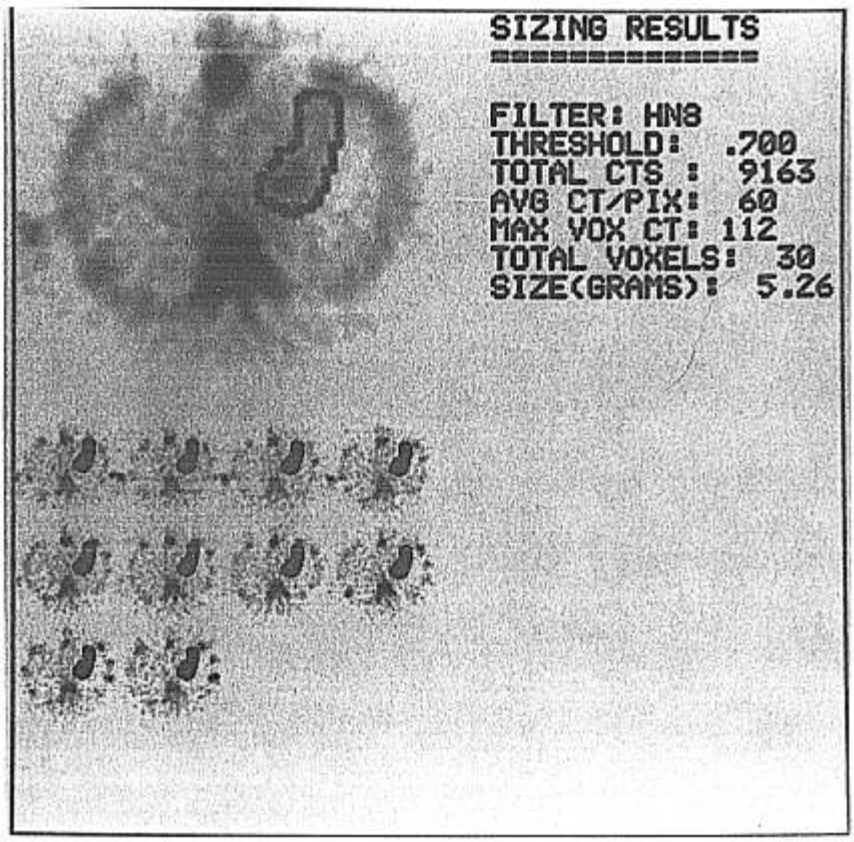

FIG. 1. Example of positive Technetium Pyrophosphate imaging using single-photon emission computed tomography (TcPPi-SPECT). The "hot spot" represented uptake of TcPPi by damaged myocardium without superimposed blood pool and bone activity. The smaller transaxial tomograms were reconstructed in one-pixel thick slices by filtered back projection using a Hanning Filter. Myocardial infarct mass was calculated using an automated computer algorithm which identified the "hottest" voxel within the three-dimensional infarct region and determined the number of voxels containing at least 70% of this activity.

TABLE 1. Comparison of Perioperative Characteristics of Patients with Positive and Negative Postoperative TcPPi-SPECT

Characteristics	Positive (n = 7)	Negative (n = 21)
Age (yr)	53.3 ± 2.8	56.9 ± 2.0
Sex	4 men, 3 women	21 men
BSA (kg/m^2)	1.87 ± 0.09	1.95 ± 0.03
LVEDP (mmHg)	20.3 ± 4.5	14.4 ± 1.4
Pre-RVEF (%)	50.4 ± 0.05	53.3 ± 0.03
Pre-LVEF (%)	59.4 ± 0.06	59.9 ± 0.03
No. of Grafts	4.0 ± 0.6	3.3 ± 0.2
Cross-clamp (min)	57.0 ± 8.5	48.3 ± 3.3

No significant difference between the two groups (mean ± SEM).
BSA = Basal Surface Area.

TABLE 2. Distribution of Preoperative Cardiac Medications in Patients with Positive and Negative Postoperative TcPPi-SPECT

Postoperative TCPPi-SPECT	BB	CEB	BB-CEB	Vasodilator Only
Positive	0	1	5	1
Negative	4	3	14	0

BB = β-adrenergic blocking drugs.
CEB = calcium channel blocking drugs.

Only one patient with positive postoperative TcPPi-SPECT developed new ECG Q-wave, giving the overall incidence of PMI as 3.6% using electrocardiographic criteria.

Total CPK level peaked over 2,000 U/l at 24 h postoperative in both groups. However, serial CPK-MB fraction was significantly higher from 8–24 h in patients with positive postoperative TcPPi-SPECT, ranging from 20–50 U/l.

PREBYPASS MYOCARDIAL ISCHEMIA

Compared with the preinduction baseline control ECG leads II and V_5, the incidence of ischemic ECG changes during prebypass period (post: induction, intubation, skin incision, sternotomy, and aortic cannulation) was 39.3% (11 of 28 patients) with most occurring poststernotomy (63.6%) and postaortic cannulation (63.6%). The sensitivity (five of seven patients) and specificity (15 of 21 patients) of prebypass ischemia to PMI were both 71.4%. The relationship between prebypass ischemia and incidence of PMI showed that five of 11 patients with prebypass ischemia (45.4%) developed PMI, whereas two of 17 patients without prebypass ischemia (11.8%) developed PMI. Therefore, patients who manifested prebypass ischemia had a 3.9-fold increased risk of developing PMI ($P < 0.044$, chi-square).

Data comparison between patients with and without prebypass ischemia showed no significant differences in their preoperative characteristics, aortic cross-clamping time (48.9 ± 5.8 *vs.* 51.5 ± 4.0 min), intraoperative hemodynamic response or drugs used, and postoperative ventricular ejection fraction. However, patients with prebypass ischemia sustained a significant elevated release of CPK-MB level that peaked at 24 h postoperatively (fig. 2).

POSTBYPASS MYOCARDIAL ISCHEMIA

The incidence of ischemic ECG changes postbypass was 32.1% (nine of 28 patients) and most occurred post protamine injection (77.8%). Of the seven PMI patients, two

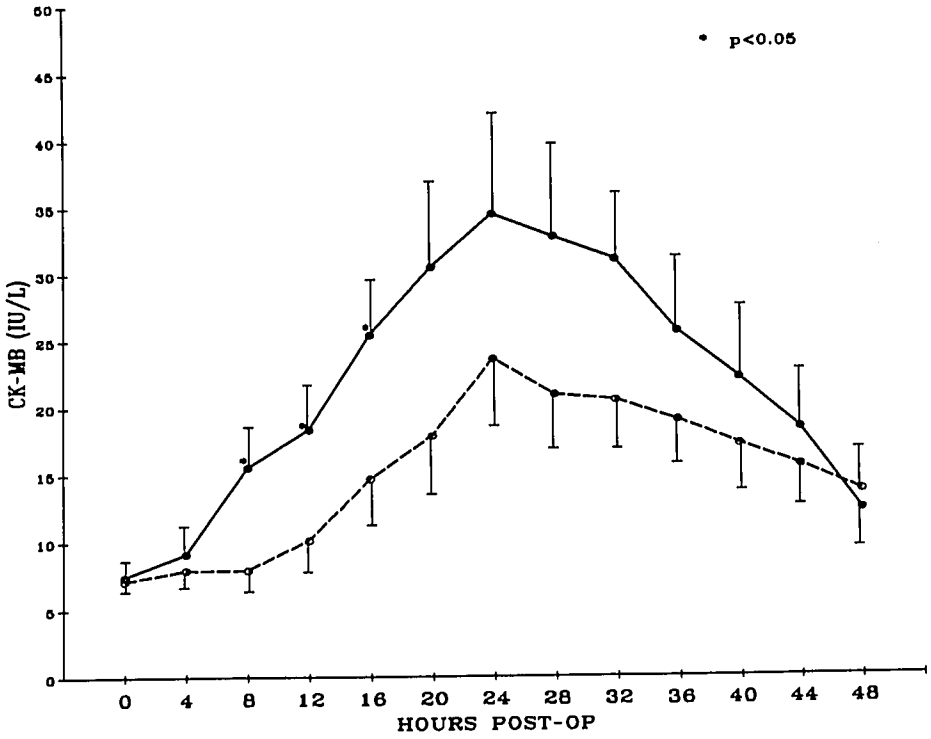

FIG. 2. Comparison of postoperative CPK-MB level between patients with (n = 11) (- - - ● - - -) and without (n = 17) (- - - O - - -) prebypass myocardial ischemia.

manifested postbypass ischemia. The sensitivity and specificity of postbypass ischemia to PMI were 28.6% (two of seven patients) and 66.7% (14 of 21 patients), respectively. There was no relationship between postbypass ischemia and incidence of PMI as only two of nine patients with postbypass ischemia (22.2%) compared with five of 19 patients without postbypass ischemia (26.3%) developed PMI.

Prebypass Ischemia and Location of PMI (Table 3)

The prebypass ischemic changes in leads II and V_5 ECG were shown to occur more frequently with lateral myocardial infarction and not with the inferior or anterior apical infarcted location.

Intraoperative Drugs and Hemodynamics Response

The induction, prebypass, and postbypass interval total dosages of fentanyl and diazepam were not different between the MI and non-MI groups (table 4). No statistically significant hemodynamic changes (HR, MAP, CI, PCWP, SVR) were observed between the MI and non-MI groups during the prebypass (fig. 3) and postbypass periods (fig. 4). The intraoperative requirement of propranolol, nitroglycerin, and nitroprusside was not significantly different (table 4).

Discussion

In this prospective study, seven of the 28 patients (25%) suffered PMI, as documented by the extremely sensitive and specific TcPPi-SPECT imaging.[7,8] These 30 patients in this study represent a subset of a larger group in whom TcPPi-SPECT was used to determine the incidence of acute myocardial infarction in patients undergoing CABG.[8] The reported incidence of PMI following CABG in the literature depends upon diagnostic technique. For example, PMI is reported as low as 2.8%[3] diagnosed by standard electrocardiographic criteria, or as high as 31%[4] using planar TcPPi scintigraphic imaging. There is increasing concern about the use of the insensitive determinants of new Q-waves and CPK-MB > 80 U/l as the diagnostic criteria for PMI. As they document only large transmural MI, many other subtle outcomes such as subendocardial MI may not be detected.[9] On the other hand, TcPPi is selectively adsorbed to tissue calcium stores. Its localization in the myocardium is directly proportional to the degree of tissue damage and the residual blood flow to the area. The peak intensity of TcPPi image does not occur until 24–72 h after infarction. The advantage of SPECT over the planar TcPPi imaging is that it enables three-dimensional reconstruction of cardiac images from multiple planar projections for localization and quantitation of myocardial uptake without superimposed blood pool and bone activity.[10]

Our patients with PMI sustained necrosis of significant myocardial mass averaged 35 g, which represents approximately 10% of the weight of a normal-size heart. Late survival was shown to be adversely affected by PMI;[11–13] thus, it may place them at risk of cardiac complications from future stressful situations.[14] Previous studies have shown that size of infarct determined by TcPPi-SPECT imaging correlated well with prognosis.[7] As well, it has been shown that the size of infarction estimated by TcPPi-SPECT correlated well with CPK-MB estimation of infarction size in patients with acute myocardial infarction.[15]

Risk factors attributable to PMI are complex and multifactorial,[16,17] and we were unable to demonstrate any

Table 3. Relationship between Prebypass Ischemia* and Infarct Location in Patients with PMI

Patient		IND	INT	INC	STERN	CANNU	Location
1	V_5	2 mm	—	—	—	—	Lateral
	II	2 mm	—	—	—	—	
2	V_5	—	2 mm	—	—	—	Lateral
	II	2 mm	—	—	—	3 mm	
3	V_5	2 mm	—	—	—	—	Lateral
	II	—	2.5 mm	—	3.5 mm	2 mm	
4	V_5	—	—	—	2 mm	1 mm	Lateral
	II	—	—	—	4 mm	3 mm	
5	V_5	—	—	—	—	—	Anterior
	II	—	—	—	—	—	Apical
6	V_5	—	—	—	—	1 mm	Apical
	II	—	—	—	—	2 mm	
7	V_5	—	—	—	—	—	Inferior
	II	—	—	—	—	—	

* ST depression.
IND = Postinduction.
INT = Postintubation.
INC = Postskin incision.
STERN = Poststernotomy.
CANNU = Postaortal cannulation.

Table 4. Comparison of Intraoperative Medication Requirements in Patients with Positive and Negative Postoperative TcPPi-SPECT

Medications (mg)	Positive TcPPi-SPECT	Negative TcPPi-SPECT
Fentanyl		
(Induction)	3.6 ± 0.4 (n = 7)	3.8 ± 0.2 (n = 21)
(+ prebypass)	5.1 ± 0.2 (n = 7)	5.5 ± 0.2 (n = 21)
(+ postbypass)	6.7 ± 0.4 (n = 7)	6.7 ± 0.2 (n = 21)
Diazepam		
(Induction)	4.2 ± 0.5 (n = 7)	6.3 ± 0.7 (n = 21)
(+ prebypass)	12.9 ± 3.2 (n = 7	12.5 ± 1.4 (n = 21)
(+ postbypass)	17.6 ± 3.2 (n = 7)	16.8 ± 1.4 (n = 21)
Nitroglycerin		
(Induction)	—	1.1 (n = 1)
(+ prebypass)	11.7 ± 5.9 (n = 4)	10.1 ± 2.8 (n = 10)
(+ postbypass)	15.8 ± 6.0 (n = 5)	11.8 ± 3.6 (n = 10)
Nitroprusside		
(postbypass)	10.0 (n = 1)	5.74 ± 3.0 (n = 5)
Propranolol		
(prebypass)	5.0 (n = 1)	2.7 ± 0.9 (n = 4)
(+ postbypass)	5.0 (n = 1)	2.4 ± 0.7 (n = 5)

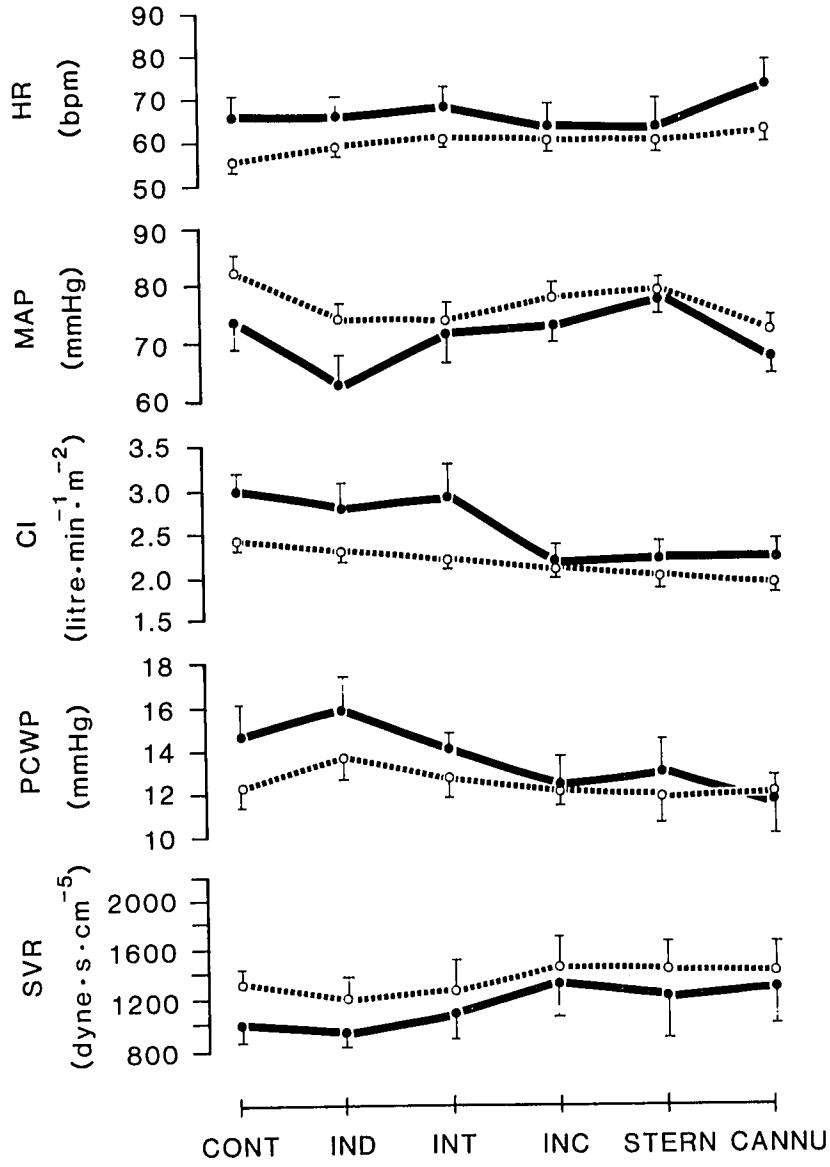

FIG. 3. Comparison of prebypass hemodynamic profiles between patients with PMI (n = 7) (– – – ● – – –) and no MI (n = 21) (– – – O – – –). Hemodynamic changes were not significantly different between the two groups (mean ± SEM).

relationship between age, preoperative clinical status, preoperative cardiac medication, aortic cross-clamp times, standardized anesthetic technique, or intraoperative hemodynamic changes to PMI as documented by TcPPi-SPECT. However, in this study, we found a significant relationship of prebypass myocardial ischemia to PMI. Although Chung *et al.*[18] and Slogoff and Keats[19] suggested that preoperative β-adrenergic blocking drugs may reduce the incidence of myocardial ischemia, we did not show any relationship of preoperative medication to PMI. This may be due to the limitation of a small sample size in our study group.

The incidence of myocardial ischemia during the prebypass period in our study was 39.3%, which is comparable with that in larger series[5,20,21] in patients undergoing CABG (37–55%), as well as in patients with ischemic heart disease who underwent noncardiac surgical procedures (38%).[22] The significance of prebypass ischemia was implicated by its association with an almost fourfold increased risk of developing PMI. These prebypass ischemic episodes occurred most frequently following sternotomy or aortic cannulation, which were usually the most stressful stimulation periods to the myocardium in terms of oxygen supply and demand. No association was found in their preoperative characteristics, intraoperative hemodynamic, or drugs used as predictors of the occurrence of prebypass myocardial ischemia. The importance of prebypass ischemia in our study is further exemplified by its association with elevated CPK-MB release that occurred only when irreversible injury has been induced to the myocardium, peaked at 24 h postoperatively.

One of the limitations in our study is that the incidence

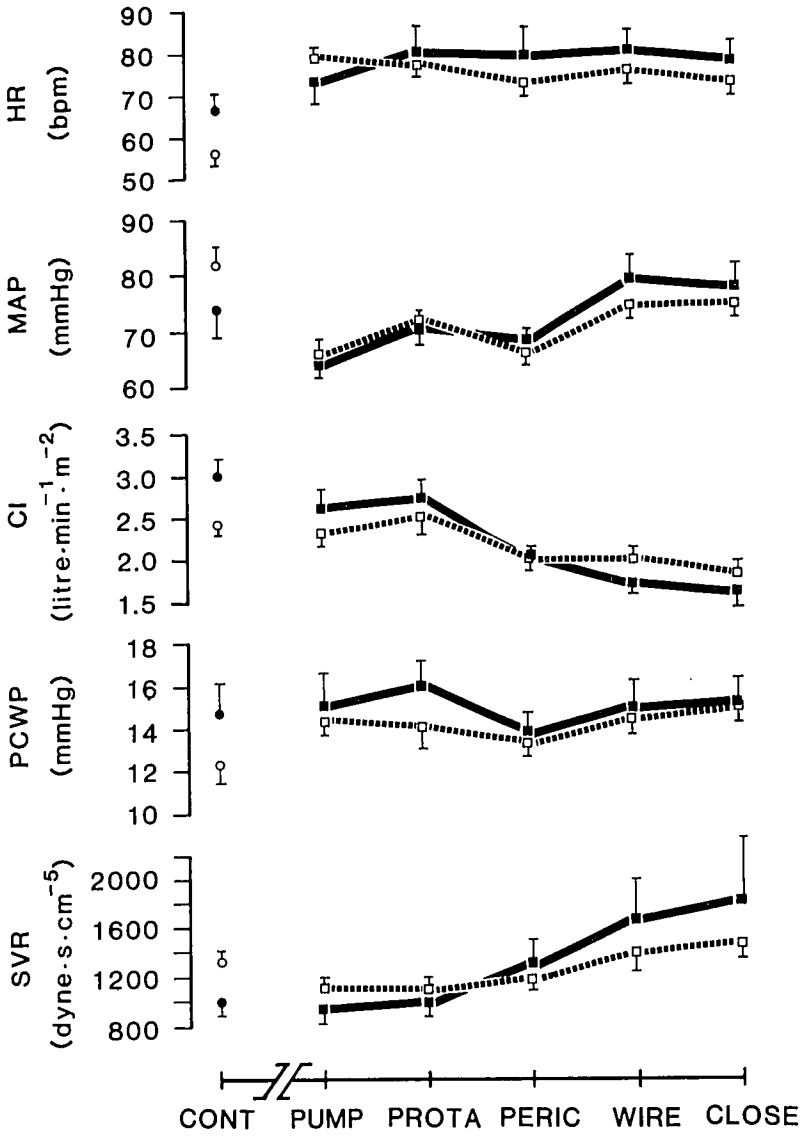

FIG. 4. Comparison of postbypass hemodynamic profiles between patients with PMI (n = 7) (– – – ■ – – –) and no MI (n = 21) (– – – □ – – –). Hemodynamic changes were not significantly different between the two groups (mean ± SEM).

of PMI we have reported would likely be conservatively estimated, as the ECG was recorded intermittently at selected critical intervals. The total duration of monitoring was approximately 22 min. Nonetheless, our results are in agreement with Slogoff and Keats,[21] in that their reported incidence of preinduction and prebypass ischemia were 27% and 34%, respectively. In contrast, using a continuous bipolar leads system (Holter monitor), Knight *et al.*[23] suggested that perioperative ischemia is not specific predictor of PMI. Their reported incidence of ischemia during preinduction was 4%, prebypass was 14%, and post-bypass was 40%. However, their intraoperative anesthesia was not standardized or controlled and their outcome of PMI was 12% by using new Q-wave and CPK-MB level ≥ 80 IU/l. Furthermore, in our study, a standard unipolar precord lead (true V_5) of a multiple-lead ECG system as recommended by Kaplan and King[24] was used for monitoring perioperative ischemia with simultaneous displacement of leads II and V_5. The lead systems have recently been validated by London *et al.*[25] using continuous 12-lead ECG monitoring that combining leads II, V_4 and V_5 will increase the sensitivity of detecting ischemia to 96% in noncardiac surgical patients.

No correlation between postbypass ischemia and PMI was found, suggesting that ischemic changes in the rewarmed heart may not be specific in predicting the outcome for PMI.

Another limitation in this study is that, although hemodynamic monitoring was continuous throughout the operative period, only selected critical intervals were used in the analysis. A direct correlation of HR, MAP, CI, PCWP, and SVR between patients with and without PMI revealed no difference during both prebypass and postbypass periods. PMI was not shown to be preventable,

despite careful control of hemodynamic indices in these patients. In particular, HR was maintained below 90 bpm in our patients, as it has been shown that myocardial ischemic changes were associated with tachycardia and increased MAP during fentanyl-pancuronium anesthesia, which could not be prevented by iv nitroglycerin prophylactically.[20] In addition to the intermittent recording of ischemic changes, the relationship between hemodynamics and ischemia cannot be precisely quantitated; therefore, the belief that hemodynamically unrelated myocardial ischemia may lead to PMI cannot be definitely determined in this study.[21] Our data showed no difference in intraoperative requirement of propranolol, nitroglycerin, and nitroprusside between patients with or without PMI.

In our study, the electrocardiographic incidence of PMI by new Q-wave criteria was 3.6%. This would seriously underestimate those patients with significant PMI for postoperative follow-up management. In addition, the prognosis for PMI diagnosed by CPK-MB release and new Q-wave ECG changes varied from that diagnosed by TcPPi-SPECT.[7,13,26] The TcPPi-SPECT imaging is particularly useful in documenting infarct size as well as location,[6,7,15] which has an important influence on patient morbidity and mortality as it directly affects the ventricular performance. However, the correlation between electrocardiographic leads detecting myocardial ischemia and the exact location of PMI in CABG surgery by TcPPi-SPECT has not been documented. Using Chi-square analysis with trend, our data only showed that intraoperative ischemic changes in leads II and V_5 were associated more frequently with lateral myocardial infarction, and not as often in the detection of inferior or anterior apical infarcted location. This might be attributed to the displacement of the leads when the sternum of the patient was split open during CABG, implying that the standard intraoperative electrocardiographic five-lead system may not be adequate in detecting myocardium at risk other than those in the lateral position during CABG. With the significance of perioperative ischemia, other means of early ischemia detection such as transoesophageal echocardiography, cardiokymogram, regional thallium perfusion scan, or cardiac metabolic studies would be important to identify those myocardium at risk in oxygen imbalance during CABG.

In summary, with the technetium pyrophosphate scan using single-photon emission computed tomography (TcPPi-SPECT) as the diagnostic criteria for PMI, 25% of the patients undergoing elective CABG were documented to have PMI with an average myocardial necrotic mass of 38 g. No correlation was found between preoperative cardiac medication and the outcome of PMI. However, prebypass myocardial ischemia was significantly associated with a 3.9-fold increased risk of PMI. Patients with prebypass ischemia sustained a significant increase in the release of CPK-MB postoperatively denoting severe myocardial tissue damages. In addition, electrocardiographic leads II and V_5 ECG ischemic changes occurred more frequently in detecting lateral myocardial infarction by TcPPi-SPECT. Careful intraoperative control of hemodynamic parameters did not prevent PMI in the patients studied, and the use of electrocardiographic new Q-wave changes would seriously underestimate the incidence of PMI in CABG patients.

The authors wish to thank Miss C. Drane for her secretarial assistance, the University of Toronto Clinical Statistical unit for their statistical assistance, and Dr. J. M. R. Campbell, Chief, Department of Anaesthesia, Toronto Western Hospital, for his continuous support.

References

1. Gray RJ, Matloff JM, Conklin CM, Granz W, Charuzi Y, Wolfstein R, Swann HJC: Perioperative myocardial infarction: Late clinical course after coronary artery bypass surgery. Circulation 66: 1185–1189, 1982
2. Eugene J, Ott RA, Piters KM, Stemmer EA: Operative risk factors associated with unstable angina pectoris. Arch Surg 120:279–282, 1985
3. Miller DC, Stinson EB, Oyer PE, Jamieson SW, Mitchell RS, Reitz BA, Baumgartner WA, Shumway NE: Discriminant analysis of the changing risks of coronary artery operations: 1971–1979. J Thorac Cardiovasc Surg 85:197–213, 1983
4. Platt MR, Parkey RW, Willerson JT, Bonte FJ, Shapiro W, Sugg WL: Technetium stannous pyrophosphate myocardial scintigrams in the recognition of myocardial infarction in patients undergoing coronary artery revascularization. Ann Thorac Surg 21:311–317, 1976
5. Slogoff S, Keats AS: Does perioperative myocardial ischemia lead to postoperative myocardial infarction? ANESTHESIOLOGY 62: 107–114, 1985
6. Burns RJ, Gladstone PJ, Tremblay PC, Feindel CM, Salter DR, Lipton IH, Ogilvie RR, David TE: Myocardial infarction determined by technetium-99m pyrophosphate single-photon tomography complicating elective coronary artery bypass grafting for angina pectoris. Am J Cardiol 63:1429–1434, 1989
7. Corbett JR, Lewis M, Willerson JT, Nicod PH, Husley RL, Simon T, Rude RE, Henderson E, Parkey R, Rellas JS, Buja LM, Sokolov JJ, Lewis SE: 99m Tc-Pyrophosphate imaging in patients with acute myocardial infarction: Comparison of planar imaging with single-photon tomography with and without blood pool overlay. Circulation 69:1120–1128, 1984
8. Holman BL, Goldhaber SZ, Kirsch C, Polak JF, Friedman BJ, English RJ, Wynne J: Measurement of infarct size using single photon emission computed tomography and technetium-99m pyrophosphate: A description of the method and comparison with patient prognosis. Am J Cardiol 50:503–511, 1982
9. Mangano DT: Anesthetics, coronary artery disease, and outcome: Unresolved controversies. ANESTHESIOLOGY 70:175–178, 1989
10. Berger HJ, Zaret BL: Nuclear cardiology. N Engl J Med 305: 799–807, 1981
11. Arnold M, Wathanacharoen S, Reed WA, Killen DA, Crockett J, King JT, McCallister BD, Bell H: Five-year follow-up of patients undergoing coronary artery bypass. Ann Thorac Surg 27:225–229, 1979
12. Namay DL, Hammermeister KE, Zia MS, DeRouen TA, Dodge HT, Namay K: Effect of perioperative myocardial infarction

on late survival in patients undergoing coronary artery bypass surgery. Circulation 65:1066–1071, 1982
13. Val PG, Pelletier LC, Hernandez MG, Jais JM, Chaitman BR, Dupras G, Solymoss BC: Diagnostic criteria and prognosis of perioperative myocardial infarction following coronary bypass. J Thorac Cardiovasc Surg 86:878–886, 1983
14. Khambatta HJ, Sonntag H, Larsen R, Stephan H, Stone JG, Kettler D: Are post coronary artery bypass patients still at risk? (abstract) ANESTHESIOLOGY 67:A598, 1987
15. Jansen DE, Corbett JR, Wolfe CL, Lewis SE, Gabliani G, Fieipchuk N, Redish G, Parkey R, Buja M, Jaffe AS, Rude R, Sorbel BE, Willerson JT: Quantification of myocardial infarction: A comparison of single photon-emission computed tomography with pyrophosphate to serial plasma MB-creatine kinase measurements. Circulation 72:327–333, 1985
16. Burton JR, FitzGibbon GM, Keon WJ, Leach AJ: Perioperative myocardial infarction complicating coronary bypass—Clinical and angiographic correlations and prognosis. J Thorac Cardiovasc Surg 82:758–764, 1981
17. Baur HR, Peterson TA, Arnar O, Gannon PG, Gobel FL: Predictors of perioperative myocardial infarction in coronary artery operation. Ann Thorac Surg 31:36–44, 1981
18. Chung F, Houston PL, Cheng DCH, Lavelle PA, McDonald N, Burns RJ, David TE: Calcium channel blockade does not offer adequate protection from perioperative myocardial ischemia. ANESTHESIOLOGY 69:343–347, 1988
19. Slogoff S and Keats AS: Does chronic treatment with calcium entry blocking drugs reduce perioperative myocardial ischemia? ANESTHESIOLOGY 68:676–680, 1988
20. Thomson IR, Mutch WAC, Culligan JD: Failure of intravenous nitroglycerin to prevent intraoperative myocardial ischemia during fentanyl-pancuronium anesthesia. ANESTHESIOLOGY 61: 385–393, 1984
21. Slogoff S, Keats AS: Further observations on perioperative myocardial ischemia. ANESTHESIOLOGY 65:539–542, 1986
22. Roy WL, Edelist G, Gilbert B: Myocardial ischemia during noncardiac surgical procedures in patients with coronary-artery disease. ANESTHESIOLOGY 51:393–397, 1979
23. Knight AA, Hollenberg M, London MJ, Tubau J, Verrier E, Browner W, Mangano DT and The S.P.I. Research Group: Perioperative myocardial ischemia: Importance of the preoperative ischemic pattern. ANESTHESIOLOGY 68:681–688, 1988
24. Kaplan JA, King SB: The precordial electrocardiographic leads (V_5) in patients who have coronary artery disease. ANESTHESIOLOGY 45:570–574, 1976
25. London MJ, Hollenberg M, Wong MG, Levenson L, Tubau JF, Browner W, Mangano DT and The S.P.I. Research Group: Intraoperative myocardial ischemia: Localization by continuous 12-lead electrocardiography. ANESTHESIOLOGY 69:232–241, 1988
26. Haagensen R and Steen PA: Perioperative myocardial infarction. Br J Anaesth 61:24–37, 1988