Postoperative haemodynamic and pharmacological responses in patients with positive technetium pyrophosphate single-photon emission computed tomography following CABG

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The aim of this prospective study was to evaluate the postoperative haemodynamic variables and medication requirements in patients with perioperative myocardial infarction (PMI), following elective coronary artery bypass graft (CABG) surgery, as documented by technetium pyrophosphate scintigraphy using single-photon emission computed tomography (TcPPi-SPECT). A high-dose fentanyl anaesthetic technique was applied. Twelve of 58 patients (21%) developed PMI with an infarcted myocardial mass of 35.7 ± 3.9 g. Over the 48 hr postoperative period, patients with positive TcPPi-SPECT (n = 12) did not differ from those with negative TcPPi-SPECT (n = 46) in mean heart rate (below 100 bpm), systolic blood pressure (100–120 mmHg) or central venous pressure (8–16 mmHg). However, patients with positive TcPPi-SPECT had higher pulmonary artery diastolic pressures at 5–8 hr after surgery. No differences were found in the incidence and dosage requirements for postoperative sedative or vasoactive drugs (morphine, diazepam, propranolol, lidocaine, nitroglycerin and nitroprusside) between the two groups. There was no difference in the incidence of dopamine requirement between the groups (positive-scan: 16.7%, negative-scan: 13.0%). However, the dopamine dosage for inotropic support was higher in the positive TcPPi-SPECT group over 24 hr (318.5 ± 125.2 mg vs 71.2 ± 24.7 mg, $P < 0.05$) and 48 hr (869.1 ± 19.0 mg vs 142.3 ± 49.4 mg, $P < 0.001$) periods after surgery. We postulate that careful control of postoperative haemodynamic variables did not prevent but may limit the extent of PMI in elective CABG patients.

Les buts de cette étude prospective étaient d'évaluer les variables hémodynamiques postopératoires et les médicaments requis chez des patients ayant un infarctus du myocarde périopératoire (PMI), après pontage aortocoronarien électif et documenté par une scintigraphie au technitium pyrophosphate utilisant la tomographie computée par émission d'un photon unique (TcPPi-SPECT). Une technique anesthésique à haute dose de fentanyl fut utilisée. Douze des 58 patients (21%) ont développé un PMI avec une masse myocardique infarcisée de 35,7 ± 3,9 g. Au cours d'une période de 48 heures postopératoires, des patients avec un TcPPi-SPECT positif (n = 12) n'ont pas démontré de différence avec ceux qui avaient un TcPPi-SPECT négatif (n = 46) quant à la fréquence (inférieure à 100 bpm), pression artérielle systolique (100–120 mmHg) ou la pression veineuse centrale (8–16 mmHg). Cependant, les patients ayant présenté un TcPPi-SPECT positif avaient des

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pressions diastoliques de l'artère pulmonaire élevées à 5–8 heures après la chirurgie. Aucune différence entre les deux groupes ne fut trouvée dans l'incidence et la nécessité de sédation postopératoire ou dans les drogues vasoactives (morphine, diazépam, propranolol, lidocaïne, nitroglycérine et nitroprussiate). Il n'y avait aucune différence dans l'incidence de la nécessité d'administrer de la dopamine entre les groupes (scan positif: 16,7%, scan négatif: 13,0%). Cependant, le support inotrope avec la dopamine fut significativement plus élevé dans le groupe TcPPi-SPECT positif au cours des 24 heures (318,5 ± 125,2 mg vs 71,2 ± 24,7 mg, P < 0.05) et 48 heures (869,1 ± 19,0 mg vs 142,3 ± 49,4 mg, P < 0.001) après la chirurgie. On postule qu'un contrôle méticuleux des variables hémodynamiques en période postopératoire n'a pas empêché mais aurait pu limiter l'étendue du PMI chez les patients ayant subi un pontage aortocoronarien électif.

It is well known that haemodynamic stability is essential in the anaesthetic management of coronary artery bypass graft (CABG) surgery. A strong correlation between intraoperative tachycardia[1,2] and myocardial ischaemia[1–3] leading to perioperative myocardial infarction (PMI) has been established. Although prebypass myocardial ischaemia is associated with scintigraphic PMI, careful intraoperative control of haemodynamic indices did not prevent such an outcome.[3] Recent studies have begun to focus on the prognostic significance of postoperative course in patients undergoing CABG surgery.[4,5]

The objective of this prospective study was to compare the postoperative haemodynamic control and pharmacological requirements in elective CABG patients with PMI as documented by positive postoperative technetium pyrophosphate-SPECT.

Methods

The protocol was approved by the University of Toronto Human Ethical Committee. Informed consent was obtained from 65 patients with stable angina undergoing elective CABG. Exclusion criteria included unstable angina, previous CABG, associated procedures (e.g., ventricular aneurysm resection) and an ECG that prevented the diagnosis of ischaemia (e.g., LBBB, digoxin treatment). All data related to perioperative characteristics, electrocardiographic, ventriculographic, haemodynamic, pharmacological requirement 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. The TcPPi-SPECT, intraoperative myocardial ischaemia, electrocardiogram Q waves, creatine kinase MB fractions, as well as early and late postoperative radionuclide ventricular ejection fractions on this group of patients were previously reported.[3,6]

Anaesthetic, cardiopulmonary bypass and surgical techniques

All patients received standardized doses of diazepam (0.15 $mg \cdot kg^{-1}$ *po* two hours before surgery); morphine (0.15 $mg \cdot kg^{-1}$) and perphenazine (0.07 $mg \cdot kg^{-1}$) intramuscularly one hour before surgery. Intraoperative monitoring included ECG (leads II and V_5), radial arterial blood pressure, pulmonary artery pressure and pulse oximeter. Anaesthesia was induced with fentanyl 50 $\mu g \cdot kg^{-1}$ and pancuronium 0.1 $mg \cdot kg^{-1}$ for tracheal intubation, and a total dose of 100 $\mu g \cdot kg^{-1}$ fentanyl was used for surgery. Muscle relaxation was maintained with pancuronium. Anaesthesia was supplemented with 1–5 mg diazepam. Haemodynamic variables were maintained within 20% of the average ward value. For increased blood pressure (BP), an additional fentanyl bolus 0.5–1 mg with or without nitroglycerin infusion was given. For decreased BP, volume was titrated to a pulmonary capillary wedge pressure (PCWP) of 10–14 mmHg and phenylnephrine infusion was given, if necessary. For increased HR $\geq$ 90 bpm, incremental *iv* propranolol 1 mg was used.

Following anticoagulation with heparin, cardiopulmonary bypass was performed using a membrane oxygenator and systemic hypothermia at 28–30° C. A single two-stage right atrial cannula and an ascending aortic arterial return cannula were employed. The heart was arrested with 1000 ml of cold (9° C) hyperkalaemic cardioplegic blood into the aortal root. All proximal and distal anastamoses were performed during a single aortic cross-clamp period. After completion of each graft an additional 300 ml of cardioplegic solution was administered into the aortal root. The heart was reperfused for 10–20 min after removal of aortic cross-clamp and weaned from cardiopulmonary bypass.

Postoperative observations

Patients' tracheas remained intubated for 12–18 hr in the Intensive Care Unit. Haematocrit was maintained between 0.25–0.30. Central venous pressures and systolic BP were maintained between 8–16 mmHg and 100–120 mmHg, respectively with the administration of 5% albumin, crystalloid, dopamine (3–10 $\mu g \cdot kg^{-1} \cdot min^{-1}$) and nitroprusside by the intensivists. Propranolol was titrated to treat heart rate greater than 100 bpm, lidocaine was infused to treat ventricular arrhythmias and nitroglycerin was infused to treat ischaemic ECG changes. Haemodynamic patterns, vasoactive drug requirements, analgesic and sedative drugs administered were recorded Q15 min for 1 hr, Q 30 min for 1 hr, then hourly for a total of 48 hr

after operation. Serial 12 lead ECG tracings were recorded daily in the postoperative period.

Scintigraphy protocol

On the day before surgery, patients underwent first-pass radionuclide ventriculography with bolus injection of 925 MBq of Tc-PPi containing less than 1% free pertechnetate into a large anticubital vein. Right anterior oblique (30°) 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 sec at 25 images · sec^{-1}. Right and left ventricular ejection fractions were calculated as previously described.[6] SPECT imaging was performed four hours after first-pass acquisition using the same detector with 360° step-and-shoot acquisition, 3° · $step^{-1}$. The speed of rotation was varied to ensure a minimum of seven million total counts. The projection images were stored in 64 × 64 matrixes and immediately normalized for motor speed, isotope decay and camera face sensitivity. Transaxial tomograms were reconstructed in one-pixel thick slices by filtered back projection using a Hanning Fi-Her®. Areas of abnormal myocardial uptake were identified by the consensus of two independent observers as myocardial infarction. Forty-eight hours after surgery, first-pass radionuclide ventriculography and TCPPi-SPECT were repeated according to the preoperative protocol. Myocardial infarction 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^{-1}).

Statistical analysis

Univariate data comparisons between patients with positive and negative postoperative TcPPi-SPECT were performed using chi-square analysis or unpaired Student's t test. Haemodynamic variables were analyzed by two-way ANOVA with repeated measures to test for differences between positive and negative postoperative TcPPi-SPECT over time and in contrast to postoperative baseline haemodynamics. Differences were considered significant at $P < 0.05$. All data are given as mean ± SEM.

Results

Patient characteristics and scintigraphy

Fifty-eight of 65 patients completed the study. One patient developed new postoperative Q waves and died 24 hr after surgery. Three patients were too haemodynamically unstable to be transported to the nuclear cardiology laboratory for the postoperative follow-up TcPPi-SPECT scan. Three patients withdrew after operation due to fatigue and refused postoperative study. None of the latter six patients had new Q waves.

TABLE I Comparison of perioperative characteristics of patients with positive and negative postoperative TcPPi-SPECT

Characteristics	*Positive (n = 12)*	*Negative (n = 46)*
Age (yr)	54 ± 3	58 ± 4
Sex (M/F)	10/2	44/2
Weight (kg)	83.8 ± 4.1	81.8 ± 1.5
Preop RVEF (%)	0.52 ± 0.02	0.51 ± 0.02
Preop LVEF (%)	0.61 ± 0.05	0.61 ± 0.02
Postop LVEF (%) 48 hr	0.59 ± 0.01	0.64 ± 0.02
Postop LVEF (%) 6 mo	0.63 ± 0.04	0.65 ± 0.02*
Number of grafts	4.0 ± 0.4	3.4 ± 0.4
Aortic cross-clamp time (min)	58 ± 6	49 ± 5

*$P < 0.01$ (postop vs preop).

Patients with positive TcPPi-SPECT did not differ from patients with negative TcPPi-SPECT with regard to age, weight, preoperative NYHA classification, preoperative left or right ventricular ejection fractions (EF), number of coronary vessels grafted and anoxic aortic cross-clamp time (Table I).

Twelve of the 58 patients (21%) developed PMI as documented by postoperative TcPPi-SPECT at 48 hr after surgery. Their irreversible myocardial necrotic mass was 35.7 ± 3.9 g. Two patients developed new Q waves postoperatively, but only one had a positive TcPPi-SPECT. No patient had positive outcome on TcPPi-SPECT six months postoperatively.

Postoperative LVEF was slightly decreased in the positive TcPPi-SPECT group at 48 hr after surgery and returned only to preoperative baseline after six months. In contrast, in the negative TcPPi-SPECT group, the postoperative LVEF improved significantly from preoperative baseline value over six months (Table I). These data have been presented in our previous report.[6]

Postoperative haemodynamic profile and pharmacological requirements

Forty-eight hour measurements of haemodynamic variables of heart rate ranging from 80–98 bpm, systolic blood pressure ranging from 105–124 mmHg and diastolic blood pressure ranging from 50–73 mmHg were not significantly different between patients with positive or negative TcPPi-SPECT (Figure 1). No difference was found between the two groups in regards to central venous pressure (CVP),

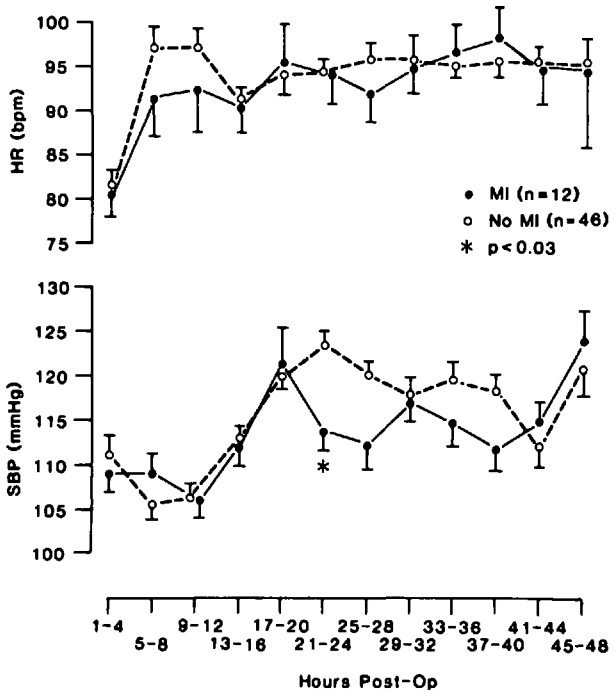

FIGURE 1 Comparison of heart rate (HR) and systolic blood pressure (SBP) in patients with and without postoperative myocardial infarction (PMI) after elective CABG over 48 hr. Data expressed as mean ± SEM.

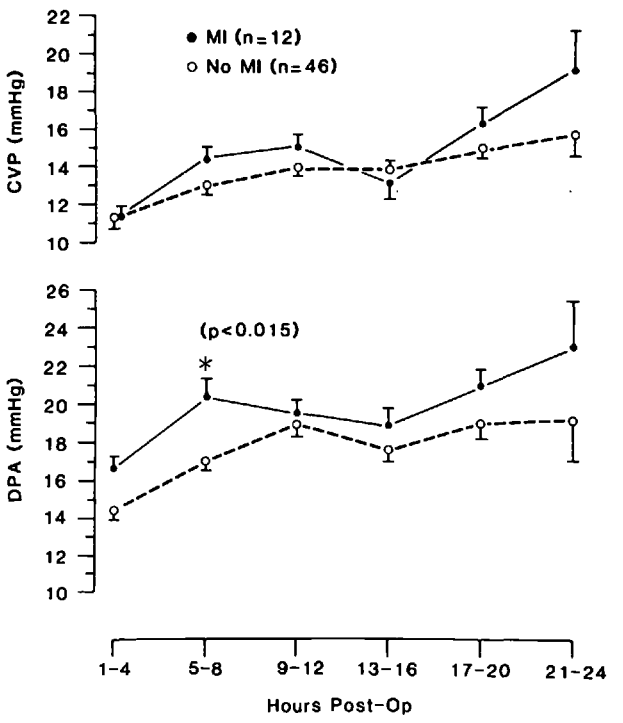

FIGURE 2 Comparison of central venous pressure (CVP) and diastolic pulmonary arterial pressure (DPA) in patients with and without postoperative myocardial infarction (PMI) after elective CABG over 24 hr. Data expressed as mean ± SEM.

but the diastolic pulmonary artery pressure (DPA) was elevated in the PMI group between 5–8 hr after surgery ($P < 0.05$) (Figure 2).

No difference was found in the number of patients requiring dopamine support in the ICU between the positive (16.7%) and negative (13.0%) PMI groups; however, the dosage of dopamine for inotropic support was higher in the PMI group over 24 hr and 48 hr periods after surgery ($P < 0.05$) (Figure 3). There were no differences in morphine, diazepam, propranolol, lidocaine, nitroglycerin (NTG), or nitroprusside (NTP) dosage requirements and incidence between the two groups during the 48 hr postoperative period (Table II).

Discussion

The identification of high-risk subsets of patients after CABG with PMI is essential to the proper management of these patients. Previous outcome[7,8] studies have focused on the identification of risk factors or predictors of adverse outcome during the preoperative and intraoperative periods. Recent studies have begun to focus on the prognostic significance of postoperative ischaemia, haemodynamic and regional wall motion abnormalities in these patients[4,5]. In a smaller subset of these patients, we have showed that careful intraoperative haemodynamic control did not prevent the occurrence of pre-bypass ECG ischaemic changes or PMI.[3] This study demonstrates that patients with PMI following elective CABG surgery, as documented by TcPPi-SPECT, required higher dopamine dosage to maintain stable postoperative haemodynamics.

Perioperative myocardial infarction

The incidence of PMI complicating CABG was 21%, as documented by segmental myocardial uptake of TcPPi using SPECT 48 hr after surgery. This is comparable to previous studies using planar TcPPi, in which the incidence varied between 11 and 31%.[9,10] The prognostic clinical outcome is relatively benign in our patients with PMI, as opposed to those diagnosed by new Q waves and marked CPK-MB elevation.[11–14] This may be related to the location and size of the myocardial infarct,[15,16] and this sensitive diagnostic technique of PMI includes those patients with non-Q wave or non-transmural infarction.[3,6,17,18] Although no patients had complications with unstable angina, PMI or death during the six-month follow-up, those patients with PMI did not improve their postoperative LVEF beyond the preoperative baseline values.

TABLE II Postoperative medication requirements in patients with positive and negative TcPPi-SPECT

TcPPi-SPECT			*Positive*	*Negative*
Morphine	(mg)	24 hr	18.5 ± 4.0 (n = 12)	25.7 ± 2.8 (n = 46)
		48 hr	44.8 ± 7.6 (n = 12)	53.9 ± 5.7 (n = 46)
Diazepam	(mg)	24 hr	14.5 ± 3.1 (n = 12)	15.5 ± 1.5 (n = 46)
		48 hr	29.3 ± 6.1 (n = 12)	31.1 ± 3.1 (n = 46)
Propranolol	(mg)	24 hr	5.3 ± 1.7 (n = 3)	6.3 ± 1.1 (n = 18)
		48 hr	10.7 ± 3.5 (n = 3)	14.2 ± 2.6 (n= 18)
Lidocaine	(mg)	24 hr	1560 ± 740 (n = 2)	1058 ± 552 (n = 3)
		48 hr	3120 ± 1480 (n = 2)	2700 ± 1016 (n = 3)
Nitroglycerin	(mg)	24 hr	29.0 ± 9.5 (n = 2)	61.0 ± 35.1 (n = 4)
		48 hr	57.9 ± 18.9 (n = 2)	122.0 ± 70.2 (n = 4)
Nitroprusside	(mg)	24 hr	71.1 ± 20.6 (n = 10)	98.8 ± 14.2 (n = 46)
		48 hr	149.7 ± 46.5 (n = 10)	200.9 ± 29.9 (n = 46)

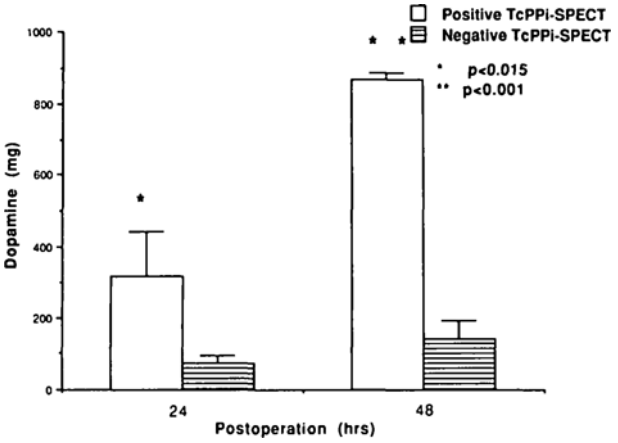

FIGURE 3 Comparison of postoperative dopamine requirements in patients with positive and negative TcPPi-SPECT after elective CABG over 48 hr. Data expressed as mean ± SEM.

Postoperative haemodynamic control

The importance of perioperative haemodynamic control in influencing outcome was underlined by Rao *et al.*[19] who reported a reduction in perioperative re-infarction in patients with coronary artery disease undergoing non-cardiac surgery monitored with pulmonary artery catheter and aggressive treatment of haemodynamic aberrations. However, Tuman *et al.*[20] suggested that pulmonary artery catheterization does not play a major role in influencing outcome after cardiac surgery. There is evidence that poor correlation between pulmonary arterial wedge pressure and left ventricular end-diastolic volume after CABG surgery, probably because of altered ventricular compliance.[21] Nonetheless, current practice is to maintain stable postoperative haemodynamic variables in patients following CABG surgery.

In this study, despite tight control in postoperative HR, BP and CVP, there was still a 21% incidence of PMI of small infarction mass, as documented by TcPPi-SPECT. The dosage requirement of propranolol was comparable in both groups over 48 hr after surgery to maintain HR between 80–100 bpm. The dosage requirement of vasodilators (nitroglycerin and nitroprusside) tended to be higher in the negative TcPPi-SPECT group, indicating postoperative hypertension is the major problem in the non-PMI group. Whereas, in order to achieve controlled haemodynamic indices, the requirement of inotropic support of dopamine is higher in the PMI group for 48 hr after surgery. This validates the significance of positive TcPPi-SPECT in these patients. It has been suggested that as many as 50% of all episodes of perioperative ischaemia[2] and 73% of post-bypass regional wall motion abnormalities[5] occurred despite optimal haemodynamic indices for myocardial oxygen supply and demand. This may reflect that haemodynamic control does not prevent all ischaemic myocardium. Furthermore, the incidence of postoperative ischaemia was found to peak shortly after revascularization and was related to adverse cardiac outcome. A relationship between ischaemia and persistently elevated postoperative HR may exist.[4] Slogoff and Keats observed that no patients suffered PMI or death following CABG although 15.8–25.9% of them experienced systolic BP greater than 200 mmHg during the first 24 hrs after surgery.[22] However, no comparisons between their PMI and non-PMI groups in postoperative HR and BP control or inotropic requirements were attempted. This suggests that some of the PMI were not haemodynamically related and were likely a manifestation of the intrinsic disease process or surgical outcome. It has been shown that PMI occurred in jeopardized segments of the myocardium with poor angiographic run-off, either revascularized or considered too small to have insufficient distribution to warrant bypass grafting,[6] and that the choice of anaesthetic agents has no influence on the outcome after CABG surgery.[22,23] Then the postoperative haemodynamic control of HR by beta-adrenergic blockade agents, filling pressure

by volume, adequate blood pressure by inotropic support and ischaemic ECG changes by vasodilators may be very important for the jeopardized myocardium in terms of preventing the extension and limiting the size of PMI.

Summary

The postoperative course of patients with perioperative myocardial infarction following elective CABG surgery was evaluated. Twenty-one percent of patients sustained irreversible myocardial injury as documented by positive TcPPi-SPECT. Careful control of their postoperative haemodynamic indices for myocardial oxygen balance did not prevent but may have limited the extent of PMI. Over the 48 hr postoperative period, no difference was found between the two groups in haemodynamic variables of heart rate, systolic blood pressure and central venous pressure. However, the diastolic pulmonary pressure (DPA) was higher in the PMI group at 5–8 hr after operation, which may reflect decreased myocardial compliance, as evidenced by lower LVEF at 48 hr after surgery. There were no differences in the incidence and dosage requirements for postoperative sedative or vasoactive drugs between the two groups. Although no difference was found in the incidence of dopamine requirement between the two groups, the dopamine dosage for inotropic support was higher in the PMI group over the 48 hr postoperative period. Unless patients suffered perioperative MI, CABG surgery results in significant improvement of their postoperative LVEF from preoperative baseline level over six months.

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