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Perioperative Myocardial Ischemia: Importance of the Preoperative Ischemic Pattern

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Previous studies investigating the incidence of myocardial ischemia in patients undergoing coronary-artery bypass grafting (CABG) surgery have not considered the potential significance of the preoperative ischemic pattern in the development of intra- and postoperative myocardial ischemia and infarction. Accordingly, the authors compared the frequency and severity of pre-, intra-, and postoperative ischemic episodes (ST-segment depression ≥ 0.1 mV or elevation ≥ 0.2 mV) in 50 men with severe coronary artery disease scheduled for elective CABG. All subjects were monitored by continuous electrocardiography (ECG) (Holter monitor) for 2 preoperative days, intraoperatively, and 2 postoperative days (total monitoring time = 4,363 h). Routine anti-anginal medications were continued until the morning of surgery, and the anesthetic management of the patient was not controlled. During the preoperative period, 42% of the patients had ECG ischemic episodes, 87% of which were clinically silent. Only 18% developed intraoperative ischemia. Postoperatively, the incidence increased to 40%. The number of ischemic episodes/hour (epis/h) of monitoring among the three monitoring periods was similar (0.09 ± 0.12 epis/h preoperatively, 0.11 ± 0.20 epis/h intraoperatively, and 0.05 ± 0.08 epis/h postoperatively; P = NS). The median duration of ischemic episodes was similar pre- and intraoperatively (16 *vs.* 18.5 min, P = NS), but greater postoperatively (41 min, $P < 0.05$). Seventy-six per cent of the perioperative ECG ischemia occurred without acute change (±20% of control) in blood pressure or heart rate. Intraoperative myocardial ischemia occurred in 33% of those patients with preoperative ischemia, but in only 7% of patients without preoperative ischemia ($P < 0.05$). However, neither pre- nor intraoperative ischemia predicted the development of postoperative ischemia. Major outcome (myocardial infarction and/or death) occurred in seven patients. Although all seven major outcomes were preceded by ischemic episodes at some time during the study, perioperative ischemia was not a specific predictor of major outcome. The authors conclude that: 1) CABG patients have frequent preoperative episodes of myocardial ischemia, most of which are silent; 2) anesthesia and surgery do not worsen the preoperative ischemic pattern; 3) ECG changes suggestive of myocardial ischemia frequently follow CABG surgery, although their pathogenesis and significance is as yet unknown; 4) the majority of perioperative ischemic ECG changes occur without acute hemodynamic changes prior to the onset of ischemia; and 5) because the preoperative ischemic pattern appears to be recapitulated intraoperatively, it is relevant to examine the preoperative ischemic pattern to assess the impact of anesthesia and surgery in the development of intraoperative myocardial ischemia. (Key words: Heart: coronary-artery disease; myocardial infarction; myocardial ischemia. Monitoring: electrocardiography; Holter monitoring. Surgery: coronary-artery bypass grafting.)

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PATIENTS WITH CORONARY-ARTERY DISEASE (CAD) have frequent electrocardiographic (ECG) changes indicative of myocardial ischemia during daily activities, unaccompanied by symptoms, changes in heart rate, or changes in blood pressure.[1–7] During coronary-artery bypass grafting (CABG), 26–78% of patients develop similar ECG evidence of myocardial ischemia,[8–13] which may be associated with a threefold increase in perioperative myocardial infarction.[8,9] Previous investigators have not considered the existence of preoperative myocardial ischemia when analyzing the frequency and significance of perioperative ischemia. Ischemia detected intraoperatively, for example, may only reflect the pre-existing preoperative pattern of chronic ischemia. Thus, to assess the impact of anesthesia and surgery upon the pre-existing preoperative pattern of myocardial ischemia, we studied ischemic patterns during the entire perioperative period using continuous ECG (Holter) monitoring on patients undergoing CABG surgery. Patients were studied for two preoperative days, intraoperatively, and for two postoperative days. The frequency and severity of ischemic episodes were compared for all three periods, and the relationship between acute changes in heart rate and blood pressure and the onset of ischemia was examined.

Materials and Methods

With approval from our Committee on Human Research and with written informed consent, we studied 50 ambulatory male patients scheduled for elective CABG surgery at the San Francisco Veterans' Adminis-

tration Medical Center. Patients were excluded if any of the following were present: 1) a preoperative ECG that interfered with the accurate diagnosis of myocardial ischemia (*e.g.*, bundle branch block), 2) valvular heart disease, or 3) prior digoxin therapy. Patients were classified according to anginal pattern,[14] history of myocardial infarction or prior CABG, preoperative ejection fraction, and number of significant coronary arterial stenoses (≥70% diameter stenosis of the left anterior descending, left circumflex, or right coronary artery and ≥50% diameter stenosis of the left main coronary artery). Also noted were diagnoses of hypertension, diabetes mellitus, and the preoperative medications.

Patients were monitored continuously with a two-channel AM Holter ECG recorder (Marquette Electronics, series 8500) for two preoperative days, intraoperatively, and two postoperative days. The preoperative monitoring period took place within the 2 days immediately preceding surgery in 45 patients, within 3 days of surgery in three patients, and within 3 weeks of surgery in two patients. Eight subjects were monitored while outpatients. The intraoperative (pre-revascularization) period began with arrival in the preoperative holding area, and ended with the onset of cardiopulmonary bypass. The postoperative (post-revascularization) period began after removal of the aortic cross-clamp and ended on the second postoperative day after 48–50 h of monitoring had been completed.

Two bipolar leads, CC5 (all patients) and either modified ML (30/50 patients) or modified CM5 (20/50 patients) were used. Lead CC5 (positive electrode placed at the left V5 position, negative electrode placed at the right V5 position) records anterolateral forces; lead CM5 (positive electrode placed at the left V5 position, negative electrode placed at the manubrium) records anterior and inferior forces; lead ML (positive electrode placed at the left iliac crest, negative electrode placed at the manubrium) records inferior forces.[15] To allow continuous monitoring throughout the study, the negative electrode of leads CM5 and ML were placed at the right supraclavicular fossa as close to the manubrium as possible (modified CM5 and modified ML). Lead CM5 was used in lieu of ML whenever inferior Q waves were present; no patient had anterolateral Q waves. Silver/silver chloride electrodes were used and skin impedance was <5 kilo ohms in all patients. No patient had ST-segment changes with change in body position. Patients were instructed to keep a diary of activity and symptoms during the preoperative monitoring period; this was supplemented with careful questioning, so that ECG ischemia could be correlated with the presence (or absence) of symptoms.

Holter tapes were analyzed on a Marquette series 8000 computerized ECG analysis system. After proper classification of all QRS complexes, ST-segment analysis was performed. All abnormal QRS complexes (*e.g.*, those reflecting ventricular ectopic beats and those showing conduction abnormalities) were excluded. A continuous two-lead ST-segment trend was then generated for the duration of the tape (usually 48 h). An ischemic episode was defined as a transient ST-segment shift from baseline of ≥0.1 mV ST depression (with slope ≤ 0), or ≥0.2 mV ST elevation at J + 60 msec, lasting for at least 1 min. Episode duration (from the beginning of ST-segment deviation from baseline to the point of return to baseline) and maximum ST change from baseline were measured. A ST-segment deviation was not considered to be an ischemic episode when associated with intermittent intra-ventricular conduction delay, bundle branch block, or acute myocardial infarction. All ischemic episodes were reviewed independently by two investigators, who were not involved in clinical care of the patient.

Blood pressure was not monitored preoperatively, but was recorded from an indwelling arterial catheter at 5-min intervals pre-bypass; post-bypass blood pressure was recorded at 5-min intervals until the patient arrived in the intensive care unit (ICU), at 15-min intervals for the next 8 h, and then at 15–30-min intervals thereafter. Heart rate (HR) was determined from the ECG recordings. A relationship between HR and/or MAP and ischemia was said to exist when either HR increased ≥20% or MAP changed ±20% just prior to the onset of ischemia. HR at the onset of an ischemic episode was expressed as the percent change from the control period, which was defined by the median HR during the 10 min before the onset of ischemia. Mean arterial blood pressure (MAP) changes were analyzed similarly. The presence of a hemodynamic abnormality upon arrival in the operating room and during the intra- and postoperative periods was also noted. A hemodynamic abnormality was defined as a systolic blood pressure (SBP) ≥ 180 mmHg, a diastolic blood pressure (DBP) ≤ 50 mmHg, or a HR ≥ 100 beats/min lasting for ≥5 min.

Four attending anesthesiologists and two cardiac surgeons were responsible for the clinical care. All were blinded to the study data. Clinical decisions (monitoring, anesthetic, therapy) were not controlled. Cardiac medications (table 1) were continued until the morning of surgery. All patients were premedicated with morphine sulfate and diazepam. The primary anesthetic was either fentanyl (29 patients), sufentanil (14 patients), or morphine (two patients), plus a benzodiazepine (valium or midazolam) or halothane/nitrous oxide (five patients). Pancuronium or vecuronium was used for muscle relaxation and to facilitate tracheal intubation. Ventilation was controlled until tracheal extubation, with

$Pa_{O_2} \geq 70$ mmHg and Pa_{CO_2} at 35–45 mmHg. The anesthetic agents administered during bypass included isoflurane, morphine, fentanyl, and/or sufentanil. While discontinuing cardiopulmonary bypass, 42 patients received a vasopressor or inotropic drug (dopamine, dobutamine, epinephrine, calcium chloride, neosynephrine), usually for hypotension or inadequate cardiac output; these drugs were administered at the discretion of the anesthesiologist and/or surgeon, and were continued until hemodynamic stability existed, as determined by the surgical team. In the ICU, patients were sedated with morphine, diazepam, and/or midazolam until the morning following surgery, at which time they were weaned from mechanical ventilation. Serum potassium levels were maintained at ≥3.0 meq/l. Starting approximately 6–8 h after aortic cross-clamp removal, 46 patients were given digoxin for prophylaxis against supraventricular arrhythmias.

Cardiopulmonary bypass was performed with a bubble oxygenator utilizing hemodilution and moderate systemic hypothermia (26–28° C). Multi-dose cold blood (8° C, Hct 20–25%) with potassium cardioplegia (20 meq/L) and topical saline/ice slush was used for myocardial protection during cardiopulmonary bypass. Distal anastamoses were performed first, during continuous aortic cross-clamping, followed by proximal vein grafting during partial aortic occlusion. An average of 3.3 (range 2–5) grafts were performed per patient. All patients received vein grafts; 41 patients received internal mammary-artery grafts to either the left anterior descending or the first diagonal coronary artery. Cardiopulmonary bypass and aortic cross-clamp times averaged 113 ± 47 and 58 ± 15 min (mean ± SD), respectively.

Major outcome was defined as either a myocardial infarction occurring within the first two postoperative days or death occurring within the period of hospitalization. Immediately after surgery, a 12-lead ECG was obtained and the serum creatine kinase (CK) and CK-MB isoenzyme levels were determined. Both tests were repeated daily until the third postoperative day. Myocardial infarction was diagnosed if the patient developed both new Q waves (≥0.04 duration) and positive CK-MB (≥80 IU). In one patient who suffered a myocardial infarction, a new segmental wall motion abnormality (dyskinesis), diagnosed by transesophageal echocardiography, occurred upon termination of cardiopulmonary bypass. This patient died before leaving the operating suite. All other patients were followed until discharge from the hospital.

Statistical Methods

Chi-square analysis with continuity correction was applied to categorical data. Student's *t* test was used to test the difference between the means in two groups. Analysis of variance was used to detect differences among more than two groups, followed by the Fisher's Protected Least Significant Difference test to determine differences between specific groups. (Continuous variables were log transformed when appropriate). A P value of <0.05 (two-sided) identified statistically significant differences. Results are expressed as the mean ± one standard deviation, unless otherwise indicated.

TABLE 1. Preoperative Clinical and Demographic Data for 50 Patients

Age	62 ± 8 (Range 41–78)
Medications	
Nitrates	50 (100%)
CA^{++} channel blockers	46 (92%)
Beta adrenergic blockers	36 (72%)
Prior MI	29 (58%)
Prior CABG	5 (10%)
Hypertension	23 (46%)
Diabetes mellitus	9 (18%)
Angina class	
1	1 (2%)
2	15 (30%)
3	33 (66%)
4	1 (2%)
# Coronary arteries w/ stenoses	
Average	3 ± 0.8
1	3 (6%)
2	7 (14%)
3	40 (80%)
LMCA	21 (42%)
Ejection fraction	.53 ± .12 (Range 0.30–0.78)

Results

Fifty patients were monitored for an average of 43 ± 12 h preoperatively (total = 2,157 h), 3.2 ± 0.8 h intraoperatively (total = 161 h), and 41 ± 10 h postoperatively (total = 2,045 h). Clinical and demographic data are presented in table 1. The population consisted of older men with severe CAD who had post-infarction angina, had "failed" maximum medical therapy, or had significant left main coronary-artery disease. There were no significant clinical or demographic differences between patients who did and did not demonstrate myocardial ischemia during any of the monitoring periods.

Ischemic Episodes

Only ST-segment changes that were labeled ischemic by both investigators were called ischemic episodes. This represented 90% of the ST-segment changes that were labeled ischemic by either reviewer. ST-segment changes suggestive of ischemia that were not agreed

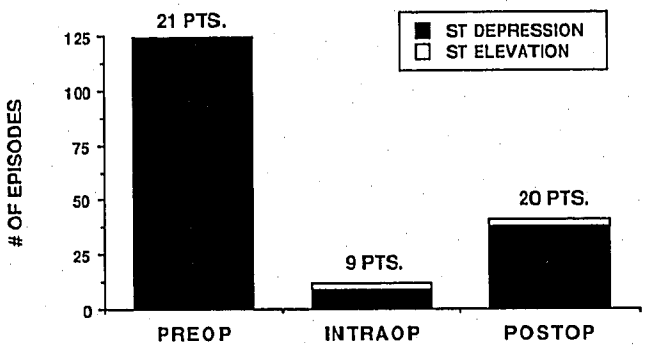

FIG. 1. The incidence and the number of episodes of preoperative (PREOP), intraoperative (INTRAOP), and postoperative (POSTOP) ischemia occurring during the measurement periods.

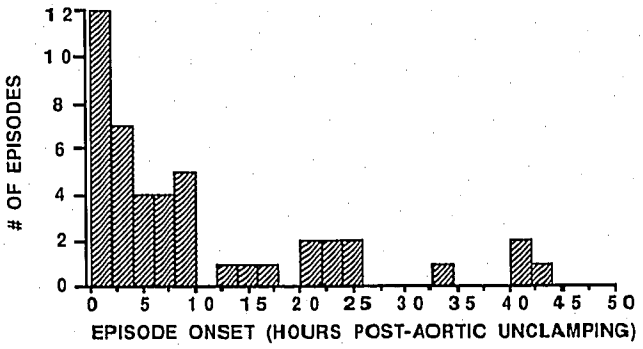

FIG. 2. Distribution of postoperative ischemic episodes according to onset time. For example, 12 episodes occurred within the first 2 h following aortic unclamping, whereas two episodes occurred between the 40th and 42nd hours following unclamping.

upon by both investigators were not analyzed. ECG changes suggestive of myocardial ischemia were frequent in all three periods (fig. 1). Preoperatively, 21 patients (42%) developed 124 episodes of myocardial ischemia lasting a total of 4,578 min (table 2). The median number of episodes per patient was three (range 1–20), and the median episode duration was 16 min (range 3–451 min). One-half of the episodes were associated with a ST-segment change of ≥0.2 mV. Only 13% of the preoperative episodes were accompanied by symptoms of angina, while the remaining episodes (87%) were clinically silent.

Intraoperatively, nine patients (18%) had 12 episodes of myocardial ischemia (three determined by ST elevation and nine by ST depression) lasting a total of 248 min (fig. 1; table 2). The median episode duration was 19 min (range 6–48 min) (P = NS intra- *vs.* preoperative). Four of the 12 intraoperative episodes (33%) were associated with an ST-segment change of ≥0.2 mV. No patient developed ischemic ECG changes between presurgical preparation and arrival in the operating room. Ischemic episodes occurred during insertion of central venous or pulmonary artery catheters (two episodes), anesthetic induction (one episode), skin incision (one episode), sternotomy (two episodes), acute hemorrhage poststernotomy (one episode), and internal mammary artery dissection (five episodes).

TABLE 2. Perioperative Ischemic Episode Characteristics

	Preop	Intraop	Postop
Patients with ischemia	21 (42%)	9 (18%)	20 (40%)
Number of episodes	124	12	41
Magnitude of ST change			
1–1.9 MM	62	8	18
≥2 MM	62	4	23
Episode duration (min), median	16	19	41*
Total ischemia time	4578	248	5103

* $P < 0.05$ compared with pre- and intraoperative periods.

Postoperatively, 20 patients (40%) developed 41 transient ST-segment shifts, suggesting myocardial ischemia lasting a total of 5,103 min (three determined by ST elevation, 38 by ST depression) (table 2). The median episode duration was longer (41 min) than those of the preoperative (16 min) and intraoperative periods (19 min) ($P < 0.05$). Fifty-six per cent of the postoperative episodes were associated with a ST-segment change of ≥0.2 mV. Seventy-eight per cent of the postoperative episodes occurred during the first 10 h after aortic cross-clamp removal, and only 4% occurred after 24 h (fig. 2).

The longer a patient with CAD is monitored with ECG, the higher the chance of detecting myocardial ischemia.[2] Therefore, because the total monitoring time differed for each period, the frequency of ischemic episodes was expressed per hour monitored, so that the three monitoring periods could be compared. The mean number of ischemic episodes/h of monitoring (0.09 ± 0.12 preoperatively, 0.11 ± 0.20 intraoperatively, and 0.05 ± 0.08 postoperatively) was similar for each period (P = NS).

PREOPERATIVE PREDICTION OF INTRAOPERATIVE ISCHEMIA

Of the 21 patients with preoperative ischemia, seven (33.3%) developed intraoperative ischemia (fig. 3). However, of the 29 patients without preoperative ischemia, only two (6.9%) developed intraoperative ischemia. Thus, intraoperative myocardial ischemia was significantly more common in those patients with episodes of preoperative ischemia ($P < 0.05$). Neither preoperative nor intraoperative ischemia predicted the development of postoperative ischemia ($P > 0.1$).

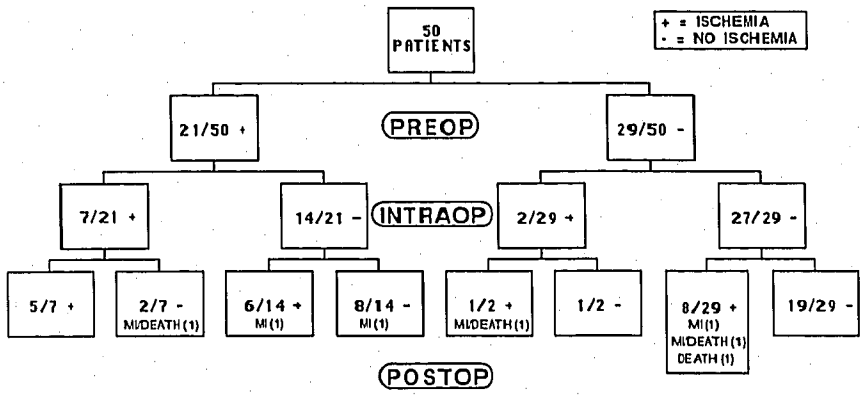

FIG. 3. The relationships between preoperative ischemia (PREOP), intraoperative ischemia (INTRAOP), and postoperative ischemia (POSTOP) and outcome for the 50 study patients.

EPISODE-RELATED CHANGES IN HR AND MAP

HR in the pre- and intraoperative periods was most often ≤80 beats/min (92% and 99% of the monitoring time, respectively) (fig. 4). Postoperative HR, in contrast, was ≤80 beats/min, 27% of the total monitored time ($P < 0.05$ postoperatively *vs.* pre- and intraoperatively), between 80 and 99 beats/min, 48% of the total time ($P < 0.05$ postoperatively *vs.* pre- and intraoperatively), and ≥100 beats/min, 25% of the total time ($P < 0.05$ postoperatively *vs.* pre- and intraoperatively). Thus, HR was well controlled in the pre- and intraoperative periods, but not in the postoperative period. Postoperative cardiac pacing accounted for only 1.7% of the postoperative monitoring time and, when instituted, generally occurred at heart rates between 60 and 90 beats/min. Intraoperative hypertension (SBP ≥ 180 mmHg) occurred in 12% of patients, and hypotension (DBP ≤ 50 mmHg) in 22%. Episodes of hypertension and hypotension were more common postoperatively (28% and 56% of patients, respectively) ($P < 0.05$).

The relationship between ischemic episodes and *acute* changes in HR and/or MAP during each of the three periods appears in figure 5A and B (MAP unavailable preoperatively). Ischemic episodes were preceded by acute increases in HR (≥20%) in 23% of preoperative episodes, 42% of intraoperative episodes, and 10% of postoperative episodes. MAP decreased (≥20%) before 25% of intraoperative episodes, but only 7% of postoperative episodes. Acute increases in MAP (≥20%) were associated with ischemia in 8% of intraoperative episodes and 7% of postoperative episodes. Overall, only 13% of preoperative ischemic episodes, 42% of intraoperative ischemic episodes, and 22% of postoperative ischemic episodes (24% collectively) occurred with an acute hemodynamic change (±20% of control).

Absolute heart rate and blood pressure at the *onset* of ischemia were also analyzed. A heart rate of ≥100 beats/min at the onset of ischemia occurred in no preoperative episodes, 17% of intraoperative episodes, and 41% of postoperative episodes. A DBP of ≤50 mmHg at the onset of ischemia occurred with 33% of intraoperative episodes and 7% of postoperative episodes. A SBP of ≥180 mmHg at the onset of ischemia occurred with no intraoperative episodes and only 2% of postoperative episodes. Overall, no preoperative episodes, 42% of intraoperative episodes, and 46% of postoperative episodes (14% collectively) occurred with a hemodynamic abnormality at the onset of ischemia.

OUTCOME

Seven of 50 patients had major outcomes (fig. 3). Perioperative myocardial infarction occurred in six patients (12%), three of whom died. One additional patient died after a prolonged ischemic episode was followed by cardiac arrest approximately 12 h after arrival in the ICU. All patients who developed a major outcome had evidence of myocardial ischemia during at least one monitoring period. However, because of our small study size and small number of major outcomes, we were unable to determine whether the relationship between ischemic episodes and outcome was significant.

Discussion

Our study documents the frequency and severity of perioperative myocardial ischemia in patients with se-

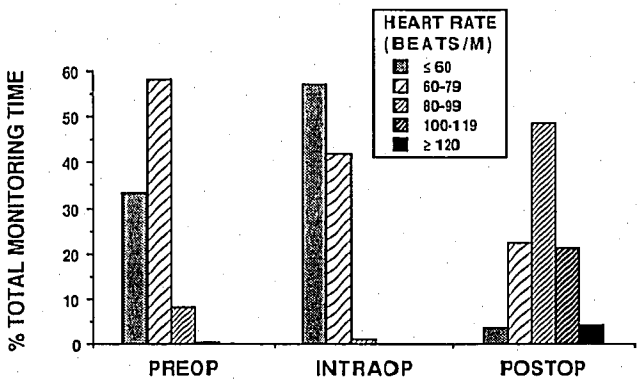

FIG. 4. Heart rate distribution for the three periods.

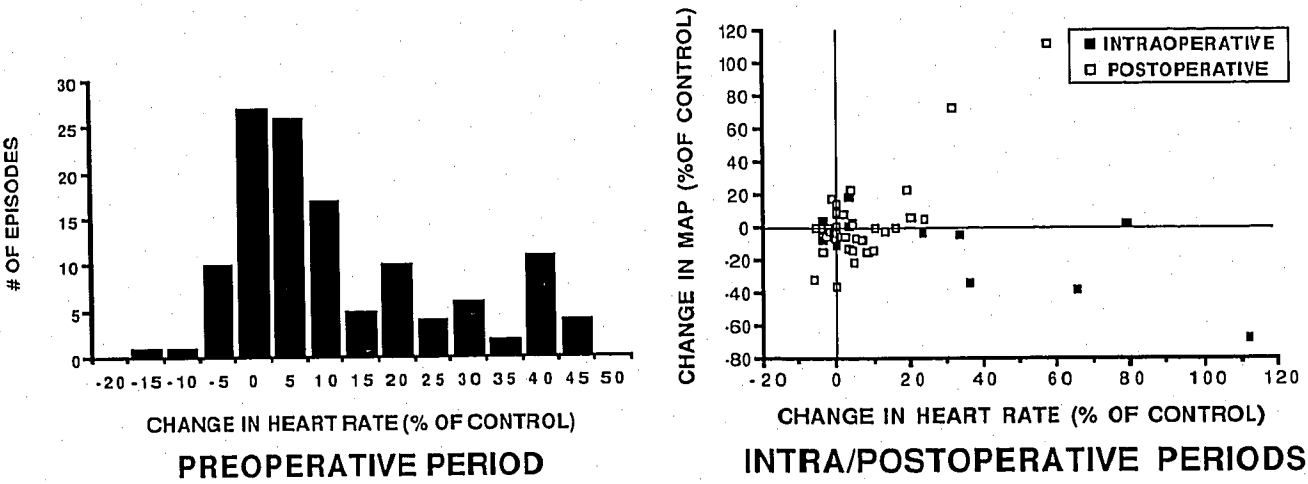

FIG. 5. *A.* Distribution of the 124 preoperative ischemic episodes according to heart rate at the onset of the episode, compared with the control heart rate measured 10 min prior to the onset of the episode. *B.* The relationship of changes in mean arterial pressure (MAP) and heart rate for the intraoperative and postoperative ischemic episodes.

vere CAD undergoing coronary artery bypass grafting. We analyzed ischemic characteristics for our patient population during the pre-, intra-, and postoperative periods, and demonstrated: 1) a prominent preoperative ischemic pattern, 2) an intraoperative ischemic pattern that was no worse than the preoperative ischemic pattern, 3) a surprisingly high incidence of ST-segment changes consistent with ischemia in the postoperative period, and 4) a lack of acute hemodynamic changes preceding most perioperative ischemic episodes.

Despite intensive medical therapy with nitrates, calcium channel blocking drugs, and beta adrenergic blocking drugs, 42% of patients awaiting elective CABG surgery displayed episodes of transient myocardial ischemia during a 2-day period of ambulatory ECG monitoring. Most episodes were clinically silent, and one-half were severe (ST change ≥ 0.2 mV). Other investigators have reported a 53–100% incidence of ambulatory ischemic episodes in patients with a history of CAD,[1–6,16] 68–78% of which were asymptomatic.[3,5,6] The lower incidence of ischemia in our patients may be due to differences in patient populations, medical therapy, or criteria used to diagnose an ischemic event. The consistently high percentage of silent ischemic events[3,5,6] has no well-accepted explanation. Some authors suggest that such silent events are due to a higher than normal threshold for pain perception.[17–19] Others suggest that silent ischemic episodes occur when there is a reduced amount of myocardium at risk for ischemia.[17,20] Still others suggest that silent ischemic episodes may represent an early stage in the "ischemic cascade" that begins with ventricular dysfunction, progresses to silent ST-segment shifts, and ends with angina pectoris.[21]

Previous studies have reported intraoperative myocardial ischemia in 26–78% of patients undergoing CABG surgery.[8–13] In the two most recent studies of patients undergoing CABG (N = 1,518), Slogoff and Keats found that 37% and 55% of patients developed myocardial ischemic episodes, and that these episodes were associated with a threefold increase in perioperative myocardial infarction.[8,9] The preoperative chronic ischemic pattern was not measured, and, therefore, its relationship to intraoperative ischemia was not studied. Our findings indicate that the intraoperative ischemic pattern was no worse than the preoperative chronic pattern. When the incidence of ischemia was adjusted for the number of hours monitored, the number of ischemic episodes per hour monitored was similar in both the pre- and intraoperative periods. Thus, the "stress" of anesthesia and surgery may not result in an increase in myocardial ischemic episodes.

The lower incidence of intraoperative myocardial ischemia in our study (18%) contrasts with that found by Slogoff and Keats (37% and 55%). This difference is surprising when one considers the continuous sampling of the ECG in our study *versus* the intermittent sampling (every 2 min) in their study. The reasons for the difference are not readily apparent, but may be related to the following factors. First, the sizes of the studies differed markedly. Second, the medical management of our patients may have been more "aggressive" than that for the patients studied by Slogoff and Keats. None of our patients, *versus* 21% of theirs, arrived in the operating room with myocardial ischemia. Similarly, only 4% of our patients, *versus* 25% of theirs, arrived in the operating room with a hemodynamic abnormality. Third, the lower incidence of intraoperative ischemia may have

been a reflection of more aggressive control of hemodynamics, since only 33% of our patients developed an intraoperative hemodynamic abnormality, *versus* 76% of their patients. Finally, the methodology of the studies differed. Our ECG data was continuously recorded on Holter tapes and analyzed using a computerized ECG analysis system. The data were then reviewed independently by two investigators. Slogoff and Keats, in contrast, used "trained independent observers" to record hemodynamics and measure ST-segment shifts every 2 min within the operative setting. This strategy may have resulted in an overestimation of the true incidence of ischemic episodes. Thus, differences in patient populations, perioperative myocardial oxygen demand indices, and methodology, or other, as yet unidentified, factors may account for the differences in reported results.

Electrocardiographic changes suggestive of myocardial ischemia occurred frequently in the postoperative, post-revascularization period. Forty per cent of our patients had such episodes, which were often prolonged (median duration = 41 min) and severe (46% had a change in ST-segment of $\geq$0.2 mV). These results, however, must be interpreted with caution. Non-specific ST-segment changes may occur due to changes in body temperature, serum electrolytes, ventilatory patterns, or the influence of drugs. Although many of our patients were given digoxin in the postoperative period, this treatment was not initiated until after most of the ischemic episodes had occurred. Alternately, the ST-segment changes we observed may, in fact, represent true ischemia. Possible mechanisms for post-revascularization ischemia include: 1) increased myocardial oxygen demand which exceeds supply due to inadequate analgesia, shivering, hyperthermia, sympathomimetic medications, increased circulating catecholamines, etc.; 2) decreased myocardial oxygen supply due to diastolic hypotension (generally observed during rewarming), tachycardia, or increased coronary arterial vascular tone; or 3) incomplete myocardial revascularization or graft occlusion.[22] Studies using other methods to validate ischemia are required to evaluate the significance of ST-segment changes in the post-revascularization period.

In the ambulatory non-surgical patient with CAD, most ischemic episodes are not associated with increased HR;[6,7] in the present study, only 23% of preoperative ischemic episodes were preceded by such changes. Furthermore, all of the episodes occurred with a heart rate of <100 beats/min at the onset of ischemia. During the intraoperative period, 42% of ischemic episodes were preceded by an acute hemodynamic change, and 42% were associated with a hemodynamic abnormality (usually tachycardia or diastolic hypotension) at the onset of ischemia. Postoperatively, only 10% of the episodes were preceded by an acute hemodynamic change; however, 46% were associated with a hemodynamic abnormality (usually tachycardia) at the onset of ischemia. These data are in agreement with those of other investigators,[8,9] and they suggest that, although one can stress the myocardium to precipitate ischemia, the majority of ischemic episodes occur without an acute hemodynamic change or with a hemodynamic abnormality at the onset of ischemia. These results support the concept that most transient ischemic episodes appear to be related to intermittent decreases in myocardial oxygen supply, rather than increased myocardial oxygen demand, perhaps because of increased coronary artery vascular tone or increased platelet aggregability.

In summary, we found that 42% of patients presenting for elective CABG surgery had frequent preoperative episodes of myocardial ischemia, despite maximum anti-anginal therapy. Most of these episodes (87%) were clinically silent. Intraoperative myocardial ischemia occurred in only 18% of our patients. Furthermore, the intraoperative ischemic pattern was no worse than the preoperative pattern. Postoperatively, ECG changes suggestive of myocardial ischemia were frequent (40%), but their meaning is uncertain. Most perioperative ischemic episodes occurred in the absence of acute changes in HR or BP, and without a hemodynamic abnormality at the onset of ischemia. Finally, because intraoperative ischemia appears to recapitulate the preoperative ischemic pattern, measurement of the preoperative ischemic pattern may be necessary to understand and analyze the effect of anesthesia and surgery upon perioperative myocardial ischemia.

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