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Comparison of Hemodynamic, Electrocardiographic, Mechanical, and Metabolic Indicators of Intraoperative Myocardial Ischemia in Vascular Surgical Patients with Coronary Artery Disease

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To compare mechanical, electrocardiographic, and metabolic indices of myocardial ischemia, the cardiokymogram (CKG), the V_5 ECG, left anterior descending coronary artery territory lactate extraction, and pulmonary capillary wedge pressure (PCWP) were measured in 53 vascular surgical patients with coronary artery disease. Measurements were performed preoperatively and at four specific intraanesthetic intervals: after tracheal intubation, before surgery, and 10 and 30 min after incision. Measurements and sampling sequence took 5–7 min, and therapy for the probable cause of ischemia was instituted following completion of this sequence. Myocardial ischemia was defined as type II or III CKG, 0.1 mV or greater horizontal or downsloping depression of V_5 ECG ST segment, 0.2 mV or greater elevation of V_5 ECG ST segment, or myocardial lactate production. Thirty-nine patients (74%) had a total of 89 episodes of myocardial ischemia. Seventy-four episodes (83%) were detected by the CKG, 31 (44%) were evident on the ECG, and 13 (15%) by evidence of lactate production. The concordance among the indices of myocardial ischemia was poor. Patients with an abnormal preoperative ECG experienced a greater number of ischemic episodes ($P < 0.001$). Elevation of PCWP or the presence of A-C or V-waves greater than 5 mmHg above the mean did not individually reflect ischemia reliably. Intraoperative myocardial ischemia is common in vascular surgical patients and is most sensitively detected by ventricular wall motion abnormality. (Key words: Anesthetic technique: general. Artery, coronary: regional blood flow. Heart: ischemia; lactate; ventricular wall motion abnormality. Measurement techniques: cardiokymography. Monitoring: electrocardiography.)

PERIOPERATIVE MYOCARDIAL ISCHEMIA has recently been established as an independent risk factor for postoperative myocardial infarction in patients undergoing coronary artery bypass grafting (CABG).[1] Although this relationship has not been established in any other patient population, detection of ischemia appears to be an important monitoring goal of the anesthesiologist managing any patient with coronary artery disease (CAD). The aim of the present study was to compare a variety of techniques that have been proposed as useful indicators of myocardial ischemia. Hemodynamics, electrocardiograms (ECG), cardiokymograms (CKG), and myocardial lactate extraction were simultaneously recorded at five specific intervals before and during anesthesia in patients with CAD undergoing major abdominal vascular surgery.

Materials and Methods

The data of 53 patients with CAD, without bundle branch block, scheduled for reconstruction of their abdominal aorta or common iliac arteries are reported. None of the patients had suprarenal clamping of the aorta. Eight additional patients were studied but excluded from data analysis because they demonstrated silent myocardial ischemia prior to anesthesia: six had myocardial lactate production, in three combined with new abnormal wall motion as detected by CKG, and in one with a new ECG abnormality; two had new wall motion abnormality only. Control ECG and CKG were obtained during the preoperative evaluation of the patients for inclusion into the study. The study was approved by the Ethics Committee of the University of Umeå, and informed consent was obtained from all patients. The diagnosis of CAD was established by one or more of the following criteria: 1) angina pectoris combined with positive arm or leg exercise test; 2) previous myocardial infarction, verified by ECG and elevated enzymes; 3) dipyridamole–thallium scintigram demonstrating permanent or temporary perfusion defects,[2,3] 4) angina pectoris plus significant stenosis of one or more coronary ateries documented by angiography; and 5) previous CABG with persistent angina pectoris or positive dipyridamole–thallium scintigraphy. Relevant clinical data are presented in table 1.

Morphine (0.2 mg/kg) or meperidine (1 mg/kg) was administered sc with the patient's normal oral cardiac medication approximately 30 min prior to the instrumentation. A radial or brachial artery catheter, a flow guided pulmonary artery thermodilution catheter, and a

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TABLE 1. Clinical Data of the Investigated Patients (n = 53)

Age (yr)	64 ± 8
(range)	45–81
Sex	6F/47M
Previous medical history	
Angina pectoris	38
Myocardial infarction	21
Congestive heart failure	10
Hypertension	23
Diabetes	6
NYHA function class	
I	5
II	20
III	25
IV	3
Cardiomegaly on chest x-ray	22
Normal preoperative ECG	26
Abnormal preoperative ECG	27
Cardiac medication (n = 38)	
Beta adrenergic blocking drugs	14
Calcium channel blocking drugs	5
Nitrates	15
Diuretics	23
Vasodilators	6
Other antihypertensive drugs	4
Digitalis	9
Antiarrhythmic drugs	3
No cardiac medications	15

great cardiac vein (GCV) thermodilution catheter were introduced under local anesthesia. Fluoroscopy was used to guide placement of the latter two catheters. Standard 12-lead ECG were recorded in 37 patients and ECG lead V_5 in the remainder. The CKG probe was positioned on the chest wall over the anterior wall of the left ventricle with the aid of fluoroscopy.

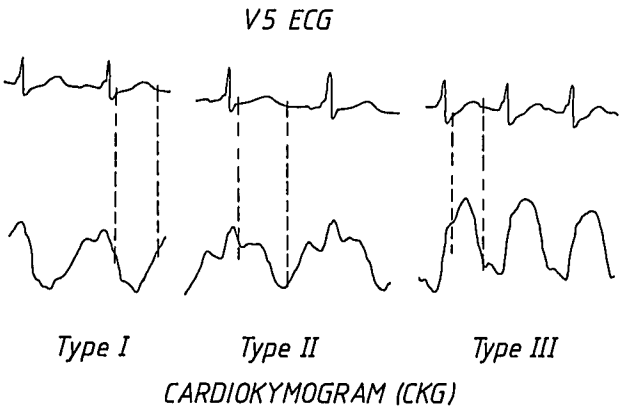

FIG. 1. V_5 ECG (top) and CKG (bottom) recorded simultaneously during tracheal intubation of one study patient. Before intubation there is a normal systolic inward motion (Type I). Within seconds of insertion of the laryngoscope, a partial early systolic outward motion (Type II) is recorded without ECG changes. Two minutes later the CKG demonstrates total outward motion during systole (Type III). At this time the ECG is still not diagnostic of ischemia. Hatched lines denote the ejection period.

After preoxygenation anesthesia was induced with thiopental 2–6 mg/kg with or without fentanyl (3 μg/kg). Tracheal intubation was facilitated with succinylcholine 1–1.5 mg/kg and the patients lungs were ventilated by an Engström® ECS 2,000 volume cycled ventilator delivering nitrous oxide/oxygen (60–70/40–30). End-tidal carbon dioxide tension was measured by an infrared analyzer (Datex or Engström) and maintained between 32 and 35 mmHg. Anesthesia was maintained with halothane, isoflurane, fentanyl, or a combination of the latter two drugs. Muscle relaxation was achieved by pancuronium bromide (0.1 mg/kg).

The V_5 ECG, arterial, pulmonary arterial, and central venous pressures were recorded continuously throughout the investigational period on a Mingograph® 82 recorder running at slow speed (2.5 mm/s). The following measurement sequence was performed at each of five predetermined intervals: 1) recording of V_5 ECG, arterial, pulmonary arterial, pulmonary capillary wedge, and right atrial pressures at 50 mm/s on a Mingograph® 82 recorder; 2) determination of thermodilution cardiac output in triplicate using 5 ml of normal saline as the indicator injected over 1.2 s at end-expiration and synchronized to the R-wave of the ECG; 3) recording of 12-lead ECG on a Mingograph® 62 recorder at 50 mm/s; 4) recording of CKG and V_5 ECG on a two-channel Cardiokinetics® recorder at 50 mm/s; and 5) sampling of arterial and great coronary venous blood (4 ml each) for analysis of lactate. The duration of the entire sequence was between 5 and 7 min.

Each measurement–sampling sequence was performed at five specific intervals: 1) prior to induction of anesthesia, 2) 2 min after tracheal intubation, 3) during stable anesthesia prior to incision, 4) 10 min after incision, and 5) 30 min after incision. Cardiac output was not measured in association with tracheal intubation.

A mean PCWP of 15 mmHg was considered the upper limit of normal. PCWP analog tracings were inspected for A-C or V-waves greater than 5 mmHg above the mean PCWP. If they were present, the waveform was considered abnormal; if absent, normal. Measurement and recording techniques for CKG and regional coronary sinus studies have previously been described in detail.[4–6]

Myocardial ischemia was defined as follows: 1) a horizontal or downsloping ECG ST-segment depression of 0.1 mV or greater, or elevation of 0.2 mV or greater compared to the stable preoperative and pre-induction tracings, measured 80 ms after the j-point; and 2) change from type I to type II or type III CKG (fig. 1) in at least three consecutive, interpretable beats[4] and/or change from myocardial lactate uptake to production. The ECG were interpreted by a single, blinded reader and the CKG

were read independently by two blinded observers. Discordant readings were not included in the data analysis.

The development of myocardial ischemia was judged by the responsible anesthetist based on inspection of the ECG and the CKG, whereas lactate analysis and PCWP waveform analyses were performed after conclusion of the case. The CKG was interpreted as demonstrating ischemia on-line only when an unequivocal type III waveform was observed. There was no instance when CKG was considered abnormal by the responsible anesthetist, which was not confirmed by the two blinded reviewers.

When myocardial ischemia was diagnosed, the probable cause was established among the following: hypotension, hypertension, tachycardia, or ischemia without associated systemic hemodynamic changes. The following criteria were used: 1) hypotension greater than 30% reduction of mean arterial pressure compared to the awake control value or the value recorded on the ward, whichever was lowest; 2) hypertension greater than 20% increase of mean arterial pressure compared to the same values as above; 3) tachycardia, heart rate elevation of greater than 20% compared to the awake preoperative value or to the value recorded on the ward, whichever was highest or to above 90 beats/min; and 4) ischemia without associated hemodynamic changes.

Treatment of ischemia was instituted after conclusion of the measurement–sampling sequence according to the probable cause. The following treatments were used: 1) hypotension: decrease of inspired volatile anesthetic concentration if a volatile anesthetic was being administered (if not effective, administration of phenylephrine); 2) hypertension: increase of anesthetic depth by starting or increasing inspired volatile anesthetic concentration or administering additional narcotic (if ineffective, administration of nitroglycerine); 3) tachycardia: after ruling out hypovolemia, increase of anesthetic dose as outlined above (if ineffective, administration of metoprolol in incremental doses of 1 mg iv); and 4) ischemia without associated systemic hemodynamic changes: discontinue isoflurane if this agent was the primary anesthetic (if this maneuver was ineffective, administration of nitroglycerine). If isoflurane was not the primary anesthetic, nitroglycerine was administered initially.

The frequency and severity of angina pectoris was evaluated during the hospital stay prior to surgery and daily during the first 3 postoperative days. Blood for serum CKMB (4, 16, 40, 64, and 78 h after surgery) and daily 12-lead ECG were obtained during the first 3 postoperative days. Criteria for the diagnosis of myocardial infarction included a new q-wave on the ECG and elevation of the CKMB.

Linear regression analysis was used to compare changes in central venous pressure to changes in pulmonary capillary wedge pressure. Paired and unpaired t tests were used to analyze changes in stroke volume index. Chi-square test was used for the remainder of the analyses. Results are presented as the means ± SD. A P value < 0.05 was considered statistically significant.

TABLE 2. Number of Patients Demonstrating Myocardial Ischemia, as Detected by the ECG, CKG, and/or Lactate Production in Relation to Their Preoperative ECG

	No Ischemia	Ischemia
Normal preoperative ECG (n = 26)	10	16
Abnormal preoperative ECG (n = 27)	4	23

Although a normal preoperative ECG was not associated with a significantly lower incidence of myocardial ischemia during anesthesia and operation (chi-square = 3.81; $P = 0.051$), the total number of ischemic events in such patients was significantly lower than in patients with an abnormal preoperative ECG (chi-square = 16.52; $P < 0.001$; table 4).

Results

Measurements were obtained a total of 261 times. Ischemic ECG changes were found in leads other than V_5 in the presence of a normal V_5 in only one patient of the 37 who had complete 12-lead ECG. All ECG changes are therefore referred to as the V_5 lead. CKG data were absent in 16 of 261 (6.2%) measurement periods, either because of discordant interpretation readings (2.3%) or because of technical problems. Lactate data were obtained at 254 of the 261 measurement intervals. The missing samples (2.7%) were due to technical sampling problems or clotting in the test tubes. Thirty-nine of the 53 patients (73.6%) demonstrated evidence of one or more ischemic events during anesthesia (table 2). If the ECG had been the only method employed to detect myocardial oxygen imbalance, ischemia would have been diagnosed in only 17 patients (32% of all patients; 44% of those with ischemia).

The distribution of the 89 ischemic events in these 39 patients relative to the intraoperative period and the method of detection are presented in table 3. An abnormal CKG was recorded during 74 of 89 ischemic events, whereas the ECG was abnormal in 31 and lactate production occurred in only 13. CKG abnormalities were

TABLE 3. Distribution and Methods of Detection of Ischemic Events

	ECG	CKG	Lactate Production
Induction of anesthesia (n = 23)	6	19	6
Unstimulated anesthesia (n = 20)	5	18	1
Surgical stimulation (n = 46)	20	37	6

TABLE 4. Method of Diagnosis of Ischemic Episodes in Relation to the Preoperative ECG

	ECG*	CKG	Lactate Production
Normal preoperative ECG† (n = 16)	3/30 (10.0%)	26/30 (86.7%)	6/30 (20.0%)
Abnormal preoperative ECG (n = 23)	28/59 (48.3%)	48/59 (82.8%)	7/59 (12.1%)

* The ECG was more commonly demonstrating ischemia in individuals with an abnormal preoperative ECG (chi-square = 12.29; $P < 0.001$).

† The incidence of ischemic episodes was greater in patients with abnormal preoperative ECG (chi-square = 16.52; $P < 0.001$).

significantly more common than either ECG ST-segment abnormalities (chi-square = 25.81; $P < 0.001$) or lactate production (chi-square = 52.57; $P < 0.001$).

Table 4 presents the incidence of ECG ST-segment, CKG, and metabolic abnormalities with respect to the presence of a normal *versus* an abnormal preoperative ECG. Although a normal preoperative ECG was not associated with a significantly lower chance of developing myocardial ischemia than an abnormal ECG (chi-square = 3.81; $P = 0.051$; table 2), the total number of ischemic events was greater in patients with an abnormal preoperative ECG (chi-square = 16.52; $P < 0.001$; table 4). New ST-segment changes during anesthesia were significantly more common in patients with an abnormal preoperative ECG than those with a normal preoperative ECG (table 4), (chi-square = 12.29; $P < 0.001$). Twice as many episodes of ischemia were recorded by an abnormality of a single variable than of more than one measurement. Only two ischemic episodes were diagnosed by all three measurements (*i.e.*, ECG, CKG, and lactate measurement) (table 5). One of these two patients (no. 5) had a lethal postoperative infarction.

The change of PCWP from awake was calculated for each measurement period and related to the appearance of myocardial ischemia at that time (as judged by ECG, CKG, and lactate) and the presence (13%) or absence (87%) of an abnormal PCWP waveform. The distribution

TABLE 5. Incidence of Abnormal Findings by Various Detectors of Ischemia in Relation to the Preoperative ECG

	Normal Preoperative ECG	Abnormal Preoperative ECG
ECG only	0	8
CKG only	22	27
Lactate production only	4	1
ECG + CKG	2	17
ECG + lactate production	0	2
CKG + lactate production	1	3
ECG + CKG + lactate production	1	1

TABLE 6. Number of Ischemic *versus* Nonischemic Observations with Normal *versus* Abnormal PCWP Waveform in Relation to Change in Mean PCWP during Anesthesia from the Awake Values

PCWP Change from Awake (mmHg) (range)	Observations with Normal Waveform		Observations with Abnormal Waveform*	
	Ischemia	No Ischemia	Ischemia	No Ischemia
<−5	1	1	—	—
−5 to −1	13	11	—	—
0 to 4	29	48	—	—
5 to 9	19	31	6	8
10 to 14	6	7	3	—
>14	3	5	9	1
Total	71	103	18	9

Note that the combination of an increase in PCWP of 10 mmHg or greater and an abnormal PCWP waveform was usually associated with myocardial ischemia, whereas an equivalent increase in PCWP with normal waveform was not.

* Abnormal waveform was defined as new A-C or V-waves of 5 mmHg or greater above the mean PCWP.

of the individual values of PCWP is given in table 6. The combination of an increase in PCWP and an abnormal waveform was only observed after tracheal intubation and/or during surgical stimulation and invariably occurred with increase in blood pressure alone or combined with tachycardia. A normal PCWP (less than 15 mmHg) was never observed in association with an abnormal waveform. Neither elevation of PCWP nor appearance of an abnormal waveform by itself discriminated sensitively between presence or absence of myocardial ischemia. However, an abnormal waveform was more likely to be associated with myocardial ischemia than was a normal waveform (chi-square = 6.34; $P < 0.02$). The changes in stroke volume index from awake to ischemic (n = 89) or nonischemic (n = 65) intraoperative episodes were not different (-4.5 ± 10.2 *vs.* -5.5 ± 7.7 ml · m^{-2}; NS).

CLINICAL POSTOPERATIVE RESULTS

Postoperatively, 15 of 53 patients experienced an increased frequency and severity of angina pectoris as compared with the preoperative period. Thirteen of the 39 patients with documented intraoperative ischemia and two of those without intraoperative ischemia experienced this problem (chi-square = 1.84; NS). One patient (no. 13) had a small posterior myocardial infarction diagnosed by ECG and enzyme analysis on the first postoperative day but recovered rapidly and uneventfully. Another patient (no. 5) developed a large, lethal, anterior wall infarction on the third postoperative day. These patients were among those demonstrating intraoperative myocardial ischemia by our three preselected criteria. A third patient without perioperative myocardial ischemia died of myocardial infarction 3 weeks postoperatively following three reoperations due to surgical complications.

Discussion

The most important findings of this study are as follows:

1. Patients with CAD undergoing major abdominal vascular surgery have a high incidence of abnormal left ventricular wall motion during anesthesia, as detected by the CKG.
2. Patients with CAD who have a normal preoperative resting ECG are less likely to develop myocardial ischemia during anesthesia and operation than those who have an abnormal resting ECG. If they do develop ischemia, the ECG will reflect it less commonly than the CKG.
3. There is a poor correlation between ischemia diagnosed by ECG, CKG, and myocardial lactate production during anesthesia for abdominal vascular surgery.
4. Neither elevation of PCWP, nor appearance of an abnormal waveform discriminates sensitively between the presence or absence of myocardial ischemia.

The purpose of this study was to compare simultaneously during anesthesia and surgery a number of indices of myocardial ischemia in patients with a known high risk of morbidity and mortality from CAD. Vascular surgical patients comprise such a population. The indices we examined range from those that can be interpreted on-line to others that require *in vitro* analysis and calculation for retrospective diagnosis. In this study we had as an aim the treatment of myocardial ischemia as soon as possible after the measurement and sampling sequence was completed, the diagnosis made, and the probable cause assigned. The measurement sequence took 5–7 min to complete. Although it is theoretically possible that myocardial ischemia could have been diagnosed by the continuously running ECG between measurement sequences and treatment started at this time, this did not occur.

The question was raised in the editorial review process whether the treatment of myocardial ischemia was responsible for the discrepancy of positive findings among the indices we examined. Abnormal ventricular wall motion predictably precedes ECG changes when coronary artery occlusion occurs. Thus, it was asked, did our therapy abort the ischemia after development of ventricular wall motion abnormalities but before ECG changes could develop? This is important because it relates to the sensitivity of the various indices. For the following reasons it is unlikely that we were able to detect, diagnose, and treat myocardial ischemia in such a rapid time frame and effective manner that this accounted for the divergence among the indices: our measurements were performed at arbitrary intervals during anesthesia; initiation of therapy was inevitably delayed by protocol design; interpretation of the CKG was performed post hoc unless clear paradoxical motion (type III) was noted; metabolic analysis was always retrospective; and pulmonary capillary wedge pressure elevation was not used as an index of ischemia. Thus, our findings indicate that there are major differences of sensitivity among the indices of ischemia we evaluated.

The high incidence of wall motion abnormality is consistent with an increasing body of knowledge indicating that intraanesthetic ischemia is more common than previously recognized or detected by the ECG, the most commonly employed technique to assess myocardial ischemia. Following tracheal intubation, Kleinman *et al.*[7] demonstrated a 45% incidence of positive thallium scintigrams, indicating heterogeneous left ventricular perfusion and/or ischemia despite a low incidence of ECG changes. Bellows *et al.*[5] found eight of 24 patients with LAD coronary artery disease to have abnormal wall motion, as detected by the CKG, during the peri-induction period. Only one of these eight patients demonstrated simultaneous ECG changes indicative of ischemia, and adverse hemodynamic changes were absent in most. In that study the clinicians responsible for management attempted only to keep hemodynamics as normal as they were able; the CKG was analyzed retrospectively. Only one of 25 control patients without CAD demonstrated an abnormal CKG despite less well-controlled hemodynamics. That study also represents the only data base, of which we are aware, for ventricular wall motion analysis during anesthesia of patients without known or suspected heart disease.

Smith *et al.*,[8] using two-dimensional transesophageal echocardiography (2-DTEE), documented new systolic wall motion abnormalities at some time during anesthesia and surgery in 24 of 50 patients with previous myocardial infarction undergoing either vascular surgery or myocardial revascularization. Only six patients experienced ST-segment changes indicative of ischemia. Furthermore, all patients demonstrating an ECG abnormality also had abnormal wall motion. In contrast to Smith *et al.*,[8] we did not find that all instances of ECG abnormality were accompanied by wall motion abnormality. It is possible that wall motion abnormalities were present in areas not sampled by the CKG or that wall motion abnormality was too slight to be detected by this technique.

Patients with abnormal preoperative ECG were more likely to demonstrate intraanesthetic ECG abnormalities. This agrees with previous observations by Coriat *et al.*[9] in comparable patients. They also observed a similar incidence of ECG abnormalities as we found in the present study. They interpreted their findings to indicate that in patients with CAD a normal preoperative ECG predicted a lower risk of intraanesthetic ischemia than an abnormal ECG. The fact that ischemia was present at all intraanesthetic measurement intervals in five of our 27 patients with an abnormal preoperative ECG, whereas this was true in only one of 26 patients with normal preoperative

TABLE 7. Individual Values of Change in PCWP and Change in CVP from the Awake Values, and the Methods of Diagnosis of Ischemia during Ischemic Episodes Associated with an Abnormal PCWP Waveform

Patient No.	PCWP Change (mmHg)	CVP Change (mmHg)	Method of Diagnosis
19	+5	+4	ECG + CKG
33	+5	0	ECG
33	+8	+3	CKG
8	+7	+4	CKG
8	+9	+6	CKG
18	+9	+3	ECG + CKG
46	+12	+6	CKG
13	+12	+5	CKG
13	+15	+4	CKG + lactate production
13	+17	+6	Lactate production
59	+16	+6	CKG
59	+18	+6	CKG
21	+21	+12	ECG + CKG
21	+25	+11	ECG + CKG
10	+25	+6	CKG + lactate production
11	+28	+6	ECG + CKG
49	+32	+10	ECG

$\Delta CVP = 0.3 \times \Delta PCWP + 1.5$ (mmHg); $r = 0.739$ ($P < 0.001$; $n = 17$). Only seven of 17 episodes were obvious by ECG changes.

ECG suggests that ischemia is also more persistent in such patients. It may also suggest that accepted criteria for interpreting the ECG to indicate myocardial ischemia during anesthesia are too stringent.

The objective of inserting the coronary vein catheter in the GCV was to sample blood from the anterior left ventricular wall, the same region of myocardium monitored by the V_5 ECG and the CKG. Sampling blood from this region minimizes dilution of blood from potentially ischemic areas within the left anterior descending coronary artery (LAD) territory by blood from nonischemic areas. Furthermore, blood from the left circumflex or right coronary arteries will be minimally represented in the samples. Only 13 of the 89 episodes defined as ischemic by our criteria were associated with lactate production. There are several possible explanations for the low sensitivity of the myocardial lactate balance studies. The GCV drains the area of the myocardium perfused by the LAD coronary artery,[10] whereas abnormalities in the V_5 ECG and the CKG may reflect ischemia in areas of the myocardium adjacent to the LAD territory. The ischemic area may be too small to produce sufficient lactate to change LAD regional lactate uptake to production. Finally, flow through this ischemic region may be too low to wash metabolites into the bloodstream at the time of sampling. The recent recognition of coronary vasospasm as a cause for intraanesthetic ischemia[7] suggests the importance of the latter mechanism even in the absence of decreased coronary perfusion pressure.

Kaplan and Wells[11] suggested that development of prominent A-C or V-waves in the PCWP tracing, a reflection of decreased left ventricular compliance or mitral valve regurgitation, represented a sensitive and independent indicator of ischemia. Our data suggest that neither elevation of PCWP nor development of abnormal waveform alone reliably indicate the presence of ischemia. Lieberman *et al.*[12] also found no relationship between elevated PCWP and ECG changes diagnostic of myocardial ischemia in patients undergoing coronary bypass surgery. Retrospective analysis of our data suggests that the combination of elevated PCWP and abnormal waveform may be a useful index (table 6). This finding may serve as a hypothesis for future prospective studies. Kaplan and Wells found that only five of 15 patients who developed abnormal PCWP waveform and an elevated PCWP demonstrated a simultaneous ECG abnormality. We confirmed this finding (table 7).

LIMITATIONS OF THE CKG

As noted above, abnormal CKG was the most common finding indicating myocardial ischemia. Previous workers have noted a high correlation of CKG abnormalities with wall motion abnormalities detected by other methods and with scintigraphic perfusion defects.[13–15] Furthermore, for the most part, abnormalities documented in humans have been limited to patients with coronary artery lesions, indicating a high specificity.[4,5,14,15] For instance, in a multicenter study of the CKG, Weiner *et al.*[15] observed a 72% sensitivity and 84% specificity of the exercise CKG in detecting angiographically demonstrable significant coronary artery disease, which compared well with their results from nonquantitative exercise thallium scintigraphy (78% and 86%, respectively). Thus, much experimental and clinical evidence indicate that an abnormal CKG connotes abnormal ventricular wall motion and abnormal CKG occur in humans almost exclusively in patients with CAD. Nevertheless, doubt exists that wall motion abnormalities are entirely specific for ischemia. For instance, Videcoq *et al.*[16] found wall motion abnormalities in the presumed absence of myocardial ischemia when a calcium channel blocking drug was administered to dogs receiving isoflurane. They attributed this to a delayed onset of cardiac contraction. Similar findings have been reported after intracoronary injection of nifedipine in humans.[17] Furthermore, unpublished experiments demonstrated that perfusion of a canine coronary artery with oxygenated, citrated blood, which causes myocardial depression by calcium binding, resulted in abnormal regional ventricular wall motion identical to that consequent to interruption of flow in the same artery.¶ This indicates that regional

¶ Geffin G, Lowenstein E: Personal communication. March, 1988.

depression of function can cause abnormal regional wall motion in the absence of ischemia. It is possible that any technique using ventricular wall motion as a "gold standard" for ischemia may overestimate its true incidence.

In summary, we searched for hemodynamic, electrocardiographic, metabolic, and mechanical indices of myocardial ischemia at four specific times during anesthesia in 53 patients undergoing vascular surgery who did not demonstrate evidence of myocardial ischemia immediately preoperatively. Seventy-four percent of patients had at least one episode meeting our criteria for diagnosis of myocardial ischemia. Patients with an abnormal preoperative ECG experienced a greater number of ischemic episodes. Abnormal wall motion, as reflected by cardiokymography, was present in 83% of episodes, whereas ECG abnormalities and lactate production were present in fewer episodes (44% and 15%, respectively). Concordance among these three techniques was not complete. Neither elevation of PCWP nor abnormal PCWP waveform was a sensitive indicator of myocardial ischemia. We conclude that intraoperative myocardial ischemia is a frequent event in vascular surgical patients.

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