Title: POSTOPERATIVE MYOCARDIAL ISCHEMIA IN PATIENTS UNDERGOING CORONARY ARTERY BYPASS GRAFT SURGERY

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Introduction. Recent evidence suggests that postoperative myocardial ischemia may be an important predictor of adverse cardiac outcome in patients undergoing coronary artery bypass graft (CABG) surgery.[1] However, the characteristics and prognostic importance of postoperative ischemia following CABG surgery are generally unknown. We determined the prevalence, characteristics, and prognostic importance of postoperative ischemia for 10 perioperative days using continuous electrocardiography (ECG).

Methods. After informed consent and IRB approval, 38 consecutive adult males undergoing elective CABG surgery were monitored with continuous 2-lead (CM5, CC5) ECG (QMED-One TC) for 1-2 days preoperatively, intraoperatively, and 7 days postoperatively. Patients with uninterpretable ECG (left bundle branch block) were excluded. All cardiac medications were continued until the morning of surgery. The postoperative period was defined as beginning with completion of the proximal coronary grafts. ECG ischemic episodes were defined as reversible ST depression from baseline ≥ 1.0 mm, or ST elevation ≥ 2.0 mm, lasting at least 1 min. Baseline was adjusted for positional changes and temporal drift. All episodes were verified by two independent blinded investigators using hard-copy ECG data. Clinical care was not controlled by study protocol, and clinicians were unaware of the research data collected. Serial CPK-MB levels and daily 12-lead ECGs were done to assess outcome, which was defined as myocardial infarction (new Q waves, MB > 50 I.U.), congestive heart failure (IABP, CI < 2 with PCWP > 18, or alveolar edema), major dysrhythmia (ventricular fibrillation or tachycardia), or cardiac death. Hemodynamic changes were determined by comparing the heart rate at onset of an ischemic episode to the median heart rate 15 min prior to onset.

Results. Postoperatively, 47% of patients developed ischemia *vs.* 16% preoperatively and 11% intraoperatively prebypass. Postoperative ischemia was most common in the early period (postoperative day 0-2, 39% of patients), peaking during the first 2 h after revascularization, but relatively uncommon during the late postoperative period (postoperative day 3-7, 19% of patients). All postoperative episodes were asymptomatic: only 9 of 54 early episodes were detected by clinical ECG monitoring. Only 43% of the postoperative episodes were preceded by a > 20% increase in heart rate. However, tachycardia persisted throughout the postoperative week (25-36% of all heart rates ≥ 100 beats/min), and patients with postoperative ischemia had significantly more tachycardia (42% *vs.* 12%, $P < 0.03$). Five adverse cardiac outcomes occurred on the day of surgery, all 5 preceded by postoperative ischemia, 3 by intraoperative prebypass ischemia, and none by preoperative ischemia. Patients with late postoperative ischemia did not have an adverse cardiac outcome.

Conclusion. We conclude that monitoring for myocardial ischemia beyond the first 2 days after CABG surgery may not be necessary in most patients. Instead, we should focus resources on the early postoperative period, during which myocardial ischemia is most prevalent. Characteristically, early postoperative ischemia is silent, difficult to detect using clinical ECG monitoring, and may be related to chronically elevated heart rate.

References. 1. Leung JM, *et al:* Anesthesiology 71:16-25, 1989

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TITLE: INDEPENDENT SIMULTANEOUS MEASUREMENT OF OXYGEN CONSUMPTION AND DELIVERY DURING NORMOVOLEMIC HEMODILUTION

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Studies of hemodynamic changes during normovolemic hemodilution (NVH) have established that the reduction in hematocrit and arterial oxygen content (CaO_2) are not deleterious since compensating mechanisms such as increase in venous return and cardiac index (CI) maintain systemic oxygen transport and tissue oxygenation (1). At normal levels of oxygen delivery (DO_2) oxygen consumption (VO_2) remains constant and independent of delivery (2). However, in most studies, the same measurements of CI and CaO_2 were used to calculate both VO_2 ($CIx10x[CaO_2\text{-}CvO_2]$) and DO_2 ($CIx10xCaO_2$). The aim of this study was to evaluate the DO_2/VO_2 relationship during NVH with simultaneous independent measurements.

Nine unpremedicated patients scheduled for abdominal aortic surgery were studied with their informed consent after institutional approval. All patients were investigated prior to surgery while in the supine position and breathing room air. A radial and a pulmonary artery catheter were inserted and the CM_5 ECG lead monitored. Patients were connected to a Beckman Metabolic Measurement Cart via an anesthetic face mask to get a continuous measurement of minute ventilation (V_E), oxygen uptake (VO_2resp), carbon dioxide output (VCO_2), respiratory exchange ratio (RQ) and end-tidal pCO_2. After a resting period of 30 min (control), NVH was performed until hematocrit reached 30%. The volume of blood withdrawn was simultaneously replaced by the same volume of 4% albumin. Six sets of measurements were carried out before, during and after NVH, including blood temperature, standard hemodynamics, arterial and mixed venous oxygen contents (CaO_2 and CvO_2) and arterial lactate concentrations. VO_2 was derived both from the Fick equation and from continuous expired gas measurements. The Wilcoxon test for paired values was used for the statistical analysis. All values are expressed as means±SD.

Relevant results are presented in Table 1.

Table I	hematocrit %	CI l/min/m2	CaO2 ml/100ml	CvO2 ml/100ml	DO2 ml/min/m2	VO2 Fick ml/min/m2	VO2 resp ml/min/m2	O2-extr. %
control	37.7±2.0	3.70±0.83	15.70±0.94	11.72±0.83	576±118	142.2±23.5	134.3±16.9	25.4±4.0
NVH	28.6±1.7	5.33±1.13	12.09±0.59	8.46±0.54	639±119	166.0±41.8	140.3±24.3	29.8±5.4
p-value	0.0004	0.0047	0.0004	0.0004	0.2893	0.002	0.3772	0.034

V_E, VO_2 and VCO_2 measured from expired gases, paO_2, $paCO_2$ and lactate concentration remained unchanged during NVH. The increase in CI compensated for the decrease in CaO_2 so that DO_2 remained constant. Since ($CaO_2\text{-}CvO_2$) was unchanged the increase in CI resulted in an increase in VO_2 calculated by the Fick equation. It is suggested that VO_2 calculated from thermodilution cardiac output might not reliably reflect the metabolic changes which may occur during NVH. This study also demonstrated that oxygen extraction ratio, $O_2extr=(CaO_2\text{-}CvO_2)/CaO_2$, increased during NVH although DO_2 was not decreased. This might be explained by a better distribution of blood flow at the microcirculatory level due to decreased blood viscocity which facilitiates peripheral oxygen unloading (3).

References

1. Messmer K, et al: Eur Surg Res 18:254, 1986.
2. Schumacker PT & Cain SM: Intensive Care Med 13:223, 1987.
3. Vicaut E, et al: Int J Microcirc: Clin Exp 6:225, 1987.