

TITLE: DOES THE CHOICE OF ANESTHETIC HAVE AN EFFECT ON MYOCARDIAL ISCHEMIA DURING ANESTHETIC INDUCTION & INTUBATION?

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Introduction. The anesthetic induction period has always been considered to be stressful. Previous reports cited that myocardial ischemia occurred frequently during laryngoscopy and intubation.^{1,2} Considerable controversy exists regarding the potential of isoflurane to cause myocardial ischemia in patients with coronary artery disease. We therefore examined the incidence of myocardial ischemia under isoflurane (F) and sufentanil (S) anesthesia during this potentially stressful period in a cohort of high-risk patients using continuous electrocardiography.

Methods. After institutional approval and informed consent was obtained, 164 men scheduled for elective coronary artery bypass graft surgery were randomized to receive F or S. 54 received F and 110 received S (5-10ug/kg). All patients were monitored with continuous ECG (Holter) using leads CC5 and CM5. Holter tapes were read by two independent blinded investigators. An ischemic episode was defined as an ST-segment shift from baseline of ≥ 0.1 mV depression at J+60msec, or ≥ 0.2 mV ST elevation at the J-point, lasting for at least one minute. HR and systolic BP were rigorously controlled within $\pm 20\%$ of preoperative baseline. Systolic, diastolic BP, and HR were measured continuously on entry into the operating room and entered into a microcomputer using a digital interface system.

Results. Hemodynamics were well controlled during the induction and intubation periods. Overall, only 1.2% of patients had HR > 100 beats/min, 6.7% of patients had SBP > 180 mm Hg, and 0.6% of patients had DBP > 90 mm Hg. The incidence of tachycardia and hypertension was similar between the 2 anesthetic groups (table 1). 5/164 (3%) patients developed ECG evidence of ischemia during this period. 1/54 (1.9%) patients receiving F developed ischemia vs. 4/110 (3.6%) patients receiving S, $P = NS$. HR, systolic and diastolic BP were within $\pm 20\%$ of pre-induction values in 4/5 patients who developed ischemia. The fifth patient, who received S, developed ischemia and had a HR increase of 49% above pre-induction value at intubation.

Table 1 Incidence of hemodynamic abnormalities

	Isoflurane	Sufentanil
HR > 100 beats/min	1/54 (1.9%)	1/110 (0.9%)
SBP > 180 mmHg	4/54 (7.4%)	7/110 (6.4%)
DBP > 90 mmHg	0/54 (0%)	1/110 (0.9%)

Discussion. In contrast to previous studies, we found that even in high-risk patients, the incidence of myocardial ischemia is low during anesthetic induction and intubation when hemodynamics are rigorously controlled. The use of isoflurane was not associated with an increased incidence of myocardial ischemia during this potentially stressful period.

References

1. Roy WL, Edelist G, Gilbert: Anesthesiology 51:393-397, 1979
2. Slogoff S, Keats AS: Anesthesiology 62:107-114, 1985

TITLE: THE EFFECTS OF ANESTHETICS AND STEAL-PRONE ANATOMY ON MYOCARDIAL ISCHEMIA

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Introduction. It has been suggested that isoflurane may cause maldistribution of coronary blood flow in patients (pts) with steal-prone anatomy (1). To investigate this, we prospectively evaluated the potential of isoflurane in producing myocardial ischemia in high-risk pts with and without steal-prone anatomy, using continuous ECG and transesophageal echocardiography (TEE), under strict hemodynamic control.

Methods. After institutional approval and informed consent, 80 men scheduled for elective CABG surgery were randomized to receive isoflurane (F) or sufentanil (S). 29 received F and 51 received S (5-10ug/kg followed by an infusion rate of 0.07ug/kg/min). Following tracheal intubation, the TEE probe was maintained at the level of the papillary muscles (short axis view). Echo data were continuously recorded until the onset of bypass. The TEE data were analyzed by two blinded investigators using: grade 0=normal, 1=mild hypokinesis, 2=severe hypokinesis, 3=akinesis, 4=dyskinesis. Each pt's best prebypass image was used as his baseline, and a TEE ischemic episode was identified if any wall worsened ≥ 2 grades. Pts also were monitored with continuous ECG (Holter) using leads CC5 and CM5. Holter tapes were read by 2 independent blinded investigators. An ischemic

episode was defined as an ST-segment shift from baseline of ≥ 0.1 mV depression at J+60msec, or ≥ 0.2 mV ST elevation at the J-point, lasting for at least one minute. HR and systolic BP were rigorously controlled within $\pm 20\%$ of preoperative baseline. Systolic, diastolic BP, and HR were measured continuously during the prebypass period. All pt's angiograms were analyzed for evidence of steal-prone anatomy: defined as total occlusion in one coronary artery which is supplied distally by collaterals from another coronary artery with a $> 50\%$ stenosis (1).

Results. Hemodynamics were well controlled [\uparrow HR ($> 20\%$ of baseline)=6.9%, \uparrow SBP=6.9%, \downarrow SBP=8.3% of total time] with no significant difference between anesthetics. 44% (35/80) of pts had steal-prone anatomy. The incidence of ischemia was similar in pts with steal-prone anatomy (34%=12/35) vs. those without (36%=16/45). The incidence of ischemia in the F group (34%=10/29) was similar to that in the S group, (35%=18/51). In the F group, 41% (12/29) of pts had steal-prone anatomy, with 5 (42%) of these developing ischemia by ECG or TEE. In the S group, 45% (23/51) had steal-prone anatomy, with 7 (30%) developing ischemia ($P=NS$ vs. F group).

Discussion. Under strict hemodynamic control, neither the presence of steal-prone anatomy, nor the choice of anesthetic affected the incidence of intraoperative myocardial ischemia. Although these data also suggest that in patients with steal-prone anatomy, isoflurane anesthesia does not appear to confer additional risk, these data are preliminary and further studies are warranted.

References

1. Buffington et al.: Anesthesiology 69:721-727, 1988