Title: EFFECTS OF ANESTHETICS ON THE REGIONAL MYOCARDIAL FUNCTION OF THE ISCHEMIC HEART AS ASSESSED BY QUANTITATED TRANSESOPHAGEAL ECHOCARDIOGRAPHY

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Introduction. The role of anesthetic agents in altering the distribution of coronary blood flow in coronary artery disease (CAD) is controversial. Most studies of this subject have utilized only global measures of adequacy of myocardial perfusion. Transesophageal echocardiography (TEE) can illustrate changes in regional myocardial function (RMF) that are sensitive measures of regional perfusion. Methods of assessing RMF using motion of the endocardial edge are influenced by translational and rotational movement of the heart. By including the epicardial edge and measuring systolic wall thickening (SWT), these artifacts are avoided, providing a more accurate definition of RMF. This study was designed to investigate the effects of several anesthetics on TEE-derived SWT in CAD.

Methods. 15 patients undergoing elective coronary artery bypass were enrolled after obtaining informed consent, and were randomly assigned to receive sufentanil (SUF), halothane (HAL), or isoflurane (ISO) (5 patients each). Arterial and pacing pulmonary artery catheters were placed prior to induction. General anesthesia was induced using etomidate (0.3 mg/kg) and fentanyl (2 μg/kg). Succinylcholine (1-1.5 mg/kg) was used to provide relaxation. Ten minutes after tracheal intubation and placement of the TEE probe, baseline (BASE) TEE images were obtained in the minor axis view at mid-papillary muscle level. Hemodynamic data, including heart rate (HR), cardiac output (CO), mean arterial (MAP) and pulmonary capillary wedge pressures (PCWP) were recorded. HAL or ISO were then administered at end-tidal levels of 0.5, 1.0, and 1.5 MAC (LVLs 1,2,3). In a corresponding fashion, SUF was given by a computerized pharmacokinetic model-driven infusion device to predicted plasma levels of 1, 2, and 4 ng/ml. Decreases in heart rate or arterial pressure as a result of anesthetic administration were corrected to baseline values by atrial pacing or 1-2 μg/kg boluses of phenylephrine, respectively. After five minutes of stabilization at each LVL, with HR and MAP changes corrected to BASE, TEE images were recorded. A custom-designed computer system was implemented to measure SWT between end-diastolic (ED) and end-systolic (ES) TEE images using a centerline algorithm. 100 evenly distributed wall thickness chord segments were derived around the circumference of the minor axis, beginning in each image at the posterior papillary muscle. SWT was expressed as the percent change in chord length from ED to ES. SWT at the anesthetic LVLs were compared to those at BASE, and the number of chords with significant ↓SWT noted. 20 contiguous chords with ↓SWT were considered to represent significant ↓RMF. The coronary angiographic data for each patient were examined to determine if an area of ↓RMF corresponded to a vascular bed that was supplied by a critically stenosed coronary artery and at increased risk for ↓RMF due to maldistribution of coronary perfusion. An "at risk" region (RISK) was defined as the quadrant (antero-/posteroseptal, antero-/posterolateral) supplied by the most stenosed coronary artery.

Results. Table 1 lists hemodynamic data. There were no changes in HR or MAP between BASE and any LVL for any anesthetic. SUF caused no change in PCWP or CO at any LVL. HAL caused ↑PCWP at LVL 3 and ↓CO at LVLs 2 and 3. ISO caused ↑PCWP at LVL 3 and no change in CO. Table 2 shows the incidence of ↓RMF in any region. More ↓RMF was found in ISO and HAL groups than with SUF. A similar percentage of ↓RMFs occurred in RISK regions for both HAL (4 of 7) and ISO (2 of 3) (Table 3).

Discussion. The noted hemodynamic changes are in keeping with the accepted effects of the drugs studied: decreased contractility by HAL, far less by ISO, and minimal effects of SUF. ↓RMF can be caused not only by negative inotropism, but also by new ischemia resulting from coronary flow maldistribution, which should be more prevalent in RISK regions. In this small number of patients, ↓RMFs occurred in RISK regions in similar proportions with each anesthetic. Clearly, a larger sample size will be required in order to draw firm conclusions regarding a distinction in these drugs' ability to affect the distribution of coronary blood flow. We conclude that our technique of measuring SWT as a quantitative determination of RMF is an objective and useful means of assessing the effects of interventions on cardiac function.

Table 1. Hemodynamic Data (mean ± S.D.)

		BASE	LVL 1	LVL 2	LVL 3
HR (/min)	SUF	68±12	66±12	66±12	66±12
	HAL	55± 6	55± 9	55± 9	55± 7
	ISO	69±14	69±15	70±14	70±14
MAP (torr)	SUF	80± 7	82± 5	83± 6	83± 5
	HAL	84± 6	88± 4	88± 7	88± 2
	ISO	80±12	80±13	80±11	85±12
PCWP (torr)	SUF	12± 2	11± 2	11± 2	11± 2
	HAL	13± 5	13± 3	14± 3	17± 6*
	ISO	8± 3	9± 4	10± 3	12± 4*
CO (L/min)	SUF	5.1±0.4	4.6±0.6	4.3±0.7	4.3±0.9
	HAL	4.0±0.6	3.5±0.7	3.3±0.7*	2.9±0.8*
	ISO	4.9±1.8	4.4±0.7	4.4±1.0	4.4±1.2

*$p < .05$ compared to BASE

Table 2. # ↓RMF - Total

	LVL 1	LVL 2	LVL 3
SUF	0	1	0
HAL	1	3	3
ISO	0	1	2

Table 3. # ↓RMF - RISK Regions

	LVL 1	LVL 2	LVL 3
SUF	0	1	0
HAL	0	2	2
ISO	0	0	2

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