REPORTS OF SCIENTIFIC MEETINGS

John J. Downes, M.D., Editor

Society of Cardiovascular Anesthesiologists 11th Annual Meeting April 16–19, 1989 Seattle, Washington

The 11th Annual Meeting of the Society of Cardiovascular Anesthesiologists was held in Seattle, Washington, April 16–19, 1989.

The workshop on transesophageal two-dimensional echocardiography the subject of some excellent scientific papers. Acampora *et al.* (Sloan-Kettering) reported the use of 2DTEE to study load-independent relationships that represent left ventricular contractility in 33 healthy patients receiving isoflurane after anesthetic induction with an opioid N_2O muscle relaxant technique. Plotting end-systolic meridional wall stress versus velocity of circumferential shortening corrected for heart rate provides a description of contractility that nullifies loading conditions. Measurements were made following anesthetic induction, after administration of isoflurane to an 0.8% end-tidal concentration, and following return to 0% end-tidal isoflurane. Isoflurane caused a downward and leftward shift of the plotted relationship indicative of diminished contractility. Following discontinuation of isoflurane, data returned to baseline values. In a separate paper, Acampora *et al.* reported use of the same load-independent relationships to determine the effect of esmolol on ventricular contractility during nitroprusside-induced hypotension in six patients undergoing retroperitoneal node dissection. Data were tabulated following induction with N_2O-fentanyl-pancuronium, with induction of hypotension using sodium nitroprusside, 20 min after addition of esmolol (200 $\mu g \cdot kg^{-1} \cdot min^{-1}$), and 20 min after addition of esmolol (400 $\mu g \cdot kg^{-1} \cdot min^{-1}$). Results indicate that esmolol, particularly at 400 $\mu g \cdot kg^{-1} \cdot min^{-1}$, impairs myocardial performance even when afterload is reduced by sodium nitroprusside.

Lasker *et al.* (Mount Sinai) compared ejection fraction determined by using 2DTEE in patients undergoing myocardial revascularization with a measure of maximal aortic blood flow acceleration obtained by placing a 3-mHz Doppler probe in the patient's suprasternal notch. They found a significant correlation between ejection fraction and maximal aortic blood flow acceleration.

Cardiac effects of inhalation anesthetics were described by a number of investigators. Neutsch *et al.* (University of Michigan) studied the *in vivo* effects of halothane, enflurane, and isoflurane on arrhythmic potential in a canine model of chronic infarction. Excitation thresholds, relative and effective refractory periods, and intraventricular conduction times were determined during 1.1 and 1.8 MAC of each anesthetic given (in random order) on days 4, 5, and 6 after infarction. Halothane and enflurane increased effective refractory periods without significantly prolonging intraventricular conduction in both normal and infarcted zones. Isoflurane increased refractoriness in normal zones only.

Bollen *et al.* (Stanford) examined the ability of halothane *versus* isoflurane to relax previously constricted isolated porcine coronary segments as a function of vessel diameter. They found that halothane (1%, 2%, and 3%) caused significant relaxation in previously constricted 0.5–1-mm pig endocardial coronary arteries, whereas only 3.0% isoflurane caused significant relaxation. For 1–1.5-mm coronaries, halothane (2% and 3%) caused significant relaxation, whereas isoflurane did not cause relaxation at any concentration studied. Based on their study, the authors concluded that for isoflurane to cause greater coronary dilation than halothane, as is generally believed, the dilation must be occurring in vessels distal to those that are 0.5 mm in diameter. In a separate study, the same authors presented evidence that halothane was able to relax previously constricted human epicardial coronary artery segments (obtained from recipient patients' hearts at the time of cardiac transplantation) at lower concentrations and to a greater extent than isoflurane. Based on their results and those of other investigators, they concluded that isoflurane-induced coronary artery dilation must be occurring distal to large epicardial arteries. However, they also concluded that for isoflurane to have a greater vasodilatory action than halothane on small *versus* large coronary arteries, these two anesthetics must have different mechanisms of action.

Levy *et al.* (Emory) reported heparin dose responses before and after cardiopulmonary bypass. In 20 patients they determined baseline ACT prior to cardiopulmonary bypass and constructed heparin dose-response curves before cardiopulmonary bypass and after administration of protamine. They concluded that heparin dose responses for anticoagulation do not significantly change following protamine reversal. This is important to know for the hemodynamically unstable patient who requires reheparinization after protamine reversal.

Kuitunen *et al.* (Helsinki University) obtained plasma heparin concentrations, ACT, and thromboelastographic measurements preoperatively, before protamine, and at intervals up to 6 h after protamine in 14 patients receiving protamine chloride and 16 patients receiving protamine sulfate after cardiopulmonary bypass for coronary artery bypass grafting. They found that the two protamine salts are equally effective antidotes to heparin after cardiac surgery. The doses derived from the heparin-ACT response curves failed to prevent the appearance of small amounts of free heparin, which peaked 2 h after protamine. There was no correlation between blood loss and peak heparin-rebound level.

Earl Wynands was inducted as the new president of the Society. The 1990 meeting is scheduled for May 13–16 in Orlando, Florida.

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