TITLE: THE EFFECTS OF KETAMINE AND THIOPENTAL ON MYOCARDIAL CONTRACTILITY AND FUNCTION IN HYPOVOLEMIC SWINE

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Anesthesia induction in the traumatized, hypovolemic patient is difficult because of the need to balance adequate anesthesia with hemodynamic stability. Thiopental can cause myocardial depression and vasodilation. Ketamine has been advocated because of its sympathomimetic action. Ketamine can also be a myocardial depressant. This model assesses the effects of ketamine and thiopental on hemodynamic parameters and myocardial contractility, (end systolic elastance (Σ es) using end-systolic pressure-dimension relationships (ESPDR) (Fig. 1) and pressure-dimension (PD) loops. (Fig. 2) The direct myocardial effects (cardiodynamics) of these agents have not been evaluated using the ESPDR and PD loops. This study was designed to examine the hemodynamic and cardiodynamic effects of ketamine and thiopental in a hypovolemic model.

Under an approved animal use protocol, 16 Yorkshire swine weighing 15-25 kg were anesthetized with halothane in a 70% N_2O/O_2 mixture, intubated, and mechanically ventilated. PAP, PCWP, CVP, and CO were measured and the ECG was monitored. An occluder was placed around the inferior vena cava (IVC) to vary preload. Two sonomicrometer crystals were placed in the A-P dimension of the left ventricle and a 5mm Konigsberg pressure transducer was placed through the apex. Halothane was discontinued. PD loops were obtained while occluding the IVC. Σ es was calculated as the slope of the linear-regression line generated through the series of end-systolic pressure-dimension points during IVC occlusion. Baseline control (BC) cardiodynamic and hemodynamic parameters were obtained. Animals were then hemorrhaged to a MAP of 40 mgHg and allowed to stabilize for 15 minutes to simulate trauma with hypovolemia. New measurements were then made at this hemorrhage control state (H). Thiopental or ketamine, 6 mg/kg was given as an IV bolus. Replicate measurements were made at 1, 5, 15, and 30 minutes (H1, H5, H15, H30, respectively). Data was analyzed with repeated a measures ANOVA and a Fischer least-significant-difference test for paired comparisons. A $p<0.05$ was considered statistically significant.

Thiopental significantly depressed Σ es at H1 and H5 and significantly increased end-diastolic dimension (Ded), end-systolic dimension (Des), PCWP, PVR, and MPAP. Thiopental did not significantly alter CO or SVR. Ketamine did not significantly alter es but increased Ded, Des, SVR, and PVR. Ketamine significantly decreased CO at H1. Both ketamine and thiopental altered the position of the PD loops at BC, H, and H1 in similar fashion (Fig. 2).

The results indicate that ketamine and thiopental depress myocardial function in this model but thiopental appears to be more of a myocardial depressant. Σ es, a load-insensitive index of contractility, and PD loops allow assessment of anesthetic effects on myocardium coupled to the vascular system. To our knowledge ESPDR or PD loops have not been used to evaluate effects of these agents in a hypovolemic model.

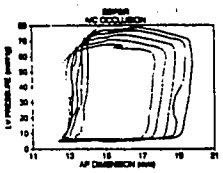

Figure 1

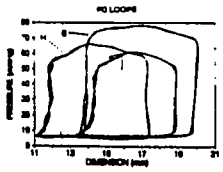

Figure 2

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TITLE: EFFECTS OF DOBUTAMINE ON HEPATIC BLOOD FLOW AND OXYGEN BALANCE DURING ANESTHESIA OF LIVER SURGERY

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The purpose of this study is to clarify whether dobutamine (DOB) can improve the hepatic blood flow and oxygen balance during hepatic surgery.

Fourteen patients (Age 57 ± 9 yr) undergoing hepatic surgery were used after institutional approval and informed consent. While surgical procedure was interrupted prior to lobectomy, systemic and hepatic hemodynamic variables were measured and radial, hepatic and pulmonary arterial and portal venous blood samples were taken for gas analysis. Hepatic arterial (HA) and portal venous (PV) blood flows were measured by ultrasonic flowmeter (Transonic Systems Inc.). After 10 min of the start of DOB infusion (3 μg/kg min), the same measurements were performed. The results were expressed as mean ± SEM. For statistical analysis of the data, paired t-test was used, and p value < 0.05 was considered significant.

Table 1-4 summarized blood flow, oxygen balance, and their systemic and hepatic distribution at pre and during DOB administration. While PV flow increased during DOB administration, both hepatic oxygen uptake and transport were increased, and hepatic oxygen balance did not change. HA to systemic flow ratio (H/S flow), PV to systemic flow ratio (P/S flow) and hepatic to systemic oxygen uptake ratio (H/S O_2) were not changed by DOB administration.

Table 1. Blood flow (l/min m^2)

	Systemic	HA	PV
Pre DOB	3.52 ± 0.32	0.15 ± 0.02	0.43 ± 0.03*
During DOB	4.29 ± 0.29*	0.20 ± 0.04	0.56 ± 0.04*

* $p<0.05$ vs pre DOB value, n = 14

Table 2. Oxygen uptake (ml/min m^2)

	Systemic	Hepatic
Pre DOB	78.5 ± 5.3	13.7 ± 3.3*
During DOB	90.9 ± 6.5	22.4 ± 6.1*

* $p<0.05$ vs pre DOB value, n = 14

Table 3. Oxygen transport (L/min m^2)

	Systemic	Hepatic
Pre DOB	533 ± 47	78 ± 5*
During DOB	667 ± 52	108 ± 8*

* $p<0.05$ vs pre DOB value, n = 14

Table 4. Hepatic and systemic distribution

	H/S flow	P/S flow	H/S O_2
Pre DOB	.046 ± .008	.132 ± .014	.166 ± .004
During DOB	.052 ± .010	.135 ± .041	.210 ± .077

* $P<0.05$ vs pre DOB value, n = 14

Reference.
1. Kainuma et al. Anesthesiology 65:A383, 1989