TITLE: OXYGENATION OF THE PORCINE GUT IS REDUCED BY HALOTHANE BUT IS MAINTAINED BY KETAMINE

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Episodes of non-occlusive splanchnic ischemia have been reported to occur during anesthesia with several agents (1). In the present study, we evaluated the effects of ketamine and halothane on the oxygenation of the gut.

METHODS: Pittman-Moore mini-pigs were chronically instrumented with mesenteric artery flow probe, Swan-Ganz, arterial, and portal catheters. Intraluminal pCO_2 of the ileum was measured using silicone balloon catheters. With the concomitantly determined arterial HCO_3 concentration, the intestinal intra-mucosal pH was calculated using the Henderson-Hasselbalch equation (2). Group K (n=6) received ketamine (20mg/kg, iv) and group H (n=6) received halothane. Data were obtained at baseline and during anesthesia (10 minutes after the ketamine bolus or at 1.5% endtidal concentration of halothane). Two way analysis of variance was used for statistical evaluation.

RESULTS: 1) Systemic oxygen delivery (sDO_2) decreased significantly in both groups but group H was significantly lower than K. 2) Mesenteric oxygen delivery (mDO_2) decreased significantly in group H, but was not altered in group K. 3) Systemic oxygen consumption (sVO_2) decreased significantly in group H but did not change in group K. 4) Mesenteric oxygen consumption (mVO_2) was significantly decreased in group H while it remained unchanged in group K. 5) Intestinal mucosal pH (pH_i) showed no change within or between the groups.

	Ketamine		Halothane	
	Baseline	Anesth.	Baseline	Anesth.
sDO_2(ml/min)	298±42	232±44*	291±20	157±12*#
mDO_2(ml/min)	34±7	29±7	32±4	13±2*#
sVO_2(ml/min)	110±16	89±23	123±15	15±5*#
mVO_2(ml/min)	14±4	12±2	12±2	6±1*#
pH_i	7.0±0.2	6.8±0.1	7.0±0.2	6.9±0.1

*:p<0.05 from baseline, #:p<0.05 between groups.

CONCLUSION: Halothane anesthesia significantly decreases both systemic and mesenteric oxygen delivery and consumption. This reduction does not cause a significant fall in the intramucosal pH of the healthy intestine, but it may produce anaerobic metabolism in the critically hypoxic gut. Ketamine anesthesia maintains mesenteric oxygen delivery and consumption and may therefore be advantageous to halothane under hypoxic conditions.

REFERENCES: 1) Intestinal Circulation during Inhalation Anesthesia, Anesth. 52:462-469, 1989. 2) Intramucosal pH Measurement with Tonometers for Detecting Gastrointestinal Ischemia in Porcine Hemorrhagic Shock, Circ. Shock 29:319-327 (1989).

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Title: Dobutamine and Norepinephrine Treatment in Relation to Oxygen Delivery and Consumption in Endotoxin Shock.

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Introduction: In septic shock the effects of adrenergic agents on oxygen delivery (DO_2) and oxygen consumption (VO_2) remains controversial. Dobutamine (DOB) could increase cardiac output (CO) and hence DO_2, but low systemic vascular resistance leading to low arterial pressure can rather require vasopressors such as dopamine or norepinephrine (NE). We studied the effects of DOB and NE administration on hemodynamics, DO_2 and VO_2 in an experimental model of endotoxic shock.

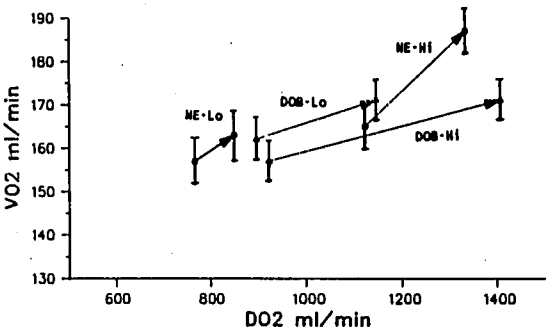
Methods: The study included 12 mongrel dogs (22 ± 5 kg) anesthetized with 25 mg/kg of pentobarbital, intubated and mechanically ventilated with room air. Endotoxic shock was induced by the slow injection of 2 mg/kg of Escherichia coli endotoxin. After a 30 min observation period, fluid challenge using 0.9% saline was performed during 30 min to restore baseline filling pressures. During the experiment fluid administration was adjusted to maintain baseline filling pressures. The protocol was randomized for the first agent (DOB/NE) and dose (Hi/Lo). The first agent was administered for 20 min followed by a drug-free interval of 30 min. Then the second agent was started using the corresponding dose. After the following drug-free interval the first agent was again administered using the other dose. The first six dogs were randomized to a protocol using DOB 5 vs. NE 0.1 and DOB 10 vs. NE 0.2 mcg/kg/min. The other six dogs were randomized to the same protocol but using different dosages of NE (0.5 and 1.0 mcg/kg/min). Measurements were taken before and after 20 min of drug administration.

Results: Analysis were performed on the grouped results of NE 0.1 and 0.2 mcg/kg/min (low dose) or NE 0.5 and 1.0 mcg/kg/min (high dose) together with DOB low dose (5 mcg/kg/min) or high dose (10 mcg/kg/min). Results were (mean ± S.D.):

	NOREPI LO (n=12)		NOREPI HI (n=12)		DOBUTAMINE LO (n=12)		DOBUTAMINE HI (n=12)	
	T = 0	T = 20	T = 0	T = 20	T = 0	T = 20	T = 0	T = 20
HR	159 ±9	160 ±10	187 ±21	197 ±29	173 ±24	190 ±19*	179 ±30	210 ±15*
MAP	86 ±12	101 ±16*	96 ±25	106 ±26&	89 ±21	100 ±21*	83 ±22	100 ±26*
CO	4.5 ±1.4	4.9 ±1.5	6.5 ±2.2	7.6 ±1.6*	5.1 ±2.0	6.4 ±1.8*	5.5 ±1.8	8.0 ±1.4*
SVR	1599 ±572	1731 ±577	1202 ±339	1106 ±370	1476 ±549	1278 ±379	1227 ±413	992 ±354*
RVSW	6.0 ±1.8	6.9 ±1.7&	6.7 ±2.9	8.8 ±1.9&	5.3 ±3.1	7.1 ±2.6*	6.8 ±3.1	7.2 ±2.2*
LVSW	51.1 ±9.5	59.4 ±11.1&	63.8 ±19.6	55.1 ±18.7*	54.5 ±16.5	64.4 ±16.8*	53.2 ±15.7	50.1 ±16.5*
DO_2	767 ±356	851 ±349	1124 ±428	1335 ±332*	898 ±411	1147 ±427*	924 ±370	1408 ±322*
VO_2	157 ±49	163 ±69	165 ±38	187 ±40*	162 ±43	171 ±44*	157 ±38	171 ±41*
ER	21.8 ±6.1	21.2 ±5.4	19.5 ±8.9	15.8 ±4.2	21.3 ±6.6	16.8 ±3.9*	20.5 ±7.5	15.0 ±4.1*

T=0 vs. T=20: & = p < 0.05 * = p < 0.01

The decrease in ER during dobutamine (low and high dose) was significantly higher when compared to low dose norepinephrine (p <.05)



Conclusion: In an endotoxic shock model oxygen extraction is better preserved during norepinephrine than during dobutamine infusion. On the other hand dobutamine infusion results in a more consistent increase in DO_2 and VO_2.