TITLE: ACUTE HYPOTENSION IMMEDIATELY FOLLOWING RAPID HYPERTONIC SALINE INFUSION IN ANESTHETIZED DOGS

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Small volumes of 7.5% hypertonic saline (HNaCl) have been shown to improve cardiovascular and metabolic function in various animal species subjected to severe hypovolemic shock (1,2). In recent clinical trials, HNaCl was reported to effectively restore blood pressure, pH and urine output in severely injured patients (3). These studies suggest that HNaCl may be a promising therapeutic intervention for resuscitation in patients with trauma, hemorrhage or cerebral injuries. Nevertheless, when infused rapidly into anesthetized animals (< 2 min) HNaCl caused an immediate and severe hypotension prior to the restoration of cardiovascular function. The present study examined the hypothesis that the early hypotension produced by HNaCl was mediated through an acute and transient depression of cardiac contractility.

Methods: Left ventricular pressure, diameter (D) and wall thickness (WT) were monitored continuously in 10 halothane anesthetized dogs. Cardiac output (CO) and coronary blood flow (CBF) were measured from flow probes placed around the ascending aorta and the left anterior descending coronary artery. HNaCl was infused intravenously at 3 ml/kg in 1 min. Physiological measurements were recorded continuously for subsequent computer analysis.

Results: Mean arterial pressure decreased by 49% (from 95±4 to 51±5 mmHg) at 45 sec after the onset of HNaCl infusion. CO, CBF and LVEDP increased from 2.8±1.0 to 3.5±1.1 l/min, 23.7±5.3 to 39.8±4.7 ml/min and 3.5±.6 to 8.8±.8 mmHg respectively; heart rate did not change. Percents of systolic shortenings of D and WT increased from 5.6±0.5 to 7.3±0.5 and 13.9±0.6 to 15.1±1.2 indicating improved cardiac contractility. Systemic and pulmonary vascular resistances decreased by 60 and 27% respectively. The fall in arterial pressure was short-lived (106±9 sec) and persisted despite pharmacological or nerve blockade.

Discussion: We reject our hypothesis; the results showed conclusively that the acute hypotension caused by the rapid HNaCl infusion was not mediated through a decrease in cardiac contractility. Furthermore, the data strongly suggest that a direct mechanism is involved in the potent vasodilation evoked by HNaCl. We conclude that HNaCl should be given slowly and with caution, particularly when hypovolemic shock is severe, in order to avoid or minimize any detrimental consequences of the acute hypotension associated with rapid infusion.

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TITLE: ENFLURANE PRESERVES RENAL BLOOD FLOW

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INTRODUCTION: Decreased or unchanged renal blood flow (RBF) during enflurane anesthesia (EA) has been reported [1-4]. Data on other aspects of renal function during EA is sparse[1,5]. In an attempt to assess the influence of enflurane on RBF and renal function and also to separate anesthetic effects from the influence of surgical stress on renal function we studied these phenomena in chronically instrumented dogs.

METHODS: Seven female mongrel dogs weighing 18-24 kg were used. Catheters were inserted into the descending aorta, the left renal vein and the urinary bladder via a laparotomy. A doppler flow probe was positioned around the left renal artery. The dogs were allowed to recover for at least two weeks. Awake control measurements of RBF, inulin clearance (GFR), mean arterial pressure (MAP), ABG's, urine flow rate (UF), sodium and osmolar excretions were obtained. Anesthesia was then induced via inhalation of enflurane in N_2O/O_2 and maintained with enflurane in O_2. Anesthetic levels of 1, 1.5 and 2 MAC as assessed from end tidal concentrations (masspectrometry) were used in random fashion. After achieving steady state the above mentioned measurements were repeated. In three dogs where MAP was 50 mmHg or lower at 1.5 MAC the anesthetic depth was not increased further. Control studies of renal function after anesthesia were performed within one week. ANOVA was used for statistical analysis.

RESULTS: MAP decreased as expected with increasing anesthetic depth. RBF was unchanged from awake controls at all levels of anesthesia. Renal vascular resistance (RVR), GFR, UF, sodium and osmolar excretions all decreased with increasing depths of anesthesia. (Table).

DISCUSSION: RVR decreases in parallel with anesthetic depth such that RBF is maintained despite decreased perfusion pressure. This seems to indicate that enflurane does not interfere with renal autoregulation.

TABLE

x ± SEM		MAP mmHg	RBF ml/min	GFR ml/min	UF ml/min	RVR mmHg/ml/min
Control	n	101 ± 7	167 ± 28	131 ± 32	1.75 ± 21	0.60 ± 0.08
1 MAC	7	71 ± 3[a]	200 ± 41	87 ± 15[a]	0.49 ± 0.08[b]	0.35 ± 0.11[b]
1.5 MAC	7	55 ± 4[b]	178 ± 22	63 ± 17[a]	0.19 ± 0.06[b]	0.30 ± 0.06[b]
2 MAC	4	50 ± 3[b]	166 ± 19	29 ± 9[b]	0.14 ± 0.04[b]	0.30 ± 0.03[b]
Postanesth.	7	98 ± 6	155 ± 31	122 ± 23	1.93 ± 0.35	0.65 ± 0.09

a = p < 0.05 from control
b = p < 0.01 from control

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