Title

: RENAL FUNCTION AND HEMODYNAMICS DURING INDUCED HYPOTENSION IN DOGS

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Introduction. The deleterious effect of hemorrhagic hypotension on kidney function is well known. We have evaluated the effects of sodium nitroprusside (SNP), trimethaphan (TMP), and hemorrhagically induced (hemo) hypotension on glomerular filtration rate (GFR), effective renal plasma flow (ERPF), sodium reabsorption (TNa), renal venous PO2 (Trv 2), urine PO2 (PuO2) and urine flow (Vij) in three groups of dogs anesthestized with halothaneoxygen anesthesia. Percent changes of each variable from control level were determined during hypotension and after restoration of blood pressure to normal level.

Methods. Anesthesia was induced with thiopental 4-6 mg kg, the trachea was intubated and ventilation was controlled to keep PaCO2 32±1. Anesthesia was maintained with 1% halothane in oxygen. An arterial line was placed in the aorta at kidney level via the femoral artery. Two peripheral veins were cannulated for fluid and drug administration. Following base line blood and urine sampling, loading doses of PAH (10mg/kg) and creatinine (70mg/kg) were given intravenously. PAH (5mg/kg/min) and creatinine (35mg/kg/min) were given by a fixed rate infusion pump throughout the experiment, to provide a constant arterial concentration. Five percent dextrose in lacated Ringer's solution (0.5ml/kg/min) was administered to maintain urine output. The left kidney was exposed by a flank incision, the left wreter was separated and cannulated for urine collection. left renal vein was catheterized with 20 gauge catheter. After establishing a steady state of infusion and anesthesia, the study was divided in three forty minutes periods, control, hypotension and recovery. Mean arterial pressure (MAP) was dropped from a control value of  $105\pm2$  to  $62\pm2$  by continuous infusion of SNP in (7 dogs) or TMP (8 dogs) or by hemorrhage (6 dogs). MAP was returned to control value by discontinuation of SNP or TMP or retransfusion of the shed blood. Arterial, renal venous and urine samples were taken at 20 and 40 minutes intervals in each period.
MAP was continuously recorded. Data were analyzed for statistical significance using the paired t-test within each group.

Results. GFR, ERPF, TNa, Vu were significantly decreased during induced hypotension in all groups (Table). These variables returned to control values in SNP Group. Vu returned to control value in all three groups. GFR, ERPF, Na and PrvO2 were significantly lower than control levels in the TMP and hemo group.

Discussion. These data suggest that, although the renal hemodynamic changes during hypotension induced by three different techniques are similar, recovery of depressed functions are different. They return toward control valves at different rates.

The which correlates well with renal oxygen consumption returns to normal only in the SNP group. SNP may be a safer drug since renal function returns to normal more rapidly following its use.

TABLE. RENAL CHANGES DURING HYPOTENSION (Percent Change from Control)

	Hypotension			Recovery		
	TMP	HEMO	SNP	TMP	HEMO	SNP
GFR	-74 <sup>a</sup>	-75 <sup>a</sup>	-76 <sup>a</sup>	-43 <sup>c</sup>	-30 <sup>d</sup>	- 2
ERPF	-70 <sup>a</sup>	-74 <sup>a</sup>	-70 <sup>c</sup>	-64 <sup>a</sup>	-51 <sup>a</sup>	+23
† <sub>Na</sub>	-74 <sup>a</sup>	-73 <sup>a</sup>	-72 <sup>a</sup>	-45 <sup>b</sup>	-31 <sup>đ</sup>	- 4
Ϋu	-76 <sup>b</sup>	-92 <sup>c</sup>	-94 <sup>a</sup>	-11	- 8	+15
$P_{\mathbf{u}}O_2$	+ 8	+ 9	+ 6	- 9	- 8	- 9
P <sub>rv</sub> O <sub>2</sub>	- 2	-15	+16	-24 <sup>c</sup>	-32 <sup>d</sup>	-17
(a) P < 0.001 (b) P < 0.01 (c) P < 0.02 (d) P < 0.05						

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