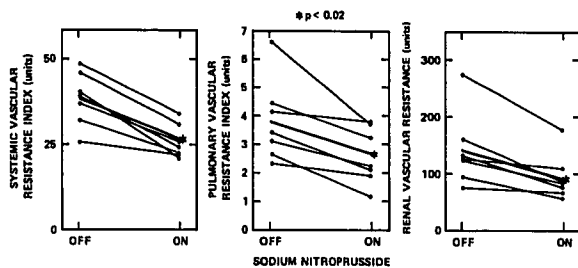


Date :
 Title : THE RENAL EFFECTS OF SODIUM NITROPRUSSIDE IN POSTOPERATIVE CARDIAC SURGICAL PATIENTS
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Nitroprusside (NP) causes renal vasodilation when infused into isolated kidneys, however the reported effects of NP on renal vascular resistance and blood flow in intact animals and man have varied. To define the effects of NP, cardiac and renal function were measured within 24 hrs of cardiac operation.

Methods. Informed consent was obtained preoperatively from 7 patients scheduled for coronary bypass grafting. The protocol and consent procedures had institutional approval. Cardiac and renal function were measured using standard techniques, previously described.¹ [Cardiac output using indocyanine green; pulmonary capillary wedge pressure (PCW) through a Swan-Ganz catheter; glomerular filtration by the clearance (C) of inulin (Cin), and renal plasma flow by the C of para-aminohippurate (Cpah).] Baseline measurements were performed; NP was infused to lower mean arterial pressure (MAP) to 85 torr, PCW was restored (which increased MAP), and the measurements were then repeated. The data were analyzed by paired t-test.

Results. (mean ± S.D.) NP infusion averaged 0.76 ± 0.71 µg/kg/min and decreased group MAP from 109 ± 14 to 91 ± 9 torr (p < 0.01) and increased cardiac index from 2.5 ± 0.4 to 3.0 ± 0.3 l/min/M² (p < 0.01). Because of intentional blood volume expansion, PCW and central venous pressures did not change significantly. Uniform and significant decreases occurred in calculated systemic, pulmonary, and renal vascular resistance indices (figure 1).



NP	Urine Flow (ml/min)	Cin ---(ml/min/1.73m ²)---	Cpah	RBF
OFF	1.3 (± 0.4)	105 (± 35)	354 (± 101)	653 (± 193)
ON	1.5 (± 0.7)	109 (± 35)	424 (± 102)	792 (± 210)
P	> 0.1	> 0.1	< 0.02	< 0.02

Cpah and renal blood flow increased significantly in all patients during NP administration (table). No significant changes occurred in urine flow, Cin, sodium excretion, or potassium excretion.

Discussion. One explanation for the reported variation in the renal response to NP lies in the profound renal vasoconstriction which may result from left atrial hypotension. Thus, Kahl² reported that superior vena caval obstruction decreased cardiac index, MAP and left atrial pressure and resulted in profound renal vasoconstriction. Balloon inflation in the left atrium decreased cardiac index and MAP, however, left atrial pressure was elevated and renal vascular resistance remained unchanged. Previous studies in which NP administration increased renal vascular resistance or decreased renal blood flow were performed under conditions likely to decrease atrial pressures to below normal levels, however this variable was not measured. By contrast, although left atrial pressure declined during NP infusion in patients with congestive heart failure, it remained in the high-normal range; under these conditions renal blood flow improved significantly, although MAP fell to 22 torr.³ Additional factors which may influence the effects of NP on renal function appear less important; these include a decline in MAP below the autoregulatory range, the abolition of autoregulation by renal vasodilators, and/or differing hemodynamic responses of the normal and failing circulation.

Given the documented effects of NP on the renal vasculature and the equivalent reductions in systemic (-31%), pulmonary (-29%), and renal (-33%) vascular resistance we observed, it seems likely that NP causes direct vasodilation of all three vascular beds. Our findings indicate that NP infusion is associated with renal vasodilation and increased renal perfusion in postoperative cardiac surgical patients.

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