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TITLE : HORMONE CHANGES WITH NITROPRUSSIDE OR TRIMETHAPHAN

AUTHORS : G. Lane, M.D., R.E. Bolles, B.G.S., R.N. Hensinger, M.D., P.R. Knight, M.D., Ph.D.

AFFILIATION: Department of Anesthesiology, University of Michigan Medical Center, Ann Arbor, Michigan 48109

Introduction. Recently, substantial interest has been generated regarding hormonal changes following vasodilator therapy. Investigations in our laboratory have shown that deliberate hypotension using pentolinium tartrate and propranolol does not cause stimulation of the autonomic or renin-angiotensin systems[1]. In this report we compare hormone changes during hypotension induced by Trimethaphan camsylate and sodium nitroprusside.

Methods. Twelve patients undergoing surgery for Harrington rod placement and spinal fusion for correction of idiopathic scoliosis were prospectivly assigned to two treatment groups using random numbers. Informed consent was given by the patient and his or her parent. One group received Trimethaphan camsylate (TMC) while the other group recieved sodium nitroprusside (SNP) to decrease the blood pressure and bleeding intraoperatively. Arterial blood was drawn for norepinephrine (NE), epinephrine (E), dopamine (DA), plasma renin activity (PRA) and Angiotensin II (AII) levels at the following time periods: (1)following induction of anesthesia, prior to the start of surgery and hypotension, (2) after 1 hour of stable hypotension,(3) after 2 hours of hypotension, during placement of the Harrington Rods, (4) after return to control blood pressure following discontinuation of TMC or SNP but, prior to leaving the O.R. and (5) after 1 hr. in the recovery room. In addition, the ECG,continuous arterial blood pressure, and central venous pressure were monitored as were arterial and venous blood gases.

Results. The two vasodilators induced different hormonal changes. These results are summarized in the following table. DA and AII levels are not included in the table since the former did not change and the latter paralleled the PRA changes. After one hour of deliberate hypotension, NE, E, PRA, and AII were significantly elevated in the group of patients receiving SNP. These hormones were unchanged or lower than control levels in the group treated with TMC. Mean arterial blood pressure was reduced from 80 to 62 torr in the group recieving TMC and from 75 to 54 in the group recieving SNP. Heart rate was significantly elevated in both groups. These changes persisted during the period of deliberate hypotension. After deliberate hypotension, NE, E, PRA, and AII decreased and returned toward control values in those patients treated with SNP, while in those patients treated with TMC, E and NE increased. The group which had recieved TMC continued to manifest elevated E and NE levels into the recovery phase. These hormones were also increased in the recovery room in the group which had recieved SNP. Clinically, three patients in the group which recieved SNP were noted to have rebound bleeding in the recovery room, requiring multiple dressing changes. The bleeding in this group was also associated with a mean blood pressure which was significantly higher than seen in the group which had recieved TMC.

Discussion. The results are consistent with the pharmacological effects of the drugs. TMC would be expected to decrease autonomic outflow during the time of its use. The rebound in E and NE has not, however, been reported with discontinuation of this agent. The secretion of these hormones in response to SNP hypotension has been reported. The decrease in catecholamine levels following discontinuation of SNP is in direct contrast to those patients recieving TMC although the levels of E and NE in this patient group were again elevated in the recovery room. These changes in hormone responses to induced hypotension may have significance in the redistribution of regional blood flow, and in rebound bleeding postoperatively.

References.

1. Knight PR, Lane GA, Nicholls MG, et al: Hormone and hemodynamic changes induced by pentolinium and propranolol during corrective scoliosis surgery. Accepted for publication; Anesthesiology 1980.

Table: Summary of Results (mean ± SEM)

Period:		1	2	3	4	5
MAP (torr)	TMC	80 ±5	62+ ±2	66+ ±2	79** ±7	83 ±5 *
	SNP	75 ±2	54+ ±5	59+ ±2	73** ±2.1	94** ±4.3
HR (min/-1)	TMC	79 ±2.2	98+ ±7.0	98+ ±4.5	94+ ±4.8 *	88 ±5
	SNP	69 ±4.7	93+ ±12	88+ ±12	79 ±9	81 ±8.7
E (pg/ml)	TMC	44 ±11	51 ±4.5 *	77 ±15 *	279**+ ±78	309+ ±61
	SNP	56 ±38	365+ ±123	579 ±141	90** ±25	381**+ ±126
NE (pg/ml)	TMC	121 ±19	65 ±11 *	92 ±17 *	219**+ ±81	313+ ±52
	SNP	108 ±17	382+ ±101	468+ ±60	116** ±17	250+ ±37
PRA) (ng/ml/hr)	TMC	3.8 ±.58	4.7 ±1.9 *	5.7 ±2.2 *	6.5 ±2.2 *	5.6 ±2.7
	SNP	5.0 ±1.1	12.3+ ±2	16+ ±4	11.1+ ±3.2	7.1 ±2.5

*P <.05 (group)
**P <.05 paired (prior value)
+P < .05 paired (control)