

Title: RENIN, CATHECHOLAMINE, AND VASOPRESSIN RESPONSE TO THE "STRESS" OF ANESTHESIA AND SURGERY

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Introduction. There are conflicting reports in the literature concerning the hormonal response to the "stress" of anesthesia and surgical stimulation^{1,2,3}. Improved methods of assay now allow for more detailed and specific measurement of the hormone responses. This study was undertaken to determine the changes in plasma vasopressin and catecholamine levels and renin activity in patients subjected to anesthesia and surgery.

Methods. Eight patients scheduled for elective coronary artery bypass graft surgery were selected for study. The protocol was approved by the human studies committee of the institution and informed consent was obtained in each case. All patients were premedicated with morphine 0.1 mgm/kg and scopolamine 0.4 mgm intramuscularly approximately 90 minutes prior to the beginning of the study. Percutaneous radial and pulmonary artery and central venous catheters were introduced under local anesthesia. Electrocardiogram leads were also placed, including V₅, and then control measurements were obtained. All patients were then induced with pentothal 2 mgm/kg, IV, while breathing 50:50 oxygen:nitrous oxide mixture with 0.5% halothane. Patients were intubated aided by the use of an intravenous succinylcholine drip. Measurements were then repeated after 15 minutes of anesthesia and again at 15 and 30 minutes following sternal skin incision. During each period the following variables were measured or recorded: mean BP, PCW and CVP, cardiac output (thermal dilution), systemic vascular resistance, plasma renin activity and plasma vasopressin, epinephrine and nor-epinephrine levels.

Results. The major data are recorded in the table. Anesthesia alone produced significant increases in PCW, and nor-epinephrine levels. The decreases in BP, SVR, and cardiac output were not significant, nor were the changes in renin activity or vasopressin levels. At 15 minutes following surgical incision, nor-epinephrine levels had decreased but were still significantly elevated. Epinephrine and vasopressin levels were significantly elevated but renin activity was not. After 30 minutes of surgery the nor-epinephrine levels were no longer significantly different from control but both renin and epinephrine were. The PCW remained elevated.

	Control	Anesthesia	Surgery-15'	Surgery-30'
BP mmHg	87±3	79±4	88±4	72±10
PCW mmHg	7.0±1	9.0±1 ^x	10.0±1 ^x	10.0±1 ^x
C.O. L/min	5.6±0.4	5.2±0.3	4.6±0.3	4.9±0.7
SVR units	15.5±1	14.2±1	18.5±1 ^x	17.3±2
Renin ng/ml/hr	0.898 ±0.16	1.297 ±0.24	1.42 ±0.3	2.58 ⁺ ±0.7
Epi pg/ml	91±25	78±15	196±48 ⁺	203±41*
Nep pg/ml	327±33	567±110 ^x	479±81 ^x	460±99
Vasopressin pg/ml	3.2±1	3.3±1.2	43.0±9 [*]	36.0±7*

x = p<0.05

+ = p<0.02

* = p<0.01

Discussion. These data demonstrate that pentothal/halothane anesthesia alone does not significantly affect plasma vasopressin or epinephrine levels or plasma renin activity but can significantly affect nor-epinephrine levels. This perhaps explains why the decrease in both cardiac output and BP did not achieve significance in this period. Surgical stimulation results in significant elevations of both vasopressin and epinephrine levels and at the same time significant increases in SVR and PCW. The lack of a significant increase in renin activity until 30 minutes suggests that it does not play an important role in the immediate responses to stimulation under these conditions. These data would suggest that anesthesia alone produces relatively little hormonal stress response in patients with relatively normal levels. They also support the concept that the stress response to surgical stimulation is primarily vasopressin-catecholamine mediated with renin playing a relatively minor role in any immediate response.

References.

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