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Introduction. Oliguria is common in the immediate postoperative period and may result from volume depletion or vasopressin excess. Non-osmotic secretion of vasopressin is stimulated by surgical stress, pain, and nausea. Vasopressin may decrease urine output into the oliguric range (<0.5 cc/kg/hr) but is usually associated with an expanded intravascular volume. The treatment for hypovolemic oliguria consists of fluid administration while the treatment for vasopressin-induced oliguria involves prevention of free water formation within the kidney (i.e., with furosemide). Thus, a major differential in the evaluation of patients with prerenal oliguria is to determine whether they are hypovolemic or have vasopressin excess. This study was performed to evaluate the utility of urine sodium (Na), urea, creatinine, and osmolality in the evaluation of pre-renal oliguria.

Methods. One hundred consecutive ICU patients were prospectively followed for the development of oliguria (<0.5 cc/kg/hr for two consecutive hours). patients developed oliguria. Eighteen patients had blood obtained for serum Na (meq/1), osmolality (mOsm/kg H2O), BUN, and creatinine. spot urine sample was analyzed for Na, osmolality, urea, and creatinine. A microscopic examination of the urine was also performed. The fractional excretion of Na (FENa) and renal failure index (RFI) were calculated.

Hypovolemia was defined clinically by the occurrence of a low blood pressure (MAP <80 nm Hg), tachycardia (HR >100 beats/min), orthostatic blood pressure and HR changes, a low CVP (<5 cm H2O), and a low PCWP (<5 mm Hg). Oliguric patients were given a fluid bolus (500 ml normal saline over 5-10 min) and urine output response recorded.

Data are reported as mean \pm SE and were analyzed by two-tailed Student's "t" tests. *p<0.05.

Results. Eleven oliguric patients were classified as normovolemic and seven hypovolemic. The normovolemic patients had a significantly (p<0.05) lower serum osmolality and serum Na concentration compared to the hypovolemic patients (See Table). The normovolemic patients had higher values for urine Na, FENa, and RFI. There were no significant differences between normovolemic and hypovolemic patients in regards to urine osmolality, urine

sediment, BUN, serum creatinine, lowest urine output, or diagnoses. The hypovolemic patients increased their urine output (>0.5 ml/kg/hr) following a fluid bolus while the normovolemic patients did not (urine output 13±2 ml/hr to 19±3 ml/hr). The normovolemic patients increased their urine output to levels >0.5 ml/kg/hr with furosemide. All the patients in this study left the ICU and none developed renal failure.

Discussion. Vasopressin excess was the most common cause of oliguria in our patients (61%). This oliguria was characterized by concentrated urine associated with a low serum Na, elevated urine Na, and a FENa and RFI greater than one. Hypovolemic oliguria was characterized by concentrated urine, a normal serum Na, low urine Na, and a FENa and RFI less than one. Despite the findings of a FENa and RFI greater than one, no patient developed renal failure. Thus, these indices are inaccurate indicators of renal failure and values greater than one may occur in patients with vasopressin-induced oliguria. This group of patients responded well to furosemide. Furosemide works well in patients with vasopressin excess by diminishing free water production in the loop of Henle. Our data suggest that urine Na excretion can be used to separate acutely oliguric patients with hypovolemia from those with normovolemia and vasopressin excess. In addition, urine Na excretion may be useful for choosing between fluid administration and furosemide therapy.

	Normovolemic	Hypovolemic
n	11	7
age (years)	63 ± 4	53 ± 9
Urine output (m1/hr)	13 ± 2	17 ± 2
Serum osmolality	278 ± 3*	290 ± 5
Serum Na	132 ± 1*	138 ± 3
Urine osmolality	522 ±36	525 ± 34
Urine Na	83 ±12*	11 ± 2
FENa	1.15±.2*	0.15±.03
RFI	1.5±.3*	.21±.04
BUN (mg/dl)	14 ± 3	15 ± 3
Creatinine (mg/dl)	0.9±.08	1.0±.13
BUN/creatinine	15 ± 3	15 ± 3
Microscopic	benign	benign