DI

DECREASED ADH

Causes: *Neurogenic* - hypothalamus or pituitary lesion causes decreased synthesis, transport and release of ADH. *Nephrogenic* - inadequate response at the kidney tubules to ADH. Results in dilute urine.

Signs and Symptoms: polydipsis, polyuria, hypernatremia, change in mental status, fatigue, weakness, hemodynamic instability.

Diagnosis: DDAVP test. A pt with neurogenic DI will respond by having an increase in urine osmolality concentration.

Treatment: *Neurogenic* - 100-200 mU/h DDAVP with isotonic crystalloid. *Nephrogenic* - Chlorpropamide, clofibrate and thiazide diuretics.

SIADH

INCREASED ADH RELEASE

Cause: small cell lung carcinoma - (50% of these pt's develop SIADH). Medications that stimulate the release of ADH: chlorpropamide (sulfonyl-urea), clofibrate (fibrate), thiazide diuretics, antineoplastic agents.

Signs and symptoms: Nausea, weakness, lethargy, confusion, depressed mental status, seizures

Lab values: hyponatremia, decreased serum osmolality, normal/in-creased Na+ excretion, normal/increased urine osmolality

Treatment: Surgery to treat primary malignancy. Free water restriction (500-1000mL/day). Demeclocycline: inhibits ADH at distal tubule. Convaptan: V2R antagonist (vasopressin 2 receptor). Severe hypona-tremia (<115mEq): 3% NS or 0.9% NS with furosemide.

Anesthetic considerations: Replacement Na+ slowly to prevent *pontine myelinolysis*