	SIADH	CSW
	(first described 1957)	(first described 1950)
Pathology	Disruption of hypothalamic – neurohypophyseal system, resulting in abnormal excretion of ADH	Disruption of neural input to kidney, or central elaboration of circulating natriuretic facto
Urine specific gravity	Elevated	Elevated
Urine osmolality	Increased (urine osm > serum osm)	Increased
Urine sodium	Elevated (18mEq/L)	Elevated
Serum osmolality	Decreased (<280 mOsm/L)	Decreased
Plasma volume (CVP/PAOP)	Increased (but peripheral edema is NOT seen)	Decreased
Serum sodium	Decreased (<135mEq/L)	Decreased
Serum potassium	Normal or low	Normal or elevated
Clinical manifestations	Eu- or hypervolemic, low urine output, low BUN	Hyopvolemic, high urine output, increased BUN
Serum uric acid	Low *	Low or normal* (note: since this is a volume contracted state, you would expect it to be HIGH)
ADH excretion	Increased	Increased
Fractional excretion of urate	High *	High *
Physiology	Inappropriate release of arginine vasopressin (expanded blood volume)	Appropriate release of arginine vasopressin in response to volume depletion
Treatment	Fluid restriction	Fluid/sodium replacement
Caveats to treatment	Saline administration can cause further lowering of serum sodium	Fluid restriction can worsen underlying condition

- In the setting of hyponatremia, the normal response of the kidney is to elaborate a maximally dilute urine.
- In hyponatremic patients with inappropriately concentrated urine, one needs to assess the
  effective blood volume.
- Serum and urine electrolytes and osmolalities may be the same in patients with SIADH and CSW.
- SIADH and CSW may occur in the same patient, particularly after SAH!

<sup>\*</sup>While correction of serum sodium concentration in SIADH leads to a normalization of uric acid handling by the kidney, hypouricemia and increased renal uric acid excretion remains a persistent finding following the correction of serum sodium in CSW.