

## CASE E—EVALUATION OF HYPONATREMIA

A 74 year old man was admitted in February with a stroke. After about one month in the hospital, he developed shortness of breath and was found to have pneumonia, which was resistant to treatment, raising the possibility of an ongoing source. His intake of food was limited, and he was maintained on intravenous fluids until about the beginning of March when a feeding tube was inserted. He was noted to be hyponatremic at this time, and his serum sodium did not increase despite addition of extra salt to his tube feedings. What do the urine and serum osmolalities reveal? What is the most likely etiology for these findings?

SPECIMEN	Na	K	Cl	CO <sub>2</sub>	BUN	Creatinine	Glucose	Osmolality
Early Feb.	138	3.6	98	25	17	0.9	143	
Early Mar.	120	4.6	83	23	15	0.5	131	257
Late Mar.	113	4.9	81	21	9	0.7	89	239
Urine	70	70.1	41			90.7		694

### Discussion

His serum osmolality was low, confirming that there is a true sodium abnormality present. His urine osmolality indicates a specimen that is significantly concentrated compared to serum, indicating the presence of ADH activity.

In evaluating a patient with ADH production, the first step is to determine whether it is being produced appropriately. Because the major stimulus to ADH release is increased plasma osmolality, hypoosmolar patients would be expected to produce the hormone only if they had a decrease in intravascular volume. Physicians often look for evidence of an abnormal blood pressure change when the patient stands up as an indicator for decrease in blood volume; however, this is not present until there has been a decrease of at least 5-10% in total volume. The ratio of BUN to creatinine increases with volume depletion, since urea is reabsorbed from urine along with water in cases of dehydration (causing a selective decrease in urea clearance and an alteration of the normal BUN/creatinine ratio).

From the time of admission, this patient's serum BUN and creatinine gradually decreased with a lowering of the BUN/creatinine ratio. This pattern is most frequently seen in patients who have water overload, as discussed in case C above. The combination of hyponatremia, decreased BUN and creatinine with low ratio, and evidence of ADH production without appropriate stimulation are thus diag-

nostic of the syndrome of inappropriate antidiuretic hormone (SIADH) production. This disorder is an extremely common cause of hyponatremia in hospitalized patients. The most common causes are diseases of the lung and brain; a large number of different illnesses affecting these organs can cause this syndrome. In addition, cancers are often associated with SIADH.

In this patient, there are two likely causes for this illness, pneumonia and the initial stroke. Salt administration is usually ineffective in treating patients with SIADH, since the expansion in their vascular volume stimulates ANH production, increasing urinary sodium losses. For mild cases of SIADH, restriction of water intake often is adequate in controlling the hyponatremia. In more severe cases, as in this patient, or if the syndrome is due to a malignancy, it is often necessary to use a drug which antagonizes the effects of ADH on the kidney; the most widely used agent is the antibiotic, demeclocycline, which was used in this patient when he did not respond to fluid restriction.