iciency typically involves destruction of the adrenocortical glomerulosa and fasciculata, measurement of renin and aldosterone would have been of interest in this case.

Author Contributions: All authors confirmed they have contributed to the intellectual content of this paper and have met the following 3 requirements: (a) significant contributions to the conception and design, acquisition of data, or analysis and interpretation of data; (b) drafting or revising the article for intellectual content; and (c) final approval of the published article.

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Commentary

Robert Richardson

Patients with chronic hyponatremia have vasopressin acting on the kidney, causing water retention and dilution of the sodium in the extracellular fluid. In surveys of hospitalized patients with hyponatremia, the stimulus for vasopressin secretion is low effective circulating volume via the baroreceptor in about two-thirds of patients. The remaining one-third have a variety of stimuli for vasopressin secretion, including surgery, nausea, hypothyroidism, adrenal insufficiency, and SIADH. Although physical examination is often extremely helpful in detecting signs of altered effective circulating volume (such as edema, ascites, hypotension, and abnormal jugular venous pressure), physical exam is unreliable in a significant proportion of patients.

The urine sodium concentration is a critical diagnostic aid in differential diagnosis. It should be low (<30 mmol/L) with reduced effective circulating volume, because sodium-retaining factors, including angiotensin II, catecholamines, the sympathetic nervous system, and aldosterone, are activated and stimulate renal tubular sodium reabsorption. When the urine sodium concentration is high, as in this case, SIADH should be strongly considered. This rule has 2 important exceptions, however. The first is diuretic use, which can cause baroreceptor-mediated vasopressin release through reduced effective circulating volume with a high urine sodium concentration. The second is adrenal insufficiency, in which cortisol or aldosterone deficiency can lead to vasopressin secretion. Urine sodium is high because of aldosterone deficiency. Adrenal insufficiency must always be ruled out in cases of severe hyponatremia, because the classic clinical and biochemical features may not be present.

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