fluids were 2800 ml of lactated Ringer's and normal saline. Urine output was 650 ml. In the post-anaesthesia care unit (PACU) she was awake with stable vital signs.

In the PACU the intravenous fluid was changed to D_5W. The next morning she was walking and taking ice chips. At 10:00 she complained of dizziness and a floating sensation; one hour later she complained of a headache. At 17:00 she was noted to be smiling and feeling drowsy but at 19:15 p.m. she was found unresponsive, cyanotic and pulseless. Cardiopulmonary resuscitation restored a haemodynamic function. Laboratory values were normal except for a plasma sodium concentration of 116 $mEq \cdot L^{-1}$, a chloride of 80 $mEq \cdot L^{-1}$ and a glucose of 416 $mg \cdot dl^{-1}$. Computerized tomography showed diffuse cerebral oedema. A neurology consultation and an isoelectric EEG were obtained and the patient was declared brain dead. An autopsy showed hypoxic-ischaemic damage to the brain.

In 1986 Arieff reported 14 female patients with postoperative hyponatraemia who suffered grand mal seizures.[2] These patients were of good health and underwent relatively minor procedures. Two patients had received only intravenous sedation, three had local anaesthesia and the others general anaesthesia. They were ambulatory, communicating, voiding spontaneously and taking fluids by mouth shortly after surgery. During the first two postoperative days the patients all complained of headache, nausea and vomiting. Later, half became incontinent of urine; four were described as hostile; and four were disoriented. Seven had hallucinations and eight were noted to be lying in bed with only mild symptoms just before having seizures. Serum sodium concentrations averaged 108 $mEq \cdot L^{-1}$ at that time. Within an hour of the seizures all suffered respiratory arrest. All were initially resuscitated but four died shortly thereafter and nine others remained in a persistent vegetative state. The other regained consciousness but suffered permanent partial paralysis.

Seven of Arieff's patients awoke after correction of their hyponatraemia. However, they all had neurological deterioration, suffered recurrent seizures and lapsed back into coma.

Soroker *et al.* presented a case of postoperative SIADH in a young boy. Previous cases have concerned only female patients thus postulating a sex influence. Perhaps these cases are not as rare as supposed but diagnosis and documentation occurs only rarely.

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REPLY

We have read with interest the case of postoperative hyponatraemia that ultimately progressed to coma and hypoxic-ischaemic damage to the brain, which was described by Fuhrman et al. *in their letter.*

The diagnosis of the syndrome of inappropriate antidiuretic hormone secretion (SIADH) should be confirmed by laboratory findings of urine osmolality hypertonic to serum and by demonstration of elevated serum ADH concentration, as not all postoperative hyponatraemia can be ascribed to the SIADH secretion.[1,2] Therefore, the diagnosis of SIADH could not be definitively made in their case, or in the 15 cases described by Arieff.[3]

Furthermore, in our case[4] the outcome was good, which is opposite to the grave outcome in the case desribed by Fuhrman et al. *in their letter and in those of Arieff.[3] Perhaps female sex influence predisposes to a less favourable outcome.*

Some degree of inappropriate ADH secretion is present in virtually all patients after surgery,[1] and about 5% of postoperative patients have hyponatraemia (serum sodium <130 $mEq \cdot L^{-1}$);[1] however, the condition is rarely symptomatic. The purpose of our report[4] was to describe a rare and well documented symptomatic occurrence of SIADH secretion after minor surgery.

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