may treat or prevent acute hypotonic hyponatremia and is more important in these patients than is the justification for seeing urine appear. In this regard, proposing a new classification in which ADH excess is listed as a prerenal cause of oliguria may lead to a further misconception that these normovolemic patients also need volume repletion. In patients with nonosmotic secretion of ADH, therapy should be directed toward the signs and symptoms of hypotonic hyponatremia rather than toward a transient reduction in urine output.

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Excess ADH and Oliguria in Patients with Normal Renal Function: II.

To the Editor:—The article by Zaloga and Hughes provides interesting data from patients with normal renal function who had undergone surgery and were oliguric in the postoperative period.[1] In order to better understand this population of patients, it is important to know whether their lungs were being mechanically ventilated in the intensive care unit during the measurements of renal function, since this therapy may have had a significant effect on renal function.[2]

The authors conclude that antidiuretic hormone (ADH) excess was responsible for the oliguria in these critically ill patients, since plasma ADH concentrations were increased over normal in hydrated patients. Could it be possible that the ADH concentration was increased in these oliguric patients but had no relevance to the changes in renal function? In order to establish a role for ADH, it is necessary to measure plasma ADH concentrations in postoperative, critically ill patients who are not oliguric to compare to the values from the groups in Zaloga and Hughes's study. In addition, the classic effect of ADH is to produce a negative free water clearance, a parameter that was not calculated for the study patients.

ADH concentrations may be increased as a result of many stimuli, but this hormone may not always have an effect on renal function. Hemodynamic changes, renal blood flow, sympathetic stimulation, or other hormones may have been responsible for the oliguria noted in this sample of postoperative patients. Without the appropriate control group, interpretation of the ADH data is impossible.

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In Reply:—Most nephrology textbooks define oliguria as a urine output of less than 500 ml per 24-h period. Although this definition is satisfactory for outpatients, it is unsatisfactory for critically ill inpatients. Renal perfusion may change rapidly in critically ill patients, and generally it is believed that diminished renal perfusion (manifested by oliguria) in the setting of nephrotoxic drugs, sepsis, or other conditions common in these patients may lead to renal failure. In addition, urine output is frequently used as a monitor of other organ perfusion (*i.e.*, liver and gut). Thus, urine output is usually measured on an hourly basis in critically ill patients, and most textbooks of critical care and anesthesiology define oliguria as a urine output less than 0.5 $ml \cdot kg^{-1} \cdot h^{-1}$ (less restrictive than the value we used). We believe that hourly monitoring of urine output is essential to the detection of renal hypoperfusion before permanent renal damage occurs. Since hourly monitoring of urine output is the standard of practice in critical care and anesthesia, we chose this method of monitoring to define oliguria.

We agree that low urine output in a patient with clinical features of hypovolemia does not require urinary indices to aid in a diagnosis of volume depletion. On the other hand, in many patients clinical features are not conclusive, and in these patients, we believe, urinary indices may be helpful. A major point of our study[1] was the demonstration that patients with low urine output and a physical exam that does not indicate hypovolemia frequently have hormonal-excess-associated oliguria and do well without volume administration.

Normovolemic patients with oliguria and normal renal function do not require treatment with fluids. In fact, large volume infusion in

these patients may lead to pulmonary edema. Diuretics may be used safely to mobilize fluid in these patients.

A number of published articles indicate that ADH can cause oliguria. These studies are reviewed in our article.[1] In support of this concept, we occasionally produce oliguria when administering large doses of dDAVP to brain-dead patients prior to organ procurement. It is unclear whether ADH alone is causing the oliguria in our patients. We have postulated a variety of mechanisms for the oliguria.[1] Decreased ultrafiltration pressure, increased glomerular capillary osmotic forces, decreased renal blood flow, and excesses of other circulating factors (*i.e.*, prostaglandins) may have contributed to the oliguria.

We have not made any statements suggesting that furosemide prevents renal failure, nor have we stated that azotemia develops in normovolemic oliguric patients without furosemide treatment.

We agree that many of our patients were hypotonic and hyponatremic. These patients are unable to excrete free water due to ADH excess. Administration of hypotonic fluids to these patients may result in pulmonary edema or further hypotonicity. One of the points of our article is that overzealous fluid administration, in an attempt to improve urine output in these patients, may be deleterious. We agree that the avoidance of such therapy and the use of furosemide/free-water restriction may prevent these complications. Urine indices may be helpful in separating these patients (normovolemic oliguria) from those with hypovolemic oliguria.

Proposing a new classification of oliguria in which ADH excess is listed as a prerenal cause of oliguria should clarify the issue rather than cause further misconceptions. Oliguria associated with ADH excess should caution one against the use of exogenous fluid since the standard treatment for SIADH is free-water restriction.

The lungs of most of the patients in this study were being mechanically ventilated in the SIMV mode in the intensive care unit when oliguria developed and when the measurements were made. In addition, the majority were receiving 5 cm or less of PEEP. The mode of ventilation and level of PEEP were not different from those of patients who did not develop oliguria. Thus, we believe that the degree of ventilatory support was not a major factor in the etiology of the oliguria.

We have measured ADH concentrations in a number of postoperative, volume-replete, intensive care unit patients without oliguria. The ADH concentrations in these patients were uniformly less than 1.5 pg/ml (lower than in our oliguric patients). We have not measured free-water clearance. We believe that excess ADH contributed to the oliguria in out patients but was not the sole factor. The oliguria most likely resulted from a combination of factors (*i.e.*, excess hormonal effects, altered renal blood flow, and altered glomerular regulation).

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Role of Intravenous Regional Bretylium in Reflex Sympathetic Dystrophy

To the Editor:—Sympathetic blockade is an important modality in the treatment of reflex sympathetic dystrophy. After the description by Ford *et al.*[1] of use of intravenous regional bretylium for the treatment of reflex sympathetic dystrophy, this technique was used in four patients with proven reflex sympathetic dystrophy with disappointing results.

Four patients with the diagnosis of sympathetic dystrophy, as confirmed by bone scan, electronic thermography, differential neural blockade, and response to sympathetic blocks, were treated with intravenous regional bretylium because of short-lived response with sympathetic blocks. The patients were two males and two females, 38, 39, 55, and 72 yr Of age. Duration of sympathetic dystrophy ranged from 3 to 9 months. Of the four patients, two suffered reflex sympathetic dystrophy of the upper extremities, and the other two suffered that of the lower extremity. After the initial diagnosis, they were treated with either stellate ganglion block or lumbar sympathetic block.

Because of failure in obtaining significant, long-lasting relief with the above treatments, treatment with intravenous regional bretylium was begun. The four patients received a total of eight blocks. Blocks were performed with lidocaine 0.5%, 50 ml for upper extremities and 75 ml for lower extremities with double tourniquets using pressures of 300 or 400 mmHg for upper and lower extremities, respectively, after exsanguination. In addition, all patients were administered 100 units heparin. Tourniquets were inflated for 30–40 min. Bretylium dose was 2.0 mg/kg in the first two patients for their first treatment. Subsequent doses in these two patients and all four treatments in the other two patients were 3.0 mg/kg. All of the patients experienced pain relief and increased mobility for a variable period after the deflation of the tourniquet. In the three patients, this improvement lasted to a maximum of 15 min after six treatments, whereas in one patient, the improvement was of 6 h duration after the first treatment and of less than 15 min duration after the second treatment. Side effects were minimal except for postural hypotension in one patient; this was controlled by intravenous fluid administration.

Four patients with reflex sympathetic dystrophy did not show any significant improvement with bretylium intravenous blockade, similar to those in the report by Hanowell *et al.*[2] Tourniquet-induced analgesia, as described by Ramamurthy *et al.*,* also was not observed in our patients. However, all of these patients responded to sympathetic blockade, even though the relief was variable. While there is no explanation for the contrasting experience with bretylium, as well as the concept

* Ramamurthy S, Hoffman J, Walsh N, Schoenfield L: Role of tourniquet-induced analgesia in iv regional sympatholysis (abstract). ANESTHESIOLOGY 65:A207, 1986