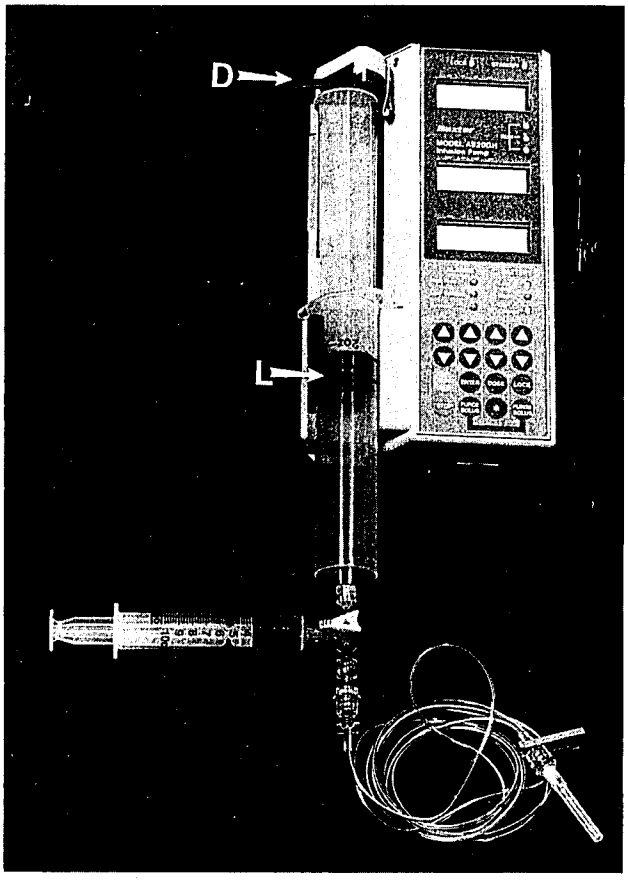

FIG. 1. Infusion set with stopcock. D = syringe driver; L = reference level.

the infusion tubing. When refilling is required, a loaded syringe is attached to the side and the stopper is appropriately turned. The syringe driver (D in fig. 1) at the plunger is disconnected and the parent syringe refilled. The syringe driver is then reconnected and infusion continued.

We recommend that a reference mark (L in fig. 1) be placed on the syringe and the level consistently checked, since we have experienced pump malfunction (without alarms) resulting in medication not being delivered to the patient.

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

To the Editor:—Zaloga and Hughes[1] present data on 18 patients with postoperative "oliguria" who have normal renal function, in an attempt to recommend criteria for diagnosing and treating these patients. However, several important questions need to be addressed.

To begin with, there is a question about the authors' definition of oliguria (*i.e.*, a random 2-h [0.33 $ml \cdot kg^{-1} \cdot h^{-1}$), which appears to be an arbitrary definition with minimal physiologic basis. As the authors note, most major nephrology texts define oliguria as urine output less than 500 ml over a 24-h period. To extrapolate this to a 2-h time frame, however, may not be clinically relevant.

In regard to the hypovolemic patient with low urinary output, the concern to treat these patients is undoubtedly justified, as sustained prerenal azotemia may lead to acute renal failure. Based on the clinical data used by the authors to assess hypovolemia (hypotension, tachycardia, orthostasis, and low CVP or PCWP), no one would question the correlation of low urine output with this hypovolemic state. However, we question the need for the additional urinary indices to aid in the diagnosis or treatment of these hypovolemic patients. The clinical diagnosis appears to be more than sufficient evidence for treating these patients with a volume challenge.

Although the authors suggest that a low urine output in normovolemic patients was due to an excess antidiuretic hormone (ADH) concentration, they present no data and offer no opinion as to why these patients with normal renal function require treatment. Indeed, data substantiating ADH elevation as a cause of diminished urinary output is lacking. The influence of other humoral substances such as prostaglandins must be considered as potential etiologies of transient reductions in urine flow. In addition, it is the authors' implication that these patients may develop azotemia without furosemide treatment. However, the use of furosemide in preventing acute renal failure in normovolemic patients with low urine output has not been proven to be of benefit.

Finally, an important aspect that was not emphasized but that merits consideration is that the serum of the patients was hypotonic and hyponatremic. Catastrophic neurologic sequelae due to acute hypotonic hyponatremia have been well described in patients receiving excess hypotonic fluids in the presence of high ADH concentrations.[2] Pulmonary edema, as seen in two patients in this study, may occur also in the setting of overzealous hypotonic fluid administration. The avoidance of such therapy and the use of furosemide or free water restriction

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