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In Reply:—Ghouri and Feinstein raise important points about the relationship between the prevalence of malignant hypothermia (MH)-susceptibility and the negative predictive value [P(D−|T−)] of a negative caffeine halothane contracture test (CHCT). Although sensitivity and specificity are stable properties of any test, and are not affected by disease prevalence, positive and negative predictive values do vary with prevalence. The sensitivity and specificity [P(T−|D−)] of the CHCT have been estimated in swine, where sensitivity = 100% and specificity = 95%.[1] The prevalence of MH-susceptibility [P(D+)], *i.e.*, the pretest probability, can be estimated in many patients who undergo MH testing. Therefore, the negative predictive value can be easily calculated, using the equation[2] quoted by Ghouri and Feinstein. For the purpose of these calculations, we will assume that the sensitivity = 95%, *i.e.*, a false-negative rate [P(T−|D+)] of 5%, since few tests in medicine are 100% sensitive. In the swine we tested,[1] the prevalence of MH-susceptibility was 45%. The negative predictive value would be 96%.

The prevalence of MH-susceptibility from a number of biopsy centers ranges from 28 to 52%.[3-5] In our center it is 45%. Thus, the negative predictive value of a negative CHCT would be greater than 99%. A patient with an MH-susceptible (MHS) first-degree relative has 50% risk of being MHS. Similarly, a child with a history of masseter muscle rigidity (MMR) also has a 50% risk of being MHS.[6] An adult with previous MMR has a 25% risk of MH-susceptibility.[7] A patient tested for MH because of perioperative temperature elevation has a 15% risk of MH-susceptibility.* In all of these instances the negative predictive value of the CHCT would be over 99%.

What is the negative predictive value of a negative CHCT in a patient with no personal or family history of MH? As prevalence falls, the negative predictive value increases. Therefore, if one assumes a prevalence of 1 in 40,000,[8] the negative predictive value of the CHCT would be 99.9%. The patients in our study did not have a pretest probability of 1 in 40,000 but rather 45% overall.

Ghouri and Feinstein have arbitrarily chosen their values for sensitivity, specificity, and prevalence to make their point. However, these values are artificial, and are not supported in the literature. We made note of the small sample size of our study, and suggested cautious interpretation of the results. Indeed, we concluded in the abstract that "until the anesthetic experience of larger numbers of MH(−) patients is known, these results should be interpreted cautiously" and that "these results suggest that 'triggering' anesthetics may be safely administered to patients who test MH(−) by *in vitro* contracture testing."

Our study was based on more than 300 referrals for diagnosis over 3 yr. To increase the sample size would require cooperation among several centers (perhaps through the North American MH Registry). We encourage such efforts so that the utility of the CHCT can be evaluated objectively.

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* Unpublished observation.

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Microinfusers: Stopcock Usage for Efficiency and Asepsis

To the Editor:—Pumps currently available for infusion of Alfentanil and other medications include the Harvard Mini Infuser 900 (Bard, North Reading, MA) and the programmable Baxter Model AS 20GH, (Baxter Health Care Corporation, Hooksett, NH) pumps.

The Bard calculates and administers drugs based on the patient's body weight and the stock concentration by either bolus or continuous infusion rates. The Baxter pump operates in a similar manner except that the patient's body weight and the drugs used can be programmed into the machine.

Refilling the infusion syringe during the procedure is tedious and time-consuming. The infusion must be stopped, and the syringe removed to draw up the drug and then replaced on the pump. Moreover, interruption of continuous inotropic support by using the parent syringe to reload the system may create unwanted hemodynamic changes.

Placement of a three-way stopcock (Cobe, Lakewood, CO) with male luer locks on the infusion syringe allows for uninterrupted refilling of the 60-ml parent syringe with decreased risk of contamination of the system (fig. 1). The stopcock is placed between the parent syringe and

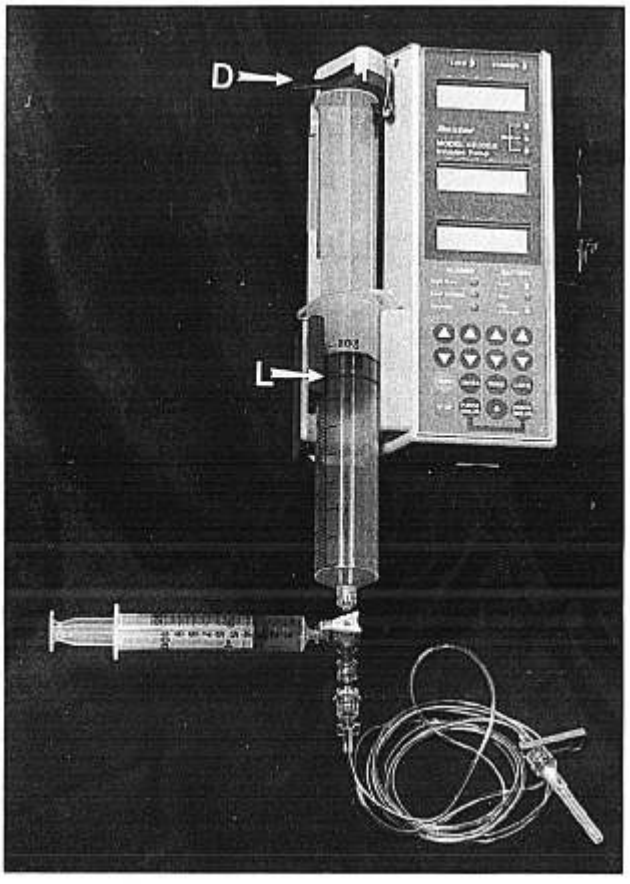

FIG. 1. Infusion set with stopcock. D = syringe driver; L = reference level.

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