pothesis, that the larger values of $\dot{V}_E$ consistently seen with halothane was the effect, rather than the cause, of greater rebreathing in that group. But this is unlikely. Even when rebreathing was minimal or absent ($\dot{V}_F$ = 200 ml·kg^{-1}·min^{-1}) $\dot{V}_E$ was larger in the halothane group. Moreover, end-tidal CO_2 concentrations were consistently lower in the halothane group despite the presence of a greater degree of rebreathing. This suggests that less respiratory depression was present with halothane than with enflurane, and that the $\dot{V}_E$ was always larger in the halothane group on that basis. Obviously a larger $\dot{V}_E$ at a given $\dot{V}_F$ will result in a smaller ratio of $\dot{V}_F$ to $\dot{V}_E$, and therefore more rebreathing will be seen.

It should be noted in fairness to the authors that controlling for differences in $\dot{V}_E$ during spontaneous ventilation, especially with different anesthetics, is extremely difficult. Nevertheless, the differences in $\dot{V}_E$ between patients breathing enflurane and those breathing halothane seen in this study can explain the observed differences in rebreathing. One could argue that the respiratory waveforms played no role at all.

It may be possible to get at the precise role of the respiratory waveform from the data presented by Byrick and Janssen, if a way could be found to adjust the data for differences in the $\dot{V}_F/\dot{V}_E$ ratios between the two groups. To this end, I calculated $\dot{V}_F/\dot{V}_E$ ratios for both anesthetic groups at each fresh gas flowrate, using the mean values of minute volume from their table 1 and assuming a body weight of 70 kg. I then plotted these against the inspired CO_2 volume per minute (their measure of rebreathing) and found that the results from both groups fell amost exactly along the same curve (see fig. 1). Perhaps with the original data, the authors might be able to see differences, presumably due to variation in respiratory waveform. My own guess is that if there is a difference due to waveform, it is small.

This paper implies that the Bain circuit is unpredictable in its performance because of the wide and uncontrollable variability of respiratory waveforms seen during spontaneous ventilation. The data presented by Byrick and Janssen—useful as they are—do not support this implication.

RICHARD L. KEENAN, M.D.
Department of Anesthesiology
Medical College of Virginia
Richmond, Virginia 23298

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In reply:—The aim of our study,[1] as the title suggested, was to analyze and compare the respiratory waveform of anesthetized patients during halothane and enflurane anesthesia, and to relate these waveform differences to rebreathing in T-piece circuits. The key role of minute volume ($\dot{V}_E$) and the $\dot{V}_F/\dot{V}_E$ ratio has been experimentally verified as Dr. Keenan suggests. Indeed, our hypothesis assumed that this relationship would exist, although presenting data in this manner would seem to verify our technique of measuring the inspired CO_2 load. The impact of respiratory waveform on rebreathing can only be analyzed when one considers the basic components of $\dot{V}_E$ which characterize a waveform, that is the inspiratory flow rate and the timing of each phase.

Milic-Emili et al.[2] introduced the concept of analyzing a given minute volume ($\dot{V}_E$) in terms of inspiratory drive (V_t/T_i) and the effective timing ratio. This relationship,

$$\dot{V}_E = V_t/T_i \times T_i/T_{tot} \times 60$$

characterizes the interdependence of $\dot{V}_E$ and the components of the respiratory waveform. By plotting the ratio $\dot{V}_F/\dot{V}_E$, Dr. Keenan is including the waveform characteristics on the x-axis which he wishes to isolate for examination. There are many variables (including dead-space, end-tidal CO_2 levels, and waveform) which will influence the relationship between inspired CO_2 volume and the $\dot{V}_F/\dot{V}_E$ ratio. The key finding of our study was that when halothane-anesthetized patients increased V_t/T_i, the fraction of CO_2 inspired increased. When enflurane was used, the exact opposite

pattern was noted, that is, the fraction of CO_2 inspired decreased as V_t/T_i increased.

The purpose of our paper was to demonstrate that anesthesiologists have two agents available that result in significantly different breathing patterns. The clinician is interested in only two facts when selecting an appropriate $\dot{V}_F$: 1) Can rebreathing be eliminated by choosing an agent which results in a favourable waveform?; and 2) Does the prevention of rebreathing with this waveform result in less CO_2 retention at comparable levels of anesthesia? Our study shows that the waveform during enflurane anesthesia (low V_t/T_i and long end-expiratory pause) reduces rebreathing when compared to the sine wave pattern with halothane at any $\dot{V}_F$. The prevention of rebreathing with enflurane, however, does not reduce arterial CO_2 tension, because it is a more potent primary respiratory depressant, and no circuit can compensate for hypoventilation.

Waveform, then, cannot be separated for analysis in a clinical study from $\dot{V}_E$ as Dr. Keenan would like because these two terms are interdependent. This interdependence is recognized by the clinician who can neither control nor select the waveform or $\dot{V}_E$ which is present during spontaneous respiration in anesthetized subjects. The levels of $\dot{V}_E$ in our study with enflurane and halothane are not significantly different from those reported by Spoerel *et al.*[3] The fact is, the clinician's choice of agent largely determines the waveform (including $\dot{V}_E$), and this is a prime determination of rebreathing as we have shown.

In concluding that our "paper implies that the Bain circuit is unpredictable in it's performance", Dr. Keenan has misinterpreted the results. This circuit is extremely predictable! Every patient breathing halothane at a $\dot{V}_F$ of 100 $ml \cdot kg^{-1} \cdot min^{-1}$ rebreathed CO_2. It is the patient's response to this inspired CO_2 load which is unpredictable during halothane anesthesia. In two other studies[4,5] using the same technique, we have demonstrated a significant increase in CO_2 tension in some patients who could not compensate for this inspired CO_2 load during halothane anesthesia. This increase in P_{CO_2} was preventable, by either increasing $\dot{V}_F$,[5] or by using another breathing system.[4]

We thank Dr. Keenan for his interest in the paper and we hope this clarifies his interpretation of the data.

ROBERT J. BYRICK, M.D. F.R.C.P.(C.)
Assistant Professor
Department of Anesthesia
St. Michaels Hospital
University of Toronto
Toronto, Ontario

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Hypothermia after Cardiopulmonary Bypass in Man

To the Editor:—We read with interest the article by Noback and Tinker[1] concerning the amelioration of hypothermia after cardiopulmonary bypass with nitroprusside-induced vasodilation. They concluded that the decrease in post-bypass nasopharyngeal temperature could be minimized by the simple procedure of using nitroprusside and increased pump flows during the rewarming period. Although their data show that the group of patients treated with nitroprusside had a smaller decrease (1.5° C *vs.* 2.5° C) in nasopharyngeal temperature than the control group upon termination of bypass, the fact remains that the nitroprusside-treated group did experience a decrease in nasopharyngeal temperature.

We have solved the problem of hypothermia after cardiopulmonary bypass by heating and humidifying the inspired gas. In our system the inspiratory gas is passed through a Bennett Cascade humidifier located between the carbon dioxide absorber and the Y-piece. The temperature of the inspired gas is controlled with